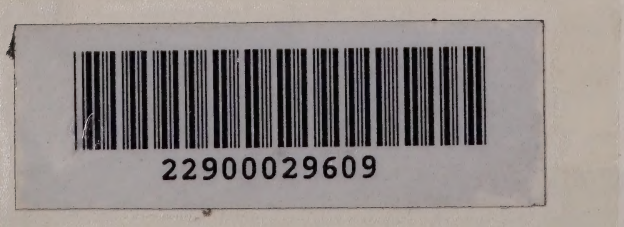

22900029609

HB.C(2)

PRESENTED TO

The Owens College,

BY

MRS. LEECH,

IN MEMORY OF

Daniel John Leech,

M.D., D.Sc., F.R.C.P.,

THE FIRST PROFESSOR OF

MATERIA MEDICA AND THERAPEUTICS

IN

THE OWENS COLLEGE,

1881—1900.

THE

PRINCIPLES AND PRACTICE

OF

MEDICINE

THE

PRINCIPLES AND PRACTICE

OF

MEDICINE

EDITED AND COMPLETED FROM THE MANUSCRIPT

OF THE LATE

CHARLES HILTON FAGGE, M.D., F.R.C.P.

PHYSICIAN TO, AND LECTURER ON PATHOLOGY IN, GUY'S HOSPITAL; EXAMINER IN MEDICINE IN THE UNIVERSITY OF LONDON

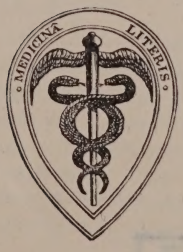
BY

PHILIP HENRY PYE-SMITH, M.D., F.R.S.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, PHYSICIAN TO, AND LECTURER ON MEDICINE IN, GUY'S HOSPITAL

Second Edition

VOL. II



LONDON
J. & A. CHURCHILL
11, NEW BURLINGTON STREET
1888

M16801

WELLCOME INSTITUTE LIBRARY

Coll.	welMOmec
Call No.	WB100
	1888
	F15p

CONTENTS OF THE SECOND VOLUME

PAGES

AFFECTIONS OF THE LARYNX AND TRACHEA

The laryngoscope and its use.

Laryngeal Paralysis—Of one or both recurrent nerves—Of the abductor muscles—Of the adductors—Aphonia and other disorders of the voice.

Laryngismus Stridulus— Name— Symptoms— Pathology—Ætiology—Prognosis, and treatment.

Laryngitis—Acute plastic inflammation—Croup—Its relation to Diphtheria and to Spurious Croup—Its symptoms and causes, anatomy, diagnosis, and treatment—Acute and chronic catarrh of the larnyx—Tubercle—Lupus—Syphilis—Œdematous laryngitis—Perichondritis.

Tumours—Papilloma—Polypus, &c.—Sarcoma and Carcinoma—Laryngeal malformations—Foreign bodies in the larynx.

Obstruction of Trachea—External compression from enlarged thyroid, aneurysm, &c.—Intrinsic stenosis from syphilis, &c.—Impaction of a foreign body—Symptoms, diagnosis, and treatment of laryngeal obstruction 1—68

DISEASES OF THE LUNGS

SYMPTOMS AND PHYSICAL SIGNS

Common Symptoms—Dyspnœa—Varieties—Phrenic dyspnœa—Cough—Bronchial, faucial, gastric, and cerebral—Pleurodynia.

Percussion—History—Methods—Terminology—Physical theory—Tympanitic resonance—Amphoric and cracked-pot sounds—Significance.

Auscultation—History—Methods—The respiratory murmur—Bronchial and tubular breathing—Physical explanation—Râles and Rhonchi—The vocal resonance in health—Bronchophony—Pectoriloquy—Ægophony.

Palpation—Tactile vibration—Inspection and measurement of the chest . 69—93

BRONCHITIS

General symptoms: cough, dyspnœa, pain—Physical signs: rhonchus, sibilus, râles—Morbid anatomy.

Acute bronchitis—Capillary form—Its symptoms and prognosis—Pulmonary collapse—Its production and relation to pneumonia.

Chronic Bronchitis—Varieties—Sequelæ—Emphysema—Its anatomy and origin—Atrophic emphysema—Symptoms and signs of emphysema—Bronchiectasis, uniform and saccular—Fœtid Bronchitis—Ætiology, prognosis, and treatment of Bronchitis generally.

Plastic Bronchitis—Its rarity—Anatomy, course, and symptoms—Treatment 94—128

PAGES

PNEUMONIA AND INFLAMMATION OF THE LUNGS

PNEUMONIA—History and definition—Anatomy and histology: stages, locality, events—Complications — Physical signs — Clinical symptoms—Prognosis—Mode of death—Convalescence—Origin and nature—Treatment.

Secondary pneumonia—Chronic lobar pneumonia.

BRONCHO-PNEUMONIA, or Pulmonary Catarrh.—Causes in children and in adults —Anatomy—Symptoms and course—Prognosis and treatment—Vesicular, caseous, and pyæmic pneumonia—Inflammatory œdema of the lungs.

CHRONIC INTERSTITIAL PNEUMONIA, or Cirrhosis of the Lung.—Anatomy—Origin—Symptoms.

SYPHILITIC INFLAMMATION OF THE LUNGS—Cases—Diagnosis—Anatomy—Acquired and hereditary forms.

GANGRENOUS INFLAMMATION OF THE LUNGS—Anatomy—Origin—Fœtor and other symptoms—Treatment . . . . . 129—162

PLEURISY

Dry pleurisy—Its physical signs—Pleuritic effusion—Compressed and carnified lung—Dulness—Ægophony—Symptoms—Course and events—Empyema—Diagnosis—Complications—Ætiology and relation to tubercle—Prognosis—Treatment of pleurisy and of empyema—Paracentesis and its results 163—188

PNEUMOTHORAX

Origin and pathology—post-mortem characters—Physical signs—Diagnosis and symptoms—Prognosis—Treatment . . . . . 189—199

PHTHISIS

History and definition—Pathology—Unity of phthisis—Localisation—Phthisis always tubercular—Histology: miliary, caseous, and infiltrating tubercle—Tubercular pneumonia and ulceration—Vomicæ: adhesions—Involution—Symptoms: wasting, pyrexia, cough, sputum—The bacillus—Hæmoptysis—Physical signs of the three stages and of involution—Diagnosis—Course—Mode of death—Prognosis—Ætiology—The question of contagion—Hereditary taint—"Diathesis"—Conformation—Overcrowding, &c.—Inhalation of dust and other irritants—Damp soil—Treatment: preventive, curative, and palliative—Diet and hygiene—Climate—Drugs . . 200—261

MILIARY TUBERCULOSIS OF THE LUNGS

Distinction from phthisis—Morbid anatomy—Physical signs—Clinical symptoms and course—Diagnosis by concomitant tuberculosis, especially of the choroid—Ætiology—Prognosis—Asserted cases of recovery and possible treatment 262—270

MINOR DISORDERS OF THE RESPIRATORY TRACT

CORYZA OR CATARRH—Its symptoms, predisposing causes and treatment—Feverish colds.

INFLUENZA OR EPIDEMIC CATARRH—History—Symptoms—Events—Ætiology —Diagnosis—Treatment.

OZÆNA—Origin in atrophy of the nasal tissues—Minor forms—Treatment.

EPISTAXIS—Ætiology—Course—Treatment.

HAY FEVER OR SUMMER CATARRH—Onset and course—Ætiology—Distribution among persons and places—Treatment.

WHOOPING-COUGH— Nomenclature— Symptoms and course—Events—Prognosis—Pathology—Treatment.

ASTHMA—The term—Description of an attack—Diagnosis and relation to structural lesions of the lung—Ætiology—Nature and physiology of the disease—Treatment and prognosis. . . . . 271—298

DISEASES OF THE ALIMENTARY TRACT

PAGES

AFFECTIONS OF THE MOUTH, THROAT AND GULLET

STOMATITIS—Vesicular ulcers of the mouth—Phagedænic ulcerative stomatitis—Gangrenous stomatitis or Cancrum oris—Thrush.

MUMPS—Ptyalism—Metastatic parotitis.

ANGINA—Catarrhal, ulcerative, and herpetic forms—Acute tonsillitis or quinsy—Chronic follicular tonsillitis—Granular pharyngitis—Adenoid growths of the pharynx.

STRICTURE OF THE ŒSOPHAGUS—Minor disorders—Spasmodic stricture—Simple stricture—Cancerous stricture—anatomy, course and treatment 299—317

DISEASES OF THE STOMACH

ACUTE DYSPEPSIA—Acute catarrhal gastritis—Its symptoms, causes, histology, and treatment—Acute paralytic distension—Acute suppurative gastritis.

CHRONIC DYSPEPSIA—Atonic and chronic catarrhal forms—Symptoms, causes, anatomy, diagnosis, and treatment.

Gastralgia—Anorexia—Eructation and Pyrosis—Vomiting—Hæmatemesis.

GASTRIC ULCER—Anatomy—Pathology—Ætiology—Symptoms—Event and duration—Treatment.

CANCER OF THE STOMACH—Carcinoma of the pylorus and of the body of the stomach—Sarcoma—Colloid cancer—Symptoms—The tumour—Consequent dilatation—Gastro-colic fistula—Duration—Diagnosis—Treatment.

Fibroid Induration of the Stomach.—Gastric concretions . . 318—367

FUNCTIONAL AND INFLAMMATORY DISORDERS OF THE INTESTINAL TRACT

COLIC—Onset—Causes—Diagnosis—Prognosis and treatment—Lead colic—History of its recognition—Modes of infection—Diagnostic characters—The blue line—Pathology and treatment—Other effects of lead.

DIARRHŒA—Acute and epidemic form—Chronic form—Tubercular enteritis—Diarrhœa from lardaceous and malignant disease—Symptoms—Treatment.

TYPHILITIS—Relation of inflammation of the cæcum to perityphlitis and disease of the appendix—Origin and course—Diagnosis—Treatment.

DYSENTERY—Anatomy—Catarrhal and diphtheritic forms—Sporadic and epidemic dysentery—Ætiology—Symptoms, diagnosis, course and event—Prophylaxis—Treatment of acute and of chronic dysentery.

CONSTIPATION—Origin—Effects—Treatment.

Acute catarrhal colitis—Ulcerative colitis—Intestinal casts . . 368—406

INTESTINAL OBSTRUCTION

INVAGINATION—Anatomy—Obstruction and subsequent strangulation—Ætiology and pathology—Symptoms: pain, tumour, hæmorrhage—Duration of first and second stages—Diagnosis—Treatment.

IMPACTION—Gall-stones—Concretions—Fæcal masses in the rectum or colon.

STRICTURE AND CONTRACTION—Cicatrices—Adhesions and contractions affecting the ileum—Simple and malignant stricture of colon and rectum.

STRANGULATION—Bands—Apertures in omentum or mesentery, &c.—Internal hernia—Volvulus—its seat, mechanism, and effects.

General results of obstruction and strangulation—Anatomy—General symptoms and diagnosis: constipation, pain, vomiting—Special symptoms of chronic obstruction—diagnosis of cause—treatment—colotomy—Special symptoms of acute obstruction—diagnosis of cause—prognosis—treatment: expectant—by abdominal section—by other mechanical methods . . 407—434

PAGES

INTESTINAL WORMS

TAPEWORMS—Anatomy and transformations—Tænia solium—its distinctive characters and distribution—its relation to Cysticercus cellulosæ of pork—Tænia mediocanellata—its specific distinctions—its relation to the cysticercus of beef—its geographical distribution—Bothriocephalus latus—its characters and distribution—its probable origin—Symptoms of the presence of tapeworms generally—Preventive and curative treatment.

ROUND-WORMS—Ascaris lumbricoides—its anatomy—natural history—distribution—symptoms, diagnosis, and treatment—Thread-worms—anatomy—symptoms and treatment—Trichocephalus dispar—Eustrongylus gigas.

SCLEROSTOMUM DUODENALE—Its discovery—anatomy—habits—Anæmia caused by its presence—Treatment.

TRICHINA SPIRALIS—Its discovery—anatomy, transmigrations, and encapsulation—Trichiniasis—its symptoms, diagnosis, treatment, and prevention.

FILARIA SANGUINIS HOMINIS—Its discovery in the blood—its transmigrations—Echinorhynchus—Dracunculus—Distomum—Bilharzia . 435—462

DISEASES OF THE PERITONEUM

ACUTE PERITONITIS—Importance—Clinical symptoms—Origin: secondary to visceral disease—Puerperal—Uræmic—Extreme rarity of idiopathic acute peritonitis—Morbid anatomy—Local peritonitis and circumscribed abscess—Diagnosis—Treatment: medical and operative.

CHRONIC PERITONITIS—General effusion—Thickening—Adhesions—Locular effusion—Frequency—Causes.

TUBERCLE OF THE PERITONEUM—Anatomy—Symptoms—Relation to phthisis—Diagnosis—Treatment.

CANCER OF THE PERITONEUM—Commonly secondary—Anatomy—Symptoms.

ASCITES—Its physical signs—its varieties—its cause—Treatment by drugs and by paracentesis abdominis . . . . . 463—486

DISEASES OF THE LIVER

BILIOUS DISORDERS AND JAUNDICE

BILIOUS OR HEPATIC DYSPEPSIA—Disturbance of secretion or elimination of bile—Disturbance in nitrogenous metabolism—Lithæmia—Bilious symptoms—Treatment by diet, drugs, and exercise.

ICTERUS—Symptoms—Tests for bile-pigment and for bile-acids in the urine.

Simple or idiopathic jaundice: its course, pathology, and diagnosis.

Symptomatic jaundice—Febrile jaundice—from general pyæmia—from portal pyæmia, suppurating hydatid and acute tubercle—Jaundice from cirrhosis—from simple and malignant obstruction of the ducts—from tumours, etc.

Jaundice from gall-stones—Their structure and varieties—Symptoms and events of biliary colic.

Effects of permanent jaundice on the liver—Pathological theories of jaundice.

Treatment of jaundice—primary, symptomatic, and calculous—of permanent jaundice—Treatment of other effects of gall-stones . . . 487—512

PAGES

INFLAMMATORY DISEASES OF THE LIVER

ACUTE SUPPURATIVE HEPATITIS—ABSCESS OF THE LIVER—Geographical distribution—Ætiology—Relation to dysenteric ulceration—Morbid anatomy—Characters of the pus—Direction of rupture—Symptoms—Physical signs—Diagnosis—Abscess between the liver and diaphragm—Prognosis—Treatment by drugs—Paracentesis.

CHRONIC INTERSTITIAL HEPATITIS—CIRRHOSIS OF THE LIVER—Anatomy—Ætiology—Effects upon the portal circulation—Early symptoms—Jaundice—Cerebral symptoms—Hypertrophic cirrhosis—Ascites—Diagnosis—Perihepatitis—Simple chronic atrophy—Syphilitic hepatitis and gummata of the liver—Cancer—Adhesive portal phlebitis—General prognosis and treatment of cirrhosis.

ACUTE ATROPHIC HEPATITIS—ACUTE YELLOW ATROPHY OF THE LIVER—History—Anatomy—Symptoms—percussion—the urine—jaundice—cerebral symptoms—Ætiology and pathology—Diagnosis—Prognosis—Treatment 513—537

NEW GROWTHS, DEGENERATIONS AND PARASITES

Malignant disease - Carcinoma hepatis—its anatomy and histology—its rarity as a primary disease and its most frequent antecedents—its symptoms and course—Cancer of the biliary passages.

Hypertrophy of the liver—Lymphatic overgrowth—leuchæmia hepatica—The fatty liver—general obesity and its treatment—Malarial enlargement—Lardaceous disease—Hydatids of the liver—and of other organs—Other parasites 538—558

AFFECTIONS OF THE URINARY ORGANS

FUNCTIONAL DISORDERS

Arrangement adopted—Polyuria and oliguria; high and low specific gravity—Diabetes insipidus—"Renal inadequacy"—Reaction: alkaline urine, and phosphatic deposits—Urobilin and Indican—Uric acid and urates—Oxalates—Cystine—Calcic sulphate—Ammoniacal urine—Bacilluria—Hæmoglobinuria 559—590

BRIGHT'S DISEASE

History—The two more frequent forms—Additional varieties—Common characters of the disease—Albuminuria: its detection and estimation, pathology, and significance—Casts: their structure, varieties, and meaning—Dropsy: its pathology, its primary and secondary form—Retinitis—Serous and other inflammations—Changes in the heart and arteries: the renal pulse, hæmorrhage—Uræmia: eclampsia, coma, amaurosis; vomiting; theory of uræmia.

Parenchymatous Nephritis—Causes—Anatomy—Symptoms—Event.

Lardaceous nephritis—Chemistry—Anatomy—Symptoms—Causes—Prevention.

Cirrhosis of the kidney—Pathology—Causes—Anatomy—Symptoms and clinical aspects—Consecutive renal cirrhosis—Its relation to local disease of the bladder, urethra, and uterus—Microcystic degeneration—Hypertrophic cystic kidneys.

General prognosis and treatment of Bright's disease . . 591—656

URINARY CALCULI AND THEIR EFFECTS

CALCULI—Lithic or uric acid—Lithate of soda—Oxalate of lime—Cystine—Xanthine—Indigo and other rare substances—Phosphate of lime—Carbonate of lime—Fusible calculus—Ætiology of calculi.

NEPHROLITHIASIS—Lumbago—Hæmaturia—Renal colic—Obstruction and suppression of urine—Atrophy—Hydronephrosis—Pyelitis and renal abscess—Solvent and operative treatment of calculi and of their results . 657—688

PAGES

TUBERCLE, CANCER, PARASITES AND ABNORMALITIES OF THE KIDNEYS

TUBERCULAR PYELITIS—Pathology—Symptoms, causes, and treatment.
MALIGNANT DISEASE—Sarcoma—Carcinoma—Symptoms—Nephrectomy.
Hydatids—Chyluria and Filaria sanguinis—Endemic hæmaturia and Bilharzia.
Renal malformations—Floating kidney . . . 689—707

DIABETES

Definition—Detection of glycose in the urine: the cupric, potash, fermentation, and other tests—Quantitative estimate by volumetric method—The amount, specific gravity, and other characters of the urine—Symptoms of diabetes—Complications—Modes of death—Physiology of the formation of sugar in the body—Glycosuria without diabetes—Theories of diabetes:—(1) escape of sugar—(2) over-formation of sugar—(3) diminished destruction of sugar—exalted glycogenetic function of the liver—Experimental glycosuria—General ætiology of diabetes—Prognosis—Treatment: by diet—by drugs . 708—732

ADDISON'S DISEASE, ETC.

The anatomical changes in the adrenal bodies—The symptoms: melanodermia, asthenia, cardiac failure, gastric disturbance—Order of the symptoms—Course and duration of the disease—Mode of death—Controversial points—Origin—Relation to caries and to tubercle—Theory of the symptoms—Prognosis—Other morbid conditions of the adrenal capsules.
Diseases of the pancreas, the spleen, the testes, and the ovaries . 733—745

DISEASES OF THE BLOOD

CHARACTERISED BY ANÆMIA AND HÆMORRHAGE

ANÆMIA—Its general characters—Amount of blood—Number of red corpuscles—Amount of hæmoglobin—Changes in the blood-discs—Anæmic murmurs—Dyspnœa—Fatty degeneration of the heart—Other symptoms—Symptomatic or secondary and idiopathic or essential anæmia.

LEUCHÆMIA—History—Anæmia splenica—Definition and characteristic symtoms—Physical signs of an enlarged spleen—The blood in leuchæmia—The other organs—Ætiology—Diagnosis—Pathology, course and treatment.

HODGKIN'S DISEASE—Anæmia lymphatica — History — Ætiology — Morbid anatomy and pathology—Course, prognosis, and treatment—Primary tuberculosis of the lymph-glands.

CHLOROSIS—Its relation to menstruation—to imperfect development of the heart and vessels—Symptoms—Prognosis—Treatment.

IDIOPATHIC OR PERNICIOUS ANÆMIA—History—Name—Age and sex—Antecedents—Course and symptoms—Ætiology and diagnosis—Pathology—Prognosis—Treatment.

SCURVY—History—Early symptoms: purpura, spongy gums, hæmorrhagic inflammations—Night-blindness—Course and Prognosis—Ætiology—Prophylactic treatment—Pathology—Curative treatment—Diagnosis.

HÆMOPHILIA—History—Inheritance usually in males through females—Course and symptoms—Prognosis—Diagnosis—Pathology—Treatment.

PURPURA—Symptomatic and primary purpura—Ætiology—Characters, events, and treatment—Morbid anatomy—Pathology—Diagnosis . 746—796

GENERAL DISEASES OF THE JOINTS AND BONES

PAGES

GOUT

History—Onset and symptoms of a first attack—Subsequent course—The affected joints—Lithate of soda—Tophi—Pathology of gout—Ætiology—Diagnosis—Course and prognosis—Gouty kidneys—Treatment by drugs, diet, and baths . . . . . 797—809

RHEUMATISM

Definition—Onset, course, and symptoms—Anatomy of the inflamed joints—The pyrexia, sweating, &c.—erythema—cardiac complications—pleurisy, tonsillitis, nodules—Ætiology and pathology of rheumatism—Prognosis: fatal cases—Rheumatic hyperpyrexia—Diagnosis, particularly from pyæmia—Treatment by older methods—Observation of the natural course of the disease—Treatment by salicin and salicylates—Treatment of hyperpyrexia in rheumatism. . . . . 810—834

GONORRHŒAL SYNOVITIS

Distinction from rheumatism—Sex and age—Relation to gonorrhœa—Distribution—Symptoms—Sclerotitis—Prognosis—Diagnosis—Pathology—Treatment . . . . . 835—839

OSTEO-ARTHRITIS

History and nomenclature—Pathology and relation to gout and to rheumatism—Anatomy of the joints—Symptoms and course—Acute cases—Resulting deformity—Involution and recovery—Ætiology—Prognosis—Treatment by drugs and external applications, by diet and climate.

Charcot's arthropathie ataxique, its relation to osteo-arthritis and tabes dorsalis 840—849

GENERAL DISEASES CHIEFLY AFFECTING THE BONES

Rickets—History—Anatomy of the rachitic limbs—of the thorax—pelvis—skull—craniotabes—Histology—Course and event—The spleen in rickets—Symptoms—Ætiology: age, sex, climate, food—Relation to tubercle and syphilis—Pathology—Diagnosis and prognosis—Treatment, preventive and curative.

Mollities Ossium—History—Ætiology—Symptoms and events—Diagnosis—Histological and chemical changes—Pathology . . . 850—864

DISEASES OF THE SKIN

INTRODUCTORY CHAPTER

General pathology—Classification—Nomenclature—Historical sketch—The elementary anatomical lesions—The local distribution of cutaneous affections: in depth (bathymetric) and over the surface (regional)—The circumstances: age, occupation, and habits of the patient, season, history of the malady, subjective symptoms, disturbance of other organs, effect of previous treatment 865—871

ECZEMA AND COMMON SUPERFICIAL DERMATITIS

Definition—Willan's—Hebra's—Its distinction from other forms of dermatitis—Histology—Anatomical lesions—Course—Distribution and local varieties—General symptoms—Ætiology—Diagnosis—Prognosis—General treatment—Local applications—Diet and regimen, baths, &c.—Internal remedies—Special treatment of local varieties.

Impetigo—Its relation to eczema—to pediculi—to contagion—Its treatment 872—896

PAGES

PAPULAR FORMS OF SUPERFICIAL DERMATITIS

Definition—Relation to common dermatitis and to eczema.

Lichen—Its traditional species—Strophulus—Lichen scrofulosus—Lichen planus.

Prurigo—Pruritus—Summer and winter prurigo—Prurigo senilis a pediculis—Idiopathic prurigo: Hebra's and milder forms—Characters and treatment 897—906

PITYRIASIS RUBRA (General Exfoliative Dermatitis)

Name—History—Accounts by Devergie, Hebra, Wilson, Hutchinson, Baxter—Course and symptoms—Histology—Prognosis—Diagnosis—Treatment 907—909

PSORIASIS

Frequency—Name—Anatomy and histology—Course—Symptoms—Distribution—Ætiology—Varieties: guttate form, inveterate form—Relation to pityriasis rubra, to eczema, and to syphilis—Prognosis—Treatment . 910—916

PEMPHIGUS

Names and definition—Anatomy—Histology—Local distribution—Age and sex—Question of an acute form of pemphigus—Diagnosis—Prognosis—Pemphigus malignus—Pemphigus foliaceus—Hutchinson's cases—Chiropompholyx—Hydroa of Bazin—Frequency of pemphigus—Treatment 917—922

SCABIES

Importance of the disease—Its nature—The acarus—The superficial dermatitis it produces—The distribution of the parasite and of the inflammation—Diagnosis—Treatment. . . . . 923—926

ERYTHEMA AND ITS ALLIES

Definition of the group—Its characters—The anatomical lesions—Course—Locality—Symptoms—Ætiology—Symptomatic and traumatic erythema—Pernio and pernio-like inflammations—Varieties (1) Erythema multiforme—Herpes—Erythema iris—Herpes gestationis—(2) Erythema nodosum—(3) Urticaria—Urticaria pigmentosa—Treatment of erythematous affections.

Rashes produced by Drugs—The erythematous rash of Copaiba—Bromides and Iodides—Belladonna—Opium—Quinine—Salicylates—Arsenic—Mercury 927—941

DISORDERS OF THE SEBACEOUS GLANDS, THE HAIR-SACS, AND THE SWEAT-GLANDS

Acne—Nomenclature—Anatomy and course of local lesions—Distribution—Age and sex—Symptoms—Ætiology—Treatment—Acne tarsi—Acneiform eruptions produced by tar—by bromide—Acne varioliformis.

Comedones without inflammation (acne cornée)—Milium—Seborrhœa oleosa—Seborrhœa sicca—Xerodermia—Steatoma, meliceris and sebaceous cysts.

Molluscum Contagiosum—Name and history—Anatomy—Pathology—Treatment.

Sycosis—Name—Anatomy and course—Locality—Diagnosis—Distribution—Treatment—Sycosis capillitii frambœsiformis.

Furunculi—Pathology—Anatomy—Course and distribution—Age—Contagion—Treatment—Carbuncle.

Affections of the Sweat-glands—Anidrosis—Hyperidrosis—Bromidrosis or fœtid sweat—Chromidrosis—Hæmatidrosis—Sudamina . . 942—961

PAGES

RINGWORM, WITH OTHER AFFECTIONS OF THE HAIR

RINGWORM—(1) Tinea tonsurans — Anatomy—Course—Events — Histology—Detection of the fungus—Ætiology—Prognosis—Treatment—Parasiticides—Irritants—Mode of application—Epilation—Precautions against contagion—(2) Tinea circinata—Form and locality—Burmese ringworm—Tinea marginata—Treatment of ringworm of the body—(3) Onychomycosis.

FAVUS—History—Anatomy—The fungus—Treatment.

TINEA VERSICOLOR—Names—Parasitic nature—Appearance—Distribution—Diagnosis—Treatment—Tinea vel Pityriasis rosea.

ALOPECIA—(1) Physiological—(2) Febrile and syphilitic—(3) Area—its appearance—locality—spread—question of its parasitic nature—prognosis—diagnosis—(4) Universal alopecia—(5) Congenital alopecia.

TRICHOCLASIA or brittleness of the hair . . . . 962—977

CHRONIC DEEP HYPERTROPHIC DERMATITIS

Deep and chronic dermatitis—its definition—its relation to eczema and other forms of superficial dermatitis—to hypertrophy—and to new growths.

GUTTA ROSEA—Origin in recurrent erythema—Development—Localities—Causes and pathology—Relation to dyspepsia—to drink—to ovarian irritation

EPIDERMIC AND PAPILLARY HYPERTROPHIES—Callosities and corns—Leucoplacia lingualis et buccalis—Warts—Condylomata and mucous patches.

ICHTHYOSIS—Anatomy—Varieties—Xerodermia—Prognosis and treatment—Ichthyosis intra-uterina—Horns.

SCLERODERMIA—History—Description—Distribution—Histology—Diagnosis—Prognosis and treatment—Sclerema neonatorum—Linear atrophy.

ELEPHANTIASIS—Nomenclature—Anatomy — Pathology—Relation to chyluria and filaria sanguinis—Clinical characters—Dermatolysis.

XANTHELASMA — History—Course and symptoms — Histology—Relation to jaundice . . . . . 978—994

LUPUS

Definition, history, and nomenclature—Anatomy, histology, and course—Locality—Symptoms—Age, &c.—Diagnosis from cancer, rodent ulcer, and syphilis—Pathology—Relation to tubercle and scrofula—Treatment.

Lupus erythematosus—Pathology—Locality—Course—Histology—Treatment.

Disseminated erythematous lupus—Rhinoscleroma. . . 995—1008

LEPROSY

History and terminology—Geographical distribution—Anatomical lesions and course—Histology—The bacillus lepræ—Symptoms and event—Ætiology—Treatment—Other exotic diseases . . . . 1009—1012

TUMOURS OF THE SKIN

MULTIPLE FIBROMA—Distinction from molluscum sebaceum—Anatomy and distribution, course and treatment of the tumours—Neuroma—Myoma.

CHELOID—Terminology and history—Appearance, course, and symptoms—Histology—Relation to scars—Distribution—Prognosis and treatment.

Angioma or vascular nævus—Elephantiasis teleangiectodes—Lymphangioma.

Mycosis fungoides—Kaposi's xerodermia maligna—Carcinoma, sarcoma, and rodent ulcer of the skin . . . . . 1013—1020

PAGES

PIGMENTAL, HÆMORRHAGIC, AND NEUROTIC AFFECTIONS

Albinism—Leucodermia, congenital and acquired—Its relation to leprosy—Canities—Melanodermia, secondary to dermatitis, syphilis, adrenal disease, malaria, &c.—Ephelis—Lentigo—Chloasma—Melanodermia and leucodermia. Petechiæ and vibices—Peliosis rheumatica. Trophic neuroses—Zona—Symmetry in cutaneous diseases . . 1021—1029

REMARKS ON THE PRACTICAL CLASSIFICATION AND DIAGNOSIS OF CUTANEOUS DISEASES . . . . . 1030—1034

INDEX OF SUBJECTS . . . . . 1035—1066

INDEX OF AUTHORS . . . . . 1067—1103

THE

PRINCIPLES AND PRACTICE OF MEDICINE

VOL. II

DISEASES OF THE RESPIRATORY SYSTEM

AFFECTIONS OF THE LARYNX AND TRACHEA

The laryngoscope and its use.

LARYNGEAL PARALYSIS—*Of one or both recurrent nerves—Of the abductor muscles—Of the adductors—Aphonia and other disorders of the voice.*

LARYNGISMUS STRIDULUS—*Name—Symptoms—Pathology—Ætiology, prognosis, and treatment.*

LARYNGITIS—*Acute plastic inflammation—Croup—Its relation to Diphtheria and to Spurious Croup—Its symptoms and causes, anatomy, diagnosis, and treatment—Acute and chronic catarrh of the larynx—Tubercle—Lupus—Syphilis—Œdematous laryngitis—Perichondritis.*

TUMOURS—*Papilloma—Polypus, &c.—Sarcoma and Carcinoma—Laryngeal malformations—Foreign bodies in the larynx.*

OBSTRUCTION OF TRACHEA—*External compression from enlarged thyroid, aneurysm, &c.—Intrinsic stenosis from syphilis, &c.—Obstruction by a foreign body—Symptoms, diagnosis, and treatment.*

THE diseases of the larynx, like those of some other parts of the body, have within recent times been made accessible, as they never were before, to actual clinical study. This is entirely due to the invention of a special instrument—the *laryngoscope*, which was first introduced into medical practice in Vienna by Türck and by Czermak. There arose a lively contest for priority in the discovery, and although the question has happily now become almost forgotten, the main facts are perhaps still worth recalling. Türck, in the summer of 1857, began to examine his hospital patients with a laryngeal mirror, such as had been used in physiological researches a few years before by Manuel Garcia, a singing-master, who read a paper on the voice before the Royal Society of London. Even this was not the first starting-point of efforts to see the interior of the larynx in the living subject, for it had been attempted, though with but little success, by several other observers, including the younger Dr Babington in 1829 and Mr Avery in 1844. Türck himself suspended his operations in the winter of 1857–58 for want of sunlight,

and he lent his mirrors to Czermak, who setting to work with artificial illumination, became rapidly convinced of the extreme value of laryngoscopy for clinical purposes, and after publishing a paper on the subject in March, 1858, in the 'Wiener Medicinische Wochenschrift,' travelled over Germany, France, and England to make it more widely known.

The laryngoscope is now in the hands of every practitioner. In using it, the first thing is to secure a bright light, whether from an Argand burner, an oil lamp, the limelight apparatus, or the sun itself. The patient is placed with his back to the source of illumination, the rays from which pass over one of his shoulders. The observer seats himself opposite, on a chair slightly more raised, and throws the light upon the lower part of the patient's face by means of a flat or slightly concave mirror. The patient is next made to open his mouth and to protrude his tongue, and this is gently grasped by the observer with his thumb and forefinger in the folds of a napkin, or given to the patient himself to hold should the observer require to keep both hands free. The light is thrown into the back of the fauces and kept steadily fixed there. In the meantime a stalked laryngeal mirror is warmed over a flame, so that its surface may not become dimmed by the moisture of the breath. To be sure that it is not too hot the observer tests it by laying it for an instant upon the back of his hand. The patient is now instructed to go on breathing quietly and regularly, and to sound an "a" (as in fate) on rather a high note. This brings the fauces into a position advantageous for the introduction of the laryngeal mirror, which is held like a pen between the finger and thumb, and gently but rapidly passed through the patient's mouth until it reaches the uvula, while the stem lies at the angle of the mouth so as not to interfere with the entrance of light. The observer should learn to use the laryngeal mirror with the left hand as freely as with the right, because it is often necessary to have the right hand disengaged for some other purpose while the mirror is held in position. In traversing the mouth the instrument must have its face turned downward, and it must take a curved course, being kept close to the palate and as far as possible from the tongue, contact with which would of course soil its surface. As it reaches the uvula it has to be tilted so that its face looks forwards as well as downwards. It has also to be gently pushed upwards and backwards, lifting the uvula and the velum on its back. While this is being done the epiglottis and the interior of the larynx commonly become visible, being reflected from the surface of the mirror. If this is not the case, slight changes in its position or in its inclination generally bring them into view. It must not be moved about in the fauces, for such a proceeding is almost certain to irritate the mucous membrane and to set up retching or cough. As a rule it should not be carried so far back as to touch the posterior wall of the pharynx, which in many patients is far more sensitive than the velum, but some persons bear the mirror perfectly well, even when it is made to rest on the pharyngeal surface. There is seldom any necessity, so far as purposes of diagnosis are concerned, for continuing a single observation for more than a very short time. Should it be found that the interior of the larynx is not completely visible it is better to withdraw the instrument and to reintroduce it a minute or two later, when the patient has had a little rest.

It may well be supposed that the practical use of the laryngoscope is in some cases attended with difficulties. In this matter, as in all others, habit goes a long way. The student finds himself baffled, and fails altogether to obtain a view of the larynx, whereas the trained observer succeeds at the

first attempt; and, on the other hand, the patient who when the mirror is first introduced in his mouth thinks he cannot tolerate its presence, finds after a few trials that it is scarcely even uncomfortable. One trouble is with the tongue, which in some persons becomes arched upwards, so as to interfere not only with the passage of the mirror, but also with the admission of light to the back of the throat. They must then be directed to practise before a looking-glass until they can "make a wide throat." Or the tongue may be held down by a spatula, which, however, is apt to cause retching. Another obstacle is the presence of enlarged tonsils narrowing the faucial space. This is best overcome by using a mirror small enough to be slipped in between them. In some patients again the uvula and the velum are so irritable that the slightest contact of the mirror causes choking, or retching, or cough. Dr Morell Mackenzie recommends that to meet this difficulty small pieces of ice should be sucked for fifteen or twenty minutes before the laryngoscopic examination is begun; this, he says, rarely if ever fails to blunt for a time the sensitiveness of the mucous membrane. The best method, however, of securing anæsthesia of the fauces is to apply cucaine locally. A 20 per cent. solution brushed over the part, or, still better, applied as a spray, will after a few minutes enable one to examine the most sensitive patient.

In some patients, who have affections of the throat or lungs, the introduction of the mirror seems at once to be followed by the entrance of a quantity of muco-purulent fluid into the fauces from below, notwithstanding the repeated use of a gargle. Or the uvula may be so long and pendulous that it curls round the under edge of the mirror, interfering with the view of the larynx, or soiling the reflecting surface. The way to correct this is to use a large mirror, so as to lift up the whole of the uvula. But the most serious difficulty of all is that which is sometimes caused by a large epiglottis, which hangs backwards over the entrance of the larynx in such a way as to prevent anything else being seen. In many cases this obstacle is easily removed by making the patient sound, or attempt to sound, the vowel *e* (as in *feet*) on a high note. The sound itself cannot actually be produced while the tongue is protruded, but Störk says that the effort to produce it is often sufficient to raise the epiglottis. Sometimes the interior of the larynx can be seen, in spite of a pendant epiglottis, if the mirror is placed rather lower in the fauces than usual and with a more vertical inclination of its surface, the patient's head being at the same time thrown far backwards. But it may happen that all these plans fail. An attempt may then be made to raise the epiglottis by a curved sound brought into contact with its posterior surface. Fränkel, in 'Ziemmsen's Handbuch,' says that when the epiglottis has been raised by such an instrument it falls back again slowly, so that after removal of the sound, one can obtain a glimpse of the interior of the larynx. In most persons the under surface of the epiglottis is so sensitive that a choking sensation is produced as soon as it is touched. But the use of the cucaine solution enables one to draw the epiglottis forward with the blunt hook in the left hand, guided by the mirror held in the right.

The parts reflected in a laryngeal mirror retain their proper positions so far as concerns the side of the body on which they seem to lie; the left vocal cord is visible upon the left side of the mirror, the right one upon the right side. But in an antero-posterior direction they appear to be inverted in such a way that one might suppose oneself to be looking at the larynx

from behind instead of through the mouth, as is actually the case. In other words, the base of the tongue and the epiglottis form the top of the laryngoscopic image, the arytænoid cartilages and the entrance of the œsophagus are at the bottom of it. It is to be observed that the appearance of the epiglottis varies widely in different persons. Sometimes little more than the edge of it is seen, sometimes a large part of its posterior surface, which has normally a bright red colour, apt to be taken for morbid congestion. The rest of the laryngeal mucous membrane is of a paler tint, the vocal cords themselves being white and glistening.

Most of the affections of the larynx interfere with the natural performance of both of its two principal functions, the formation of the voice and the passage of air into and out of the trachea; and many of them are also attended with other symptoms, such as pain, tenderness, a peculiar cough, and dysphagia, as well as with varied and complicated laryngoscopic appearances. It therefore seems advisable to begin the description of laryngeal diseases with certain affections, of which some give rise only to an impairment or loss of the voice, others only to interference with the breathing, the results of examination with the mirror being correspondingly simple and definite. These are the paralytic and the spasmodic affections of the muscles of the larynx. Although, being of secondary importance to the specialist, they are usually consigned to the end of systematic treatises, they possess peculiar interest for the general physician on account of their bearing on diseases of distant structures. Their proper place in this work would, indeed, be among the affections of the nerves or of the nervous centres, but for reasons of practical convenience it is desirable to take them with other affections of the larynx.

Different writers classify laryngeal paralyses in different ways. Störk first discusses affections of the several muscles one by one, as they may theoretically be imagined to occur; afterwards he passes to more complex forms, in which many muscles are involved together. Dr Mackenzie arranges them according to their supposed seat in the bulb, in the trunks of the vagi, or in the various laryngeal branches of those nerves. For our present purpose it will be sufficient to describe such forms of paralysis as are actually recognisable in clinical practice, noting as far as possible the nervous lesions that give rise to each and the muscle or muscles involved.

Brief accounts of the spasmodic affections to which the laryngeal muscles are liable will follow.

Paralysis of all the Muscles supplied by the Recurrent Laryngeal Nerve or Nerves.—Among the most frequent paralytic affections of the larynx, as might naturally be anticipated, is one which involves all the muscles supplied by the recurrent nerve either on one side or on both. This, when unilateral, is sometimes spoken of as "hemiplegia of the larynx," but it is clearly unadvisable to employ the term hemiplegia for a local form of paralysis. If a special name is wanted, it would be far better to follow the analogy of the word ophthalmoplegia, invented by Mr Hutchinson for a general paralysis of all the muscles of the eyeball, and to speak of "laryngoplegia" when the muscles of both sides of the larynx are affected, while the name of "hemilaryngoplegia" might be assigned to cases in which the paralysis is one-sided. It is true that the paralysis is not quite universal, since the crico-thyroid muscles must be supposed to escape. But

it does not appear that any appreciable physiological action results from their contraction when the other laryngeal muscles are powerless. Moreover, Türck is said to have observed fatty degeneration and atrophy of the crico-thyroid muscle in a case in which the recurrent laryngeal nerve was alone affected, while the superior laryngeal nerve entirely escaped.

It will be necessary to describe separately the effects of unilateral and of bilateral paralysis of the recurrent nerve.

1. *Unilateral paralysis of the recurrens* is characterised by a complete immobility of the corresponding vocal cord, whether the patient only continues to breathe or utters a vocal sound. The position occupied by the cord is usually what is termed the "cadaveric position," the same as that assumed by the vocal cords in the dead body, when no muscular force any longer acts upon them—intermediate between that of phonation and that of inspiration. But sometimes the cord stands nearer the middle line, being drawn inwards by the action of the arytænoideus muscle. The outline of the cord looks concave. The summit of the arytænoid cartilage is placed a little further forwards and inwards than that of the opposite cartilage, so that it looks larger in consequence of more of its hinder surface being exposed to view. When a sound is uttered, the opposite arytænoid cartilage moves further than usual, and the unaffected vocal cord is drawn up to and even across the middle line, until it may come close to the affected cord, so that the chink of the glottis becomes oblique. At the same time the summit of the mobile arytænoid cartilage crosses in front of the cartilage on the paralysed side. When paralysis has lasted a long time, the affected cord may be seen to be obviously atrophied, and may oscillate backwards and forwards as the stream of air passes over it.

The voice of a patient with paralysis of one recurrent nerve is often much less altered than might have been expected. It is apt to be weak and more or less hoarse, and sometimes breaks into a falsetto as soon as an attempt is made to speak forcibly. A point to which Gerhardt has drawn attention is that when two fingers are placed, one on each side of the thyroid cartilage, while the patient is speaking, a more distant vibration can be felt with one finger than with the other. There is not the slightest dyspnœa.

2. *Bilateral paralysis* of all the muscles supplied by the two recurrent nerves is characterised by immobility of both vocal cords in the cadaveric position. It is to be noted, however, that the paralysis is not seldom incomplete on one side or on the other, in which case partial movements of one cord may of course be detected.

There is complete aphonia, the voice being reduced to a whisper. The patient is unable to cough or to expectorate at all forcibly. There is no dyspnœa, at least in adults. This is a point about which there was at one time some divergence of opinion, but it seems now to have been finally settled. Scheck has recorded a case in a boy of seven, whose breathing was in no way interfered with. Ziemssen, however, still says in his 'Handbuch' that in deep inspiration the cords may be drawn a little further inwards than before, and that a stridulous sound may be produced.

Diagnosis.—It must be borne in mind that immobility of the vocal cord, whether on one side or on both, is not in itself proof of the presence of a paralytic affection. As Dr Semon pointed out in the 'Medical Times and Gazette' for 1880, precisely the same laryngoscopic appearances may be the result of ancylosis of the crico-arytænoid joints. This fact had, indeed, been recognised to some extent by previous writers, especially in Germany. But

it seems to have been generally supposed that perichondritis, whether of the cricoid or of one of the arytænoid cartilages, before leading to fixation of the corresponding joint, must almost of necessity be attended with suppuration and with swelling of the overlying submucous and mucous tissues. Dr Semon has insisted that all such changes may be absent, and that the affection may produce only a development of fibrous tissue which obliterates the synovial cavity and unites the cartilages firmly to one another. The causes of ancylosis of the crico-arytænoid joints will be discussed under the head of laryngeal perichondritis. It is of course only when the arytænoid cartilages are so fixed as to bring the cords into the cadaveric position that the case can be taken for one of recurrent paralysis.

Pathology.—The causes of paralysis of the muscles supplied by the recurrent nerve or nerves fall into two groups. On the one hand there may be *central* disease of the nuclei of the nerves of the eighth pair, on one side or on both sides, in the pons; the affection then commonly forms a symptom of bulbar paralysis or of multiple sclerosis. Such paralysis is usually bilateral. Or, on the other hand, the disease may be *peripheral*, causing interference with the roots or trunks of the spinal accessory and pneumogastric nerves near the base of the skull, or with the trunk of the pneumogastric after its separation from the spinal accessory or with the recurrent laryngeal nerve itself below its origin from the pneumogastric. As a rule, the paralysis is in such cases unilateral. Indeed, by far the most frequent cause of paralysis of the muscles of one half of the larynx is aneurysm of the aorta, in which case the affection is most often on the left side. Aneurysm extending to the innominate artery may, however, compress the right recurrent nerve. Again, mediastinal growths of various kinds may interfere with the left nerve, while either the left one or the right may be pressed upon by an enlarged thyroid, or by cancerous tumours growing from the œsophagus. It is a further peculiarity of these last-mentioned peripheral causes that they not infrequently affect in succession both recurrent nerves, and produce bilateral paralysis. Dr Mackenzie has published a case in which there were two aneurysms of different parts of the aorta, one of which compressed the right and the other the left recurrent nerve. And in 1866 Dr Bäumler recorded a very interesting example of bilateral paralysis of the recurrent nerves, apparently due to the pressure of a large pericardial exudation.

It is, however, a more remarkable fact that peripheral interference with a single vagus sometimes causes precisely the same result. This, I believe, was first pointed out by Bäumler in the 'Pathological Transactions' for 1872; two years later Dr George Johnson made it the subject of a very interesting paper, which may be found in vol. lviii of the 'Med.-Chir. Trans.' The only possible explanation seems to be that irritation or possibly ascending neuritis is conveyed upwards to the bulb by the centripetal fibres of the affected vagus. Consequently, it is obvious that a lesion involving only the recurrent nerve and not the vagus itself must be incapable of producing the same effect, as was well shown by Dr Semon in the 'Berl. klin. Wochenschrift' for 1883. The fact that the paralysis set up by such an irritant action is bilateral, may be explained by the existence of a close physiological connection between the nuclei of the two sides, as is postulated by Dr Broadbent's hypothesis (see vol. i, p. 568). But Lockhart Clarke further showed that some of the fibres of origin of the spinal accessory nerve (which include the root-fibres of the recurrent laryngeal) actually pass across the middle

line, being derived from the opposite nucleus; perhaps, therefore, it is not inconceivable that disturbance of a single nucleus should directly cause bilateral paralysis. Whether any organic change develops itself in the nucleus or nuclei is as yet uncertain. The muscles on both sides become atrophied, as was clearly shown in Dr Bäumler's case. Indeed, there is no form of paralysis in which the resulting muscular atrophy is more obvious on dissection than paralysis of the muscles supplied by the recurrent nerve. In unilateral cases, in particular, the contrast between the whitish-yellow, shrunken crico-arytænoideus posticus on the affected side and the red fleshy muscle opposite to it is more striking than any similar condition in other parts of the body. The recurrent nerve also, below the point at which it is compressed, is greatly wasted and of a dull grey colour.

The *prognosis* of recurrent paralysis depends upon its cause. In Bäumler's case, in which it was dependent upon exudation into the pericardium, the patient, whose voice had been reduced to a whisper, recovered quickly as the effusion underwent absorption; within four or five weeks from the time when he first became hoarse he could speak nearly as well as ever. As a rule, the disease which causes the paralysis is incurable, and the paralysis itself persists until death. A goitre, if this is present, should of course be actively treated. It is generally useless to prescribe strychnia or to apply galvanic or faradic currents.

Paralysis of the Abductors of the Cords (the Crico-arytænoidei Postici).—Since recurrent paralysis is commonly the result of a morbid process gradually destroying the fibres of the nerve or the nucleus from which they arise, it is natural enough that cases should be met with in which some only of the muscles supplied by it suffer, while others escape. But what is a very remarkable fact is that such incomplete forms of paralysis should invariably (so far as is at present known) affect one particular muscle, the *crico-arytænoideus posticus*, of which the function is to keep the cords apart during respiration, and especially to widen the space between them on deep inspiration. Indeed, many cases have been recorded of paralysis of both recurrent laryngeal nerves, and yet no muscle has been affected, except the posterior crico-arytænoid on each side. Gerhardt published the first example of such an occurrence in 1863, and an admirable lecture on the subject by Riegel may be found in the second volume of 'German Clinical Lectures,' edited for the New Sydenham Society in 1877. But it is especially to Dr Semon that we are indebted for an adequate appreciation of the special proclivity of the abductor muscles to become paralysed. The reason for this proclivity is still obscure When there is a nuclear lesion in the bulb it may of course be accounted for on the supposition that the nucleus for the fibres to the abductors is really distinct from that for the other laryngeal muscles, just as we explain how in ordinary cases of bulbar paralysis the lower part of the face generally becomes affected while the upper part escapes. But the result is just the same when the disease is one which involves either the upper part of the vagus or the trunk of the recurrent nerve. The only possible explanation then seems to be that the fibres to the abductors undergo complete destruction earlier than those to the adductors. Riegel, indeed, actually found in one of his cases, in which both the recurrent nerves were embedded in dense connective tissue, that although the majority of the fibres had undergone fatty degeneration, some still retained their

normal structure. May we suppose that, on account of their relatively simple function, the crico-arytænoidei postici have a smaller nerve-supply than the rest of the laryngeal muscles, so that when the recurrent nerve is pressed upon there is, in the case of the muscles in question, no possibility for undamaged fibres to take up effectively the work of those which have been destroyed, whereas for other muscles having more varied functions and a richer nerve-supply, such substitution readily takes place? During the meeting of the International Congress at London, in 1881, Rosenbach pointed out that in paralytic affections of the limbs there is an analogous fact in the greater liability of the extensors and abductors than of the adductors to suffer. Perhaps, too, a further corroboration of such a view may be found in the fact that, when the laryngeal muscles are affected with spasm, the abductors are invariably overpowered by the adductors. This is certainly consistent with the hypothesis that the innervation of the latter group of muscles is better than that of the former.

The laryngoscopic appearances which characterise paralysis of a single crico-arytænoideus posticus are that the corresponding vocal cord lies more or less near the median line, and does not move outwards as it normally should when the patient takes a deep breath. On the other hand, during vocalisation it moves freely inwards. Hence one must bear in mind that unless the state of the larynx is carefully inspected while the patient is not, as well as while he is, attempting to utter a sound, this affection will necessarily be overlooked. When both abductor muscles are paralysed, the two cords lie nearer one another than they do in health. The degree to which they are approximated varies with the duration of the paralysis. In cases of long standing they may lie so close together that during inspiration it is scarcely possible to perceive the slightest chink between them; while during expiration they slightly recede from one another. Riegel insists on the fact that this extreme narrowing of the space between the cords is the result of a gradual contraction of the antagonists of the paralysed muscles, exactly analogous to that which occurs in paralytic affections of certain muscles of the eyeball, of the face, or of the limbs. But in a case recorded by Feith it seems to have come on within a few days from the commencement of the paralysis.

Another factor in the production of such a very marked stenosis of the glottis during inspiration is, however, in all probability a sucking in of the cords towards one another, in consequence of the diminution of atmospheric pressure upon their lower as compared with that upon their upper surfaces.

In two cases seen by Dr Semon there was a modification of the usual laryngoscopic appearances; the cords were close together only along their anterior two thirds, and diverged posteriorly so as to leave a triangular opening with its base at the inter-arytænoid fold. It is suggested that this depends upon a limitation of the paralysis to the outer fibres of the crico-arytænoidei postici, their inner fibres escaping. Rühlman has, in fact, endeavoured to show that each of these muscles consists of two portions having different functions; the inner portions, he says, draw the arytænoid cartilages downwards and outwards upon the cricoid; the outer portions rotate the arytænoid cartilages upon their vertical axis.

Apart from the results of a laryngoscopic examination, *subjective symptoms* of paralysis of a single crico-arytænoideus posticus are altogether want-

ing. The patient's voice is of course perfect, and as there is plenty of room for the entrance of air, he experiences not the slightest dyspnœa, even on exertion. It is this fact which gives its special importance to Dr Semon's observations of the invariable occurrence of paralysis of this muscle as the result of partial destruction of the recurrent laryngeal, or of the vagus trunk or nucleus. He has shown that there are a great many cases of aneurysm, of mediastinal growth, of carcinoma of the œsophagus, and even of disease at the base of the brain, upon which a routine use of the laryngoscope throws as much light as does the routine use of the ophthalmoscope in cases of cerebral tumour or of chronic Bright's disease.

On the other hand, when both abductors of the vocal cords are paralysed, the symptoms may be of the most urgent and dangerous character. The voice is still unimpaired unless there is a coincident inflammatory affection of the larynx. But there may be the most distressing inspiratory dyspnœa, the air being slowly drawn into the chest with loud stridor, and the patient becoming livid, with cold extremities, and ultimately dying of suffocation. In such cases the laryngoscope is needed, not so much to distinguish the affection from other diseases of the larynx as from stenosis of the trachea or of the main bronchi.

The mere fact that during inspiration the cords are drawn close together near the middle line is not of itself proof that there is any paralysis of the abductors; there may be a spasmodic affection of the adductors. What generally at once marks the difference is the way in which the dyspnœa begins. A primary spasm is sudden and rapid in its development; the contraction of antagonists which obstruct the entrance of air in cases of paralysis of the abductors comes on slowly. At first there is difficulty of breathing only when the patient makes some effort or exertion; then it gradually becomes persistent and increases in severity.

Another morbid condition from which paralysis of the crico-arytænoidei postici muscles has to be distinguished is ancylosis between the cricoid and the arytænoid cartilages, when the latter happen to be placed in such a position that the cords lie close together. Dr Semon speaks of the diagnosis as sometimes impossible.

With regard to the *causes* of paralysis of one or both of the abductor muscles of the cords, we have already seen it may be either central, or due to some affection involving the upper parts of the vagi, or the recurrent nerves in their course. In the 'Pathological Transactions' for 1882 a case is recorded by Dr Whipham, in which a bilateral paralysis of the abductors was dependent upon implication of the left pneumogastric and recurrent nerves in the walls of a thoracic aneurysm. In some cases this form of paralysis has followed diphtheria, and once it has been a sequela of facial erysipelas. Sometimes it has been ascribed to exposure to cold; and Dr Mackenzie thinks it is not infrequently due to direct pressure upon, or irritation of, the fibres of the crico-arytænoidei postici through the anterior wall of the pharynx in swallowing. Indeed, a case by Ott is cited, in which it was the result of the impaction of a piece of meat for twenty-four hours at the orifice of the œsophagus.

Prognosis and treatment.—In some cases recovery takes place after weeks or months, without its being obvious that treatment has been directly concerned in bringing about this result. Much more often the affection remains incurable. Sometimes a large amount of relief to the subjective symptoms, with temporary disappearance of the attacks of dyspnœa, may be attained

by the subcutaneous injection of strychnia ($\frac{1}{30}$th grain of the sulphate, gradually increased to $\frac{1}{10}$th, daily) or by systematic use of faradisation. The proper method of applying the current is by a laryngeal electrode, with a flat spade-shaped extremity that can be laid upon the pharyngeal mucous membrane, over the paralysed crico-arytænoid muscles. It is, however, very important that one should not be content with a partial success from this or any other plan of treatment. So long as the objective signs of stenosis of the glottis continue, there is always the risk of the sudden supervention of a fatal attack of dyspnœa, as in a case recorded by Dr Semon in vols. xi and xii of the 'Clinical Society's Transactions,' in which the patient's life was just saved at the last moment by tracheotomy, and by artificial respiration continued for three and a half hours. The rule is therefore laid down by him that the trachea should be opened without delay, as a measure of precaution, in every case of paralysis of the abductors, attended with considerable stenosis of the glottis and with marked dyspnœa, unless by some other method one succeeds within a short time in bring ing about not merely subjective relief, but also an actual enlargement of the opening.

Paralysis of the Adductors of the Cords.—There is, in every respect, the most marked contrast between paralytic affections of the abductors and of the adductors of the cords. They differ in their causes, in their symptoms, and in their course. A paralysis limited to the adductors is never due to organic lesions affecting either the fibres of the vagi or of the recurrent nerves, or their nuclei of origin. On the other hand, such an affection is not at all infrequent as the result of other causes, which seldom or never give rise to paralysis of the abductors.

The laryngoscopic appearances which accompany paralysis of the adductors vary somewhat according to the precise seat of the affection; for it is to be borne in mind that, instead of being (like the abductors) a single pair of muscles, the adductors consist of a group of muscles on each side of the larynx, any one of which may probably be paralysed separately. These muscles are classed together by Henle and Luschka under the name of the *sphincter sive constrictor rimæ glottidis*. Now, the pair of muscles which in this country are known as the thyro-arytænoidei, are commonly supposed to have the function of relaxing the vocal cords, thus acting as antagonists of the crico-thyroidei. But the German anatomists divide the muscles in question into two on each side, the "thyro-arytænoidei externi," and the "thyro-arytænoidei interni." The thyro-arytænoidei interni are described as a pair of prism-shaped muscles, each of which has one of its edges projecting into the substance of the corresponding vocal cord. Their function is to straighten and approximate the cords in the act of vocalisation. In other words, they co-operate with, instead of being opponents of, the crico-thyroidei; and they are sometimes spoken of as the "internal tensors," the crico-thyroidei as the "external tensors" of the cords.

If then the thyro-arytænoidei interni are paralysed, the effect is that when the patient attempts to speak, the cords, instead of being straight, are both concave, and enclose between them a narrow oval space. If the affection is unilateral, the space appears bounded by a straight and a curved line. The width of the space between the cords depends partly upon the pitch of the sound which the patient is trying to utter, being greater when the pitch is low than when it is high. For we must remember that every

degree of loss of power of the vocalising muscles may occur, from the slightest possible paresis up to the most complete paralysis.

On the other hand, an isolated paralysis of the arytænoideus muscle causes the rima glottidis to gape posteriorly, between the two arytænoid cartilages, while the cords themselves meet perfectly. The laryngoscopic appearance is then that of a triangle behind, with its apex prolonged into the normal narrow chink. Pieniazck, however, writing for Störk in his work, says that in some cases the ligamentous and the cartilaginous parts of the rima form two continuous straight lines, one on each side, there being a triangular interval between, which has its apex in front at the thyroid cartilage, and its base behind, beneath the posterior commissure.

Another laryngoscopic appearance sometimes observed is that the processus vocalis of each arytænoid cartilage forms an angle inwards; it is believed to represent a combination of paralysis of the thyro-arytænoidei interni with that of the arytænoideus.

An isolated paralysis of the two crico-arytænoidei laterales is said by Pieniazck to produce in the laryngeal mirror a figure with an angle outwards corresponding with the processus vocalis on each side, the rima being quadrilateral and lozenge-shaped. Ziemssen, however, doubts whether such an affection of these muscles ever occurs.

If all the muscles forming the sphincter rimæ glottidis are paralysed at the same time, the rima glottidis, when the patient attempts to speak, forms an oval space; but this is not bounded behind by the point of contact between the two processus vocales, as when the thyro-arytænoidei interni alone are affected; it extends backwards between the arytænoid cartilages to beneath the posterior commissure.

Lastly, according to Dr Mackenzie, it is possible to recognise by the laryngoscope a paralysis limited to the crico-thyroidei muscles which, of course, differ from all the rest in receiving their supply from the superior laryngeal nerves. The appearance which indicates such an affection is said to be that the rima glottidis presents a wavy outline.

It has still to be remarked that paralytic affections of some of the muscles forming the sphincter rimæ glottidis may be associated with spasm of others, and also that the exact seat of a paralytic affection may be different on the two sides of the larynx. So that it is sometimes difficult, or impossible, to determine the precise character of such cases.

Functional Disorders of the Voice.—*Aphonia.*—The main symptom in all cases of paralysis of the adductors of the cord is impairment of voice, extending from hoarseness up to the most complete aphonia, so that the patient may be utterly unable to speak except in a whisper. Whether such a total loss of voice is ever the result of the isolated affections of individual muscles seems to be doubtful. Ziemssen says that this effect can hardly be produced even by paralysis of the thyro-arytænoidei interni, so long as the crico-arytænoidei laterales and the arytænoideus remain in action, and bring together the processus vocales of the two arytænoid cartilages. Paretic states of the various muscles give rise not only to hoarseness, but to an undue sense of fatigue in speaking or singing, and to inability to maintain the voice for long or to shout.

On the other hand it is a striking fact that many patients who have complete aphonia nevertheless continue to be able to cough, and also to

sneeze, and that these reflex acts are accompanied with a laryngeal sound, which clearly proves that for this performance the cords can be perfectly well brought into contact. This is especially apt to be the case with hysterical women, who, in fact, are the most frequent subjects of paralysis of the adductor muscles. Dr Mackenzie says that he has met with such an affection in girls only eight or ten years old; but hysteria is by no means unknown at such an age. Phthisis is another disease in which paralytic aphasia is of rather frequent occurrence. Dr Mackenzie speaks of having in 1865 examined at the Brompton Hospital thirty-seven consumptive patients in whom the voice was affected, and of having found that in eleven of them "the affection was purely functional." Sometimes a paretic, or even a completely paralytic, state of the thyro-arytænoidei and of the transverse muscles follows an attack of laryngeal catarrh; it may then continue long after the mucous membrane ceases to show congestion. Dr Mackenzie says that this frequently occurs in public speakers, and especially in clergymen (*aphonia clericorum*). In other cases paralysis of the adductors of the vocal cords appears to be caused by the direct action of cold upon the affected nerve-twigs or muscles. Sometimes it is the result of over-exertion of the voice, as in singers; such cases are usually slight and transitory. But Dr Mackenzie speaks of paralysis of the thyro-arytænoidei interni as being occasionally the result of an actual "sprain" of the muscular tissues in some undue effort at vocalisation, and as then proving exceedingly intractable. Paralysis of the transversus (or of some of the other muscles) seems to be sometimes dependent upon gummatous or other lesions directly destroying the substance of the muscle. Dr Mackenzie also mentions poisoning by lead or by arsenic as possible causes of a paralytic affection limited to one or more laryngeal muscles; a case which he cites, and which occurred in a painter, is described as one of complete loss of power of "the adductor of the right vocal cord."

It is a peculiarity of hysterical aphonia, which (as we have seen) is dependent upon paralysis of some or all of the adductors of the cords, that the patient is apt to regain the voice suddenly under the influence of violent emotion. The recovery in such cases may be either transitory or permanent. Since the introduction of the laryngoscope it has become the usual practice to treat such cases by the application of a powerful induced current to the interior of the larynx, and this leads to brilliant success. The method is as follows:—One electrode is connected with a metal plate fastened upon a necklet which is put round the patient's neck so that the metal plate rests on the front of the larynx. The other electrode consists of a small metal ball or sponge fixed to the end of a long curved stem, which can be passed down into the space between the vocal cords. The stem of this "laryngeal electrode" transmits no current until the end of it has entered the larynx; at that moment the operator with his finger presses down a key by which the circuit is completed. All observers seem to be agreed that the only way by which it is possible to count upon a successful result from this procedure is that of using on the very first occasion a current of great power, which is of course exquisitely painful, and makes the patient involuntarily utter a loud articulate cry; whereupon the electrode is instantly withdrawn. If less than this be attempted at first, Dr Semon finds that electricity often fails altogether. Dr Mackenzie speaks of having cured cases of six, seven, eight, and even ten years' standing. Sometimes it is necessary to repeat the application of the induced current

several times and during many weeks before a permanent result is attained. In some cases, however, the introduction of an electrode within the larynx is not required; it is sufficient to apply a current across the neck from one side to the other, the electrodes being placed one over each thyroid cartilage. Dr Mackenzie speaks also of having occasionally succeeded with stimulating steam-inhalations made with oil of Calamus aromaticus, or with creosote; or he has used a strong solution of nitrate of silver (ʒj ad ℥j) applied with a brush or in the form of spray.

Alteration of the pitch of the voice.—To complete the description of functional disorders of the voice certain cases must be mentioned in which there is an alteration in its pitch. Störk relates instances of children with an unduly low voice, and others of young men, with an excessively high pitch, after their voice has broken at puberty. The remedy, in the former case, is to practise speaking with a falsetto voice; in the latter, with a bass voice. This is often perfectly successful, if sufficient perseverance be shown. Sometimes the desired change in the pitch of the voice is brought about very rapidly; for instance, a young man, aged eighteen, who for about a year had spoken in a falsetto voice which contrasted ridiculously with his broad and well-built figure, was told to utter the vowel *u* for an hour daily in as deep a voice as possible, at the same time holding the head fixed, this last direction being merely for the purpose of keeping up his attention; on the fourth day his voice became normal, and from that time it remained so.

Spasm of the tensors of the vocal cords is another curious affection. This is characterised by a state of the voice so peculiar as to be at once recognised by those who are familiar with it. The following is Dr Mackenzie's description: "The patient is often able to produce some notes, either in his own natural voice, or in a slightly muffled tone; but, while he is speaking in this way, the current of the voice seems to be partially interrupted, and the sound conveys the idea of an arrested action of the respiratory muscles. In fact, it is very much like the straining and rather suppressed voice of a person engaged in some act requiring the prolonged and steady action of the expiratory muscles (parturition or defæcation). The patients often complain that they cannot get their voice out. After speaking a word or two, or even several sentences, in this peculiar tone, the patient may again utter a few words in a comparatively healthy voice, and then may immediately relapse into the diagnostic intonation." Or there may be a complete absence of sound, the lips moving in the usual way for the utterance of words and phrases, which nevertheless are lost in silence. A clergyman is described as having been greatly distressed by the fact that while he kept on reading the service some of the words dropped soundless from him.

Dr Mackenzie, in 1880, had seen only thirteen cases of this kind; eleven of them were in men, ten being clergymen, and the eleventh a barrister; two were in women, both of whom had had constantly to speak to deaf relatives. Doubtless, therefore, it is the result of over-use of the voice. The onset of the affection was sometimes gradual, sometimes sudden; in the latter case it was attributed by the patients themselves to "catching cold." No treatment was permanently successful in any instance.

Laryngismus Stridulus.*—Obstruction of the glottis by spasm is present in many diseases and not in one only, and in this disease there is, or may be, spasm of many other muscles besides those of the larynx. Hence it is both less and more than spasm of the glottis. Laryngismus stridulus in its extreme forms exhibits an organised series of phenomena, comparable only with those of an epileptic fit. As we shall presently see, there have been doubts as to whether it is dependent on a primary disturbance of the central nervous system, or whether it is reflex and due to peripheral irritation. But however this may be, its proper place in the nosology is certainly among the neuroses, where it is placed in the last edition of the College of Physicians' 'Nomenclature of Diseases,' and the only reason for discussing it in this place is that clinically its symptoms have to be studied in relation with those of laryngeal diseases. The name of laryngismus stridulus was invented by Dr Mason Good, and has since been very generally adopted; that of "child-crowing" was proposed by Dr Gooch. At one time it was called "thymic asthma," under the idea that it was caused by enlargement of the thymus. This hypothesis was maintained by Kopp in 1830, but was disproved by Bednar in 1852, and by Friedleben in 1858.

Symptoms.—In its simplest form laryngismus stridulus consists of a disturbance of the natural rhythm of the respiration, such that the child (for the disease is one of childhood) first holds its breath and then makes a more or less noisy inspiration. This occurs again and again at varying intervals —perhaps especially on the first waking from sleep—but neither parents nor nurses may attach much importance to it, thinking it is merely a "catching in the breath." Gradually, the paroxysms assume a more serious character, or they may have begun in a severe form. The child shows signs of great distress and alarm. Its neck and back become arched, its chest and abdomen rigid, its eyes turned upwards, and its limbs tonically contracted, the thumbs being bent inwards, the fingers extended, and the wrists flexed, while the legs are thrust out, the soles turned inwards, and the toes stretched wide apart. Its face, at first pale, may turn purple, or of a ghastly leaden colour. Sometimes the fæces and the urine are discharged involuntarily, sometimes there is a noisy expulsion of flatus. After a few seconds, or a minute or two at the longest, the spasm yields. In all probability while it lasts, the glottis is completely closed. As it passes off a chink is formed, through which the air can slowly enter, making a loud crowing sound. This usually ends the seizure, but sometimes two or more paroxysms occur in rapid succession. According to Steffen, in 'Ziemssen's Handbuch,' a few unrhythmical and noisy expirations, and one or more whistling or crowing inspirations, precede the stoppage of the breath which constitutes the central feature of the attack. After the paroxysm is over the child frets or cries for a little while, or falls asleep; or, it may at once seem to be as well as ever, returning with as much zest as before to its toys, or to any game in which it may have been engaged.

Pathology.—It is clear from this description that many other muscles besides those of the larynx are concerned in the production of attacks of laryngismus stridulus. The preliminary stoppage of breathing may be perhaps attributed to a mere inhibition of the respiratory centre, rather than to spasm. Steffen says that with few exceptions the diaphragm and the chest walls are "in the inspiratory position" when the pause occurs, but he can

* *Synonyms.*—Spasmus glottidis—Spasmodic Croup—Child-crowing—Thymic Asthma —Millar'sches Asthma.

hardly mean that they are in the position which they normally occupy when inspiration is completed; for if so, how is the prolonged inspiratory movement that immediately follows to be satisfactorily accounted for? Even as to the part played by the intrinsic muscles of the larynx at the time when the crowing inspiration takes place there has been some difference of opinion. Some writers have doubted whether there is only (as is generally supposed), a spasm of the sphincter of the glottis, and have thought that there must be also paralysis of the opposing crico-arytænoidei postici. Now, in experiments on rabbits it is found that if the superior laryngeal nerve on one side is divided, and its central end is then excited by a faradic current, the result is a strong bilateral adduction of the vocal cords. But if stimulation of the centres in the bulb thus normally tends to evoke adduction, the only question that remains is whether, when it occurs, the nerve-fibres to the abductors are left out of the reflex circle, or whether these muscles are overpowered by their antagonists. The preponderant action of the sphincter seems best explained by the latter hypothesis, supported as it is by the facts already mentioned with regard to the relative liability of the two groups of muscles to paralysis.

Fatal event.—But it does not always happen that a seizure of laryngismus stridulus ends favourably. Sometimes, instead of relaxing, the spasm persists until life is extinct. There is then, of course, no crowing sound, and there may be nothing whatever to indicate the cause of a death for which the parents are utterly unprepared. Some years ago the author made an autopsy on the body of an infant aged sixteen months, which had died suddenly and had been brought to the hospital by its mother. Nothing was found to account for such an occurrence, but on inquiry next day it was found that the child had previously had attacks of "child-crowing." Steffen relates the case of an infant six months old, who was one night taking the breast when it was attacked with slight spasm of the glottis, after which it went on sucking. However, the attack returned more severely, and the child fell backwards. Within a quarter of an hour Steffen was at the spot. The child had been laid in its cot under the idea that it was sleeping. He found it livid and dead, without any signs of spasm of the limbs or of any other part than the larynx. Sometimes, however, death is preceded by tremulous twitchings of muscles, or by a more or less complete epileptiform convulsion, exactly as is the case in other forms of asphyxia.

As may well be supposed, after death from laryngismus stridulus the brain and its membranes are found gorged with blood, but there is no reason whatever for imagining that the congestion is antecedent to the paroxysms or concerned in producing them. When the disease has been of some standing the lungs may, according to Steffen, be found emphysematous; if this is really the case, in uncomplicated instances of an affection in which there is only inspiratory dyspnœa, it has an important bearing on the theory of emphysema in general.

Ætiology.—The most obvious fact in the ætiology of laryngismus stridulus is its relation to *rickets*. According to Steffen, at least nine tenths of all cases occur in rachitic children. By Elsässer it was thought that craniotabes was in a special way associated with the development of laryngismus, but this appears not to be the case. It is doubtless in consequence of their having all in turn been sufferers from rickets that laryngismus has been sometimes noticed in several successive offspring of the same parents. Dr Reid mentions a family of thirteen, of whom only one escaped laryngismus and four died of it. Children affected with laryngismus are not infrequently fat, so that their parents fondly imagine them to be hearty and

strong; but this, as is well known, is quite compatible with their being highly rachitic. The relation of laryngismus with other rachitic spasmodic neuroses, tetany and carpo-pedal contractions, has been already set forth (vol. i, p. 738).

A curious point, noticed by Henoch, is that laryngismus stridulus is of far more common occurrence in the early part of the year than later on. Dr Gee, among sixty-three cases, observed no fewer than fifty-five between the months of February and June inclusive. His explanation is that the extent to which children are kept indoors during the winter increases the irritability of their nervous centres.

The *age* at which laryngismus stridulus begins is generally from four months to two years. But Dr Reid relates cases in infants only a few hours after birth; and others occur in children of various ages up to nine years.

There is, however, one remarkable fact which shows that rickets can only be looked upon as a predisposing cause of laryngismus, namely, that the latter affection is much more frequent in boys than in girls. Steffen cites figures from different observers, showing that the proportion of males to females is higher than that of two to one. In relation to it he alludes to the circumstance that membranous croup also is far more commonly seen in male children than in female; and this is still more markedly the case with false croup. Such an indication of a relationship between laryngismus and other diseases of the main air-passages no doubt tends to make one hesitate in assigning a principal place in its pathogenesis to an over-excitability of nerve-centres in the bulb.

What, however, are the facts as regards the occurrence of tetany and carpo-pedal contractions in children of the two sexes? On reference to some of the recently recorded cases of tetany it appears that the patients have been chiefly boys. And although chorea is well known to be more common in girls than in boys, one must remember that its seat is probably not in the bulb, but in the large basal ganglia; and, moreover, that it occurs at a later age, when the course of development may probably have altered the relations between the two sexes as regards the susceptibility of the nervous centres. In this connection it is perhaps worth noting that many more males than females, whether children or adults, succumb to tubercular meningitis.

Whatever may be the real bearing of these facts upon the ætiology of laryngismus, we may without hesitation reject another hypothesis which a few years ago had many supporters, namely, that it is necessarily of reflex origin and dependent upon some irritation conveyed upwards to the nervous centres from the periphery. This was declared by Dr Marshall Hall to be the "only true mode of viewing" the disease, and he maintained that the cause of it was almost always to be found in a morbid state of the teeth, in disorder of the stomach, or of the bowels. A few years earlier, Dr Hugh Ley had endeavoured to prove that laryngismus was the result of mechanical irritation of the vagi nerves by enlarged bronchial or cervical glands. He did in fact show that glandular enlargement was often present. But it certainly may be absent, and no morbid anatomist who has learnt how frequently in autopsies upon children the vagi nerves are found surrounded by caseating glands, will assign to them any important share in its ætiology. When the nervous centres are in a morbid state their successive explosions, due mainly to a gradually increasing irritability of their cells, may be directly brought about by stimuli conveyed to them from internal organs, just as in severe cases they can be excited by merely touching the surface of the child's body. The

analogy of epilepsy and of many other neuroses is entirely in favour of such an opinion. Even then, however, the fact that what must be a constant stimulation produces only paroxysmal effects shows how preponderant is the influence of the varying state of the nervous centres.

Diagnosis.—It may almost be said that there is no other affection with which laryngismus stridulus can be confounded by those who know its symptoms. At the moment of an attack, if there had been none previously, or if the child's history were unknown, one might suppose the obstruction of the larynx to be due to a foreign body. It would be quite right to pass one's finger to the back of the throat to settle the question; but the subsidence of the spasm (unless it proved fatal) would very quickly show the real nature of the disease.

Prognosis.—The natural course of laryngismus stridulus, when undisturbed by treatment, varies greatly in different cases. Sometimes the attacks continue to be slight, and occur at wide intervals; sometimes they increase in severity and in number, until there may be thirty or forty of them in the twenty-four hours. In either case they may after a few weeks gradually become less frequent, until at length they cease entirely. Different writers have expressed different opinions with regard to the proportion of deaths to recoveries, some saying that one case in every two or three proves fatal; but if mild and severe cases be reckoned up together, the prognosis is generally favourable. It is, of course, better when the child is not very young; and according to Steffen it is better in girls than in boys.

Treatment.—The treatment of laryngismus stridulus is, in the first place, that of rickets; sunlight, fresh air, good food, cod-liver oil. A change into the country or to the seaside is often quickly followed by the subsidence of the attacks. Dr Ringer strongly advocates sponging with cold water twice or thrice daily. Of drugs, the most trustworthy is bromide of ammonium, of which from three to five grains may be given as a dose, even to a young child. Many find syrup of chloral hydrate the most efficient medicine; it is readily taken by young children and is perfectly safe. It may of course be combined with bromide.

It is quite right to look out for any morbid affections of distant parts that may possibly be concerned in irritating the nervous centres. If the gums are hot and tense, they should be lanced. If the bowels are loaded, a few aperient or vermifuge doses ought to be given. But it will not often be found that great results are attained by such measures, either in diminishing the severity or in reducing the frequency of the seizures.

When a paroxysm is so prolonged as to require treatment, cold water may be dashed over the face and head, the body being perhaps immersed in a warm bath; or a bottle containing ammonia may be held to the nostrils. The inhalation of chloroform is recommended by some writers. Faradisation of the phrenic nerves seems likely to be useful. Even if life should have apparently become extinct, it may sometimes be restored by artificial respiration, as was pointed out many years ago by Mr Johnson in the fifth volume of the 'Dublin Hosp. Reports.' In all severe cases the nurse should be taught beforehand how to act should the emergency arise.

Inflammatory affections of the larynx.—They differ widely as to their exact seat, their symptoms, and the course which they run. It is therefore necessary to classify them in some way, and one obvious distinction among them lies in the fact that some mainly affect the mucous membrane, whereas others

start in the deeper structures of the larynx. Again, inflammations of the laryngeal mucous membrane fall naturally into two groups: first, cases attended with dyspnœa so severe that it threatens and often destroys life; these are commonly known as cases of *croup;* secondly, cases in which the chief symptom is impairment of the voice; these commonly pass under the name of *catarrhal laryngitis*, either *acute* or *chronic*.

CROUP.*—In 1765 Dr Francis Home, of Edinburgh, published in a short tract of sixty pages, an 'Inquiry into the Nature, Causes, and Cure of the Croup,' a disease which he declared to be entirely unrecognised by medical writers, although it was known to the common people of Scotland by several distinct names, of which "croup" is one. In each of the *post-mortem* examinations which he made he found the trachea lined by a more or less complete membranous layer. Thirty-six years later, in 1801, another Scotch physician, Dr John Cheyne, wrote on the same subject a work which has become classic; his views upon the pathology of croup were the same as those of Home. In the meantime the existence of the new disease had become matter of common knowledge in England as well as on the Continent. But certain other affections of the throat were also known, the relations of which to Home's disease are discussed by his successor, so that in Cheyne's work one may discover the rudiments of a controversy which has of late years attracted much notice, and which even now is not finally settled.

There are two questions, separate and yet closely connected with one another: (1) whether the membranous "croup" of the Scottish writers is distinct from the disease which has since been called *diphtheria;* (2) whether it is distinct from a milder affection of the air-passages, unattended with the formation of false membrane, and variously named *false croup, stridulous laryngitis, spasmodic laryngitis,*† or *inflammatory croup.*

To each of these questions an answer must be given before we can pass on to consider the clinical history of croup, and we will begin with the relation of the disease to diphtheria.

(i) The controversy in regard to this point was definitely commenced by Bretonneau, of Tours. In his earlier 'Memoirs on Diphtheria,' 1821–26, he made it his principal object to prove that that disease and croup are identical. His view was in due course adopted by his pupil, Trousseau, and by Guersent, Barthez, and almost all the other leading French physicians. In England it was for a long time repudiated by every medical writer, but within the last few years it has met with a much more favourable reception. The late Dr Hillier advocated it in 1862, and since then Dr Johnson, Dr Semple, and Sir John Cormack have maintained it, and Sir William Jenner has withdrawn his previously expressed opinion that the two complaints are distinct.

Now, in the first place, we must remember that both Home and Cheyne were perfectly acquainted with the fact that the disease which they described was liable to be confounded with one which affected the larynx secondarily, having its original seat in the fauces. The former of these writers, quoting Dr

* *Synonyms.*—Cynanche trachealis—Cynanche stridula—Angina suffocatoria—Morbus strangulatorius—*Fr.* Angine couenneuse trachéale ou membraneuse: le croup—*Germ.* Häutige Bräune: der Croup.

† By an unfortunate confusion some writers have designated as "spasmodic croup" the entirely different complaint "*laryngismus stridulus,*" described above.

Hare's graphic account of the "morbus strangulatorius" in Cornwall (which was epidemic diphtheria in its most typical form) says that that complaint "appears more nearly allied to the malignant sore-throat, although it sometimes attacked the trachea." And the latter, Dr Cheyne, begins his section on diagnosis by remarking that he had seen several children, whom he would have supposed to be suffering under the second stage of croup had he not discovered sloughs upon the tonsils and uvula. Probably each of these observers had better opportunities of studying the relations of the two complaints than any London physician at the present day; and it is worthy of notice that if they should prove to have been wrong in regarding them as distinct, the progress of medical science will in this instance lead to a result directly opposite to that which it is bringing about in other cases; for in regard to most other diseases the more our knowledge advances the more are distinctions multiplied.

One argument in favour of the identity of membranous croup with diphtheria may be first met, for stress is laid on it by the authority of Sir William Jenner. It is said that mucous membranes do not, like serous membranes, pour out lymph upon their surface when inflamed by simple irritants, so that, it is urged, an affection of the larynx attended with the formation of false membranes must be a specific inflammation. But, both for the fauces and the air-passages, it is certain that this inability is far from being absolute. In the 'Guy's Hospital Reports' for 1877 (p. 384) seven cases are recorded by the author, in which those parts presented appearances indistinguishable from diphtheria, but as the result of scalds of the throat by boiling water, which the patients (always children) had sucked from the spout of a teapot or kettle; one case of a boy, who got a bean into his right bronchus, and who had his larynx and trachea coated with lymph; two cases (already referred to in vol. i, p. 266) of children who had had their fauces irritated, one by a piece of hot potato lodging in the throat, the other by a burning stick, and in whom the morbid action took the same form; one case of a man aged twenty-four, who was admitted for a cut throat, and who died of a plastic inflammation of the larynx, trachea, and bronchial tubes; three cases in adults in which a membranous laryngitis was secondary to cancer of the pharynx, tubercular ulceration of the vocal cords, or syphilitic disease, for which tracheotomy had been performed; and, lastly, two cases, both in adults, in which a similar affection was associated with an acute or a chronic pneumonia—sixteen in all. Many of the patients, indeed, had had tracheotomy performed some days before death, and it might be plausibly argued that a badly-cleaned tube, if it had before been used for a case of diphtheria, might conceivably have inoculated the parts with the specific disease. This, however, would not apply to some of the cases, and it is also inapplicable to one recorded by Mr Parker ('Clin. Trans.,' 1875) of a child who had scalded its throat with hot water, and from whose trachea false membranes were drawn up by means of a feather almost immediately after the operation. Moreover, both Rietz and Oertel have found it easy to set up a plastic inflammation of the trachea in dogs and rabbits by dropping a few minims of Liquor Ammoniæ into it through an external wound. Oertal says that he performed this experiment on seventeen animals, and succeeded in every instance in generating an artificial croup.

On the other hand, some arguments have been adduced in favour of the distinction between croup and diphtheria, which have now been shown to

be untenable. Thus, it was long believed that the false membranes presented constant differences of microscopical structure and of chemical constitution in croup and in diphtheria, but we now know that in diphtheria itself they vary in their appearance, in the relation which they bear to subjacent parts, and even in their histological characters, according to the part of the mucous tract upon which they are developed. This fact necessarily involves the overthrow of all the histological distinctions that had been drawn as between the two diseases. A single point of difference is still declared to exist by Oertel. He believes the presence of micrococci to be essential to diphtheria, but in the false membranes which he set up artificially in animals by dropping Liquor Ammoniæ into the tracheæ, he found micrococci in small numbers, and only in the more superficial layers. He therefore argues that the criterion of a simple non-specific croup is the absence of these organisms in the inflammatory products. Now, we saw reason for believing that the micrococci which are generally present in diphtheritic membranes are far from possessing the importance which Oertel attributes to them (vol. i, p. 262); but their non-occurrence in croup might still be distinctive of that disease. As a matter of fact, however, other observers have failed in cases of diphtheria to detect the parasites in false membranes below the glottis. It seems impossible to avoid the conclusion that their presence, so far from being a necessary part of the diphtheritic process, is rather an indication that it is of an unhealthy character, or that the false membranes themselves are undergoing putrescence. The laryngeal form of diphtheria perhaps kills too quickly for such indications to be manifested.

This brings us to another distinction between diphtheria and croup, on which some observers have laid great stress, but which seems to be capable of ready explanation. It is the clinical fact that marked symptoms of depression of the vital powers—a dry, brown tongue, sordes on the lips, petechiæ on the skin, hæmorrhages upon the internal serous surfaces—are present in the former, but absent in the latter affection. In many cases of diphtheria, however, no such symptoms show themselves until several days have elapsed, and since croup destroys life rapidly by the mere effects of the presence of false membranes in the air-passages, one could not reasonably expect that it should be attended with indications of depression and of septicæmia, even if it were a modification of diphtheria.

Again, the fact that a definite exposure to cold has immediately preceded and apparently excited an attack of membranous laryngitis, seems to be no proof that the case is not one of diphtheria. At least Sir William Jenner says that he has seen cases which arose in this way, and which he believed to have been diphtheria, because albumen showed itself in the urine; and in the chapter on diphtheria (vol. i, p. 266) Dr Yeats's observations are recorded (which seem to refer to faucial diphtheria occurring in adults) as to the frequency with which, during an epidemic, those persons were attacked who had immediately before been exposed to the night air. What, we may ask, was the nature of the disease in Dr Gregory's twin children, who (as Sir Thomas Watson relates) were both seized with croup on the same night, after walking together in the evening sunshine and in a cold wind?

We must allow that no criteria based either upon morbid anatomy or upon clinical symptoms avail to distinguish laryngeal diphtheria from croup, that is to say that the cases referred to by Sir William Jenner

and others, in which diphtheria is limited to the air-passages, are really undistinguishable from croup. But this is, nevertheless, far from proving the identity of the two diseases. For it is probable (vol. i, pp. 271, 272) that, whereas in *epidemics* laryngeal diphtheria is rare, laryngitis and tracheitis is of common *sporadic* occurrence. Now, this is in itself difficult of comprehension, if we suppose the latter affection to be a form of diphtheria. One could perhaps understand that a laryngeal diphtheria might be less contagious than one affecting the fauces, as being further from the surface of the body, and so one might account for its being less apt to be the starting-point of an epidemic. But this would by no means cover the fact that while isolated cases of membranous laryngitis are frequent, a laryngeal diphtheria is rarely set up by the direct action of the specific contagion of the disease. In Guy's Hospital it has never happened, when a person admitted for diphtheria has communicated the disease to other patients in the same ward, that the morbid process in those patients has limited itself to the air-passages.

The question at issue, however, is not to be so easily disposed of. Cases in which there is an affection of the fauces, such as would be commonly called diphtheria, are much less often traceable to contagion when the air-passages are affected than when they escape. There seems to be a regular descending scale of contagiousness, according as the morbid process falls with less intensity upon the tonsils and palate and with more intensity upon the larynx and trachea. Moreover, whereas diphtheria attacking the fauces is common in adults, all the cases at Guy's Hospital in which these parts are but slightly affected, and the brunt of the disease has fallen upon the air-passages, have been in children below five years of age. Now, it is very improbable that such differences should exist, either as regards the contagiousness of the disease, or as regards its occurrence at a special period of life; but one sees at once that the recognition of these differences is exceedingly favourable towards the inclusion within the domain of diphtheria of cases in which the larynx and trachea are alone attacked, the tonsils and palate remaining free. For the latter cases are likewise peculiar to children, and their non-contagiousness is the very point on which the whole discussion turns.

Is it not preferable to adopt another solution of the difficulty which involves no such improbabilities? It is to assume that the greater number of the cases referred to at p. 19 were really not examples of diphtheria at all, although the fauces were affected. After all, it seems an absurdity to draw a fixed line at the edge of the epiglottis, and to say that so long as an inflammatory process is limited to the parts below it, the case may be one of simple membranous croup, but that if it spreads above this line, it must be due to the specific poison of diphtheria. No doubt the great difference in histological structure between the mucous membranes of the larynx and pharynx appears favourable to such a view. But we know that, whether in diphtheria or after a scald of the throat, no obstacle to the *descent* of an inflammatory process is offered by this difference in structure. Why then should it be a bar to the *ascent*, when the air-passages are first attacked?

(ii) The next question is, what relation towards the membranous croup of Home and Cheyne is borne by those cases—of not infrequent occurrence—in which the air-passages are less severely affected, so that no false membranes are found? Now, all writers of the present day describe certain

cases of this kind as entirely distinct, and give to them a special name. Bretonneau is generally supposed to have been the first observer who indicated clearly the points of difference, and his name for the cases in question was "stridulous angina." In reality, however, the English writers of the end of the last century were well acquainted with the clinical history of the affection, which was known to them by the designation of "spasmodic croup" or "spurious croup." Its peculiar characters are fully set forth in a paper which Mr Field, Secretary to the Medical Society of London, read before that body in 1796.

The most distinctive features of spasmodic or spurious croup are the suddenness of its onset and the alarming nature of the symptoms which it presents from the very commencement. A child who is apparently in perfect health, or who may have had a slight cold for a day or two, goes to bed without any sign of laryngeal affection, and falls asleep as quietly as usual. About eleven o'clock, or at midnight or a little later, he suddenly starts up in a state of extreme excitement and terror. He coughs incessantly, making a hard, hoarse, barking noise. He pants for breath, and each inspiration is attended with a loud crowing sound. His voice is hoarse and it may be very feeble, but it is not whispering as in true croup. His face, at first flushed, afterwards becomes pale and covered with a cold sweat. The nurse and parents are horror-stricken and send at once for the nearest medical man. But, instead of the child getting worse, each paroxysm of coughing is rather less severe than the preceding one. And after half an hour, or in two or three hours, he becomes calm and sleeps. In the morning, when he wakes up, his cough his still hoarse and barking, but it is not so hard; his respiration is attended with little or none of the whistling sound; his voice has nearly regained its natural tone. During the day the child is as cheerful as before and has but little cough; his pulse is not accelerated; he is scarcely, if at all, feverish. On the following evening, however, the symptoms often return, sometimes as severely as at first, but generally less so. They may even repeat themselves for several nights in succession with gradually diminished intensity.

It is doubtful whether attacks of this kind ever prove fatal. Trousseau speaks of having seen three cases in which death occurred. But the only one of which he gives details is that of a schoolboy, thirteen years old, who was suddenly seized with dyspnœa on waking in the morning, and who seems to have died at the end of about four hours. On *post-mortem* examination the laryngeal mucous membrane was found to be reddened and the arytænoepiglottidean folds were a little swollen; the vocal cords were a good deal swollen, and on one of them "there was a slight membranous concretion, possessing, however, none of the characters of diphtheritic false membrane." It seems to me that in this case a severe inflammation of the larynx would probably have developed itself if the patient had lived a little longer. It certainly was not a typical case of spurious croup as regards the time at which the attack began, and the boy was much beyond the age at which that affection is most apt to occur.

At present, therefore, the pathology of spurious croup is matter of inference only. But there can be little doubt that it depends upon a slight catarrhal inflammation of the laryngeal mucous membrane, complicated with spasm of the muscles of the glottis.

Another feature of this complaint is its tendency to recur again and again in the same individual. A child who has once had it is always likely

to be attacked a second time if he is exposed to cold or wet; and up to the age of fourteen or fifteen every slight catarrhal affection is apt to be accompanied with the peculiar hard barking cough. When one hears that a person suffered from croup repeatedly during childhood, one may pretty safely conclude that the disease was "spurious."

Now, Cheyne, in the second edition of his great work, discusses at considerable length the relation between the disease of Home and this "spurious" or "spasmodic" affection, with the description of which he was familiar through the writings of Field and of Ferrier, a physician of Manchester, who had published an essay on the subject in 1810. And he comes to the conclusion that there are no just grounds for admitting two kinds of croup. The affection in question "occurs," he says, "in those families which are subject to genuine croup; it arises from the same exciting cause (exposure to cold); it prevails during the same weather." And he goes on to point out that in many cases in which the breathing afterwards becomes permanently affected, the symptoms are for the first few days most marked during the early part of the night, the patient in the daytime seeming to have nothing the matter with him with the exception of a cough. But it must be admitted that there are two criteria which point very strongly to the existence of spurious croup as a separate member of the nosology. One is the sudden onset of the complaint with all its symptoms in full force, whereas the affection attended with the presence of false membranes comes on more or less gradually. The other is its liability to return again and again in the same individual, whereas membranous croup seems never to attack a child more than once. If spurious croup were merely an undeveloped or milder variety of the disease, one would expect that persons who were subject to it would be very apt, on some occasion when the exciting cause happened to be powerfully in operation, to have it in its severe form, and to die with membranous exudation into their air-passages. Yet it is a question whether a single instance has occurred.

There can be no doubt, therefore, that Cheyne did include in his description of croup cases which were really distinct from those in which false membranes were found in the larynx and trachea. And, indeed, it would seem that every case of which he gives the details, and in which a fatal termination did not occur, happened to be an example of the spurious form of the disease.

But it is quite another question whether all or even most of the non-fatal cases of croup that one meets with in practice are to be placed in the same category. Trousseau says that although "stridulous laryngitis" (as he terms the affection) is very common, he had had only one case in his wards at the Hotel Dieu, a principal reason for this being the sudden way in which it declares itself, and the rapidity with which it yields, so that children attacked by it are very seldom brought to hospitals. But in Guy's Hospital, between the years 1867 and 1876, there were admitted ten cases of croup at least (and probably several more) in which recovery took place, and in which there was no proof of the formation of false membranes. In many of these cases the symptoms were continuous for some days in succession; and the clinical reports very seldom say anything about previous attacks of a similar kind. Perhaps it may be that in London a form of non-membranous croup is of frequent occurrence which in Paris is not met with. At any rate it is a striking fact that Dr George Johnson—who is anxious to draw a sharp line of distinction between the cases which present

false membranes (which he regards as examples of diphtheria) and those in which no such membranes are found (which cases he terms "infantile laryngitis" or "inflammatory croup")—is obliged to extend very greatly the definition of the latter affection beyond the narrow limits which had been set for it by the earlier English and by the French writers. In fact, in the last edition of Sir Thomas Watson's 'Lectures' (in which Dr Johnson's views are adopted) the whole description of "croup" is transferred bodily to the new "infantile laryngitis." This is no longer the harmless affection of Bretonneau and Trousseau, but a disease which "proves fatal sometimes within twenty-four hours and often within forty-eight hours," and which "may continue for five or six days before it terminates." Now, so far as I can ascertain, there has at Guy's Hospital in the course of many years past been only one case which has ended fatally, and in which on *post-mortem* examination false membranes have been absent. But, as already remarked, it often happens that there is no evidence of their presence during life. It therefore seems probable that the systematic performance of autopsies would lead any believer in the view that all membranous croup is diphtheria to transfer to the latter disease almost all of his fatal cases. But surely it is without precedent in pathology to draw what is in fact an arbitrary line of distinction between those cases of a disease in which recovery takes place and those which prove fatal. There can be little doubt that in many cases of croup which recover, false membranes are really present in the air-passages, even though none may be expectorated; probably they may become disintegrated in the more fluid products of the inflammatory process and disappear; or they may be hawked up into the mouth and swallowed.

To sum up: it seems clear that no fixed line can be drawn between membranous croup and the milder forms of the disease in which no false membrane is developed, except that those cases in which it occurs over and over again—with sudden and alarming but very transitory symptoms—may be separated under a distinct designation, for which purpose the name of *spurious croup* seems to be the most appropriate. As regards the relation of diphtheria to membranous croup, it appears impossible to maintain the position which some writers formerly took, that the presence of false membranes upon the fauces proves a case to belong to the former disease. Probably in rare cases diphtheria may begin in, and remain limited to, the air-passages, but it more often happens that a non-specific membranous croup extends to the tonsils and palate, leading to the formation of milk-white patches of greater or less size upon the surface of these parts. It is an instructive fact that in two out of eleven cases of "idiopathic croup" which came under Dr West's care between 1839 and 1849, when diphtheria (at least in an epidemic form) was not prevailing in this country, there was "a scanty formation of false membrane upon the velum and tonsils." No doubt, in an individual case, the possibility that the disease may be diphtheria can never be absolutely negatived. The most important points against it would be the absence of a history of contagion, the circumstance that no other person in the house or in the neighbourhood had had anything that could possibly be set down as diphtheria, and (with due allowance for the facts stated at p. 19) there having been a direct exposure to weather or to some considerable change of temperature immediately before the commencement of the attack.

It must be admitted that the opinions above expressed are antagonistic

not only to those of French and of some English writers,* but also to those of the German physicians who have published the most recent articles on the subject. All these do, indeed, theoretically admit the existence of a simple laryngitis and tracheitis attended with the formation of false membrane. But in practice they seem to assign to diphtheria almost all the cases which come under their observation.

It is an important question whether this may not depend upon the fact that on the Continent non-specific membranous croup is really a much more rare disease than in England. Cheyne makes the assertion that it is far less known in the temperate than in the northern regions of Europe; but one can hardly tell on what facts such a statement is based. Even in Great Britain, the disease appears to be irregular in its distribution. Home states that in his time it was far more frequent in Leith and Middleburgh, which are near to the seashore, than in Edinburgh, and in that city Dr Alison found it most prevalent in those parts which are lowest in situation. Wet and marshy spots are said to be favourable to its occurrence. Some of the medical men practising in Norwich say that it is unknown in that city, which has a very dry and bracing air, although it is much exposed to cold easterly winds.

Symptoms.—Croup commonly begins as a catarrh of but slight severity. The child is noticed to be a little feverish, refusing its food, but asking frequently for water. Its voice is hoarse; it sneezes frequently; it may have rather a shrill cough. It complains of no pain in swallowing. After some hours, or not until the lapse of four or five days, symptoms characteristic of the disease first begin to declare themselves. Their onset is generally gradual, but it may be sudden. And sometimes they appear in a child who had up to that moment seemed to be perfectly well; usually then coming on in the night-time, just as is the case with spurious croup.

Of all these symptoms the most important is dyspnœa. The breathing is not only hurried but noisy. Each inspiration is attended with a peculiar whistling sound, which may be audible at a considerable distance; and a similar sound, or one of a more snoring character, may accompany the expiration. On uncovering the child's chest, one sees that the structures above the sternum and the clavicles, and the lower intercostal spaces, are all drawn inwards each time the child breathes. And as the disease advances, the epigastrium, the false ribs, and even the lower portion of the breast-bone itself, form part of a deep hollow, produced by the action of the diaphragm; this muscle, being arched upwards into the chest by the atmospheric pressure, can only drag its attachments backwards towards the spinal column, where it contracts.

The cough, at first harsh and clanging, gradually grows husky and at length is inaudible. The voice, from being hoarse, becomes whispering, and is finally extinguished. When the child attempts to speak, its lips can

* An assailant of the position so ably defended by the author would need acquaintance with hospital and private practice, and with children's diseases in town and country, as well as in different climates, such as very few physicians possess. For a most valuable summary of facts and opinions on this important subject, the reader is referred to the 'Report of a Committee appointed by the Royal Medical and Chirurgical Society,' ten years ago, "to investigate the relations of Membranous Croup and Diphtheria." There are numerous cases tabulated (64 by Dr Dickinson, 63 by Dr Gee, and 8 by Dr Fagge) and replies to a series of questions put by the committee from a large numb of physicians to London hospitals, in country practice and abroad (vol. lxii, 1879).

be seen to move, but not the slightest sound is heard. The nostrils dilate with each effort to breathe. The head is thrown backwards as far as possible, and the spine is curved in the same direction. It is not very obvious why this peculiar attitude should be adopted, but the supposition is that the trachea is stretched, so that air can better pass by the side of the false membrane. Ferrier speaks of having seen the corpse of a child who had died of croup resting on its head and heels, exactly as if it had had tetanus.

Besides its persistent difficulty of breathing, a child labouring under this disease is also liable to frequent exacerbations, attended with the most extreme suffocative distress. An extraordinary restlessness is a principal symptom of such attacks. If lying or sitting in bed, the little patient starts up and throws itself into the arms of its mother or nurse; in an instant it begs to be put back into its crib. It clutches at anything that may be within reach, or even at its own throat, as if to tear away the obstacle to the free entrance of air. After a few minutes, or a quarter of an hour, it sinks back exhausted and may fall asleep. There is still a doubt whether such paroxysms depend upon muscular spasm, or upon the impaction of portions of false membrane, or of inspissated secretion in the chink of the glottis. And, indeed, different opinions are held as to the cause of the other symptoms of croup. Some observers think that they are the mechanical result of a swollen state of the laryngeal mucous membrane, and of the presence of a membranous layer, or of muco-purulent matter upon its surface; some refer them to spasm of the laryngeal muscles; and some (including Niemeyer) attribute them to a paralysed state of the same muscles, which are supposed to be involved in the inflammatory process. Niemeyer, in fact, says that after death their substance is found to be watery, pale, and soft. Of course, it is only during the act of inspiration that a whistling sound could be produced by paralysis; and, accordingly, this writer draws the distinction that when such a sound accompanies the expiration it is invariably caused by the presence of false membranes obstructing the glottis.

It is a question whether the urine is ever albuminous. On theoretical grounds one would think it very likely that a disease which is attended with such extreme dyspnœa should be attended with albuminuria. But in Mr Lamb's series of cases, recorded in the 'Guy's Hospital Reports' for 1877, there was only one in which this symptom was detected, and in which there were not some other grounds for believing that the disease might really have been diphtheria.

Event.—When croup is to terminate favourably, the little patient's breathing becomes easier, its cough softer and more loose. It sometimes begins to expectorate muco-purulent matter in considerable quantity; and in this one may detect flakes and shreds of membrane, by floating it out in a saucer containing water, or even without doing so. In some cases large pieces are spat up, forming complete casts of the trachea. The symptoms are then greatly relieved; but the improvement is sometimes of very short duration. Sir Thomas Watson relates the case of a child who was on the brink of suffocation when tracheotomy was performed; a tubular portion of membrane of the size and shape of the trachea was presently forced through the opening, and the patient fell asleep; but within seven hours the dyspnœa returned and was soon fatal; at the *post-mortem* examination the windpipe was found to contain a new tube of lymph.

Indeed, remissions in the symptoms are of not infrequent occurrence,

especially in the morning hours, even when no false membranes are expectorated. The child breathes better; its cough is less distressing; it partially regains its voice; it may ask for food, or get a little quiet sleep. One must not be deceived by such a change. Too often, it masks a steady progress of the disease towards death. Thus Dr West relates the case of a little girl who seemed to have been freed from all her alarming symptoms when she was admitted into the hospital on the fifth day of the disease. But nine hours afterwards she died, without any great distress or violent struggle; and extensive false membranes were found in the trachea and bronchi.

In most cases the approach of a fatal determination is indicated by symptoms of asphyxia. The cheeks and lips become pale and bluish; the forehead is covered with a clammy sweat; the child ceases to take notice, and lies with half-closed drowsy eyes; the breathing becomes shallow; the pulse is rapid and intermittent; the extremities are cold. Death is often ushered in by convulsions.

Ætiology.—The chief exciting cause of croup is by all writers said to be exposure to cold. But as scarcely any of them seem to have been careful to exclude cases of spurious croup from their accounts of the disease, one hardly knows what degree of significance to attach to their statements. Dr Alison is quoted by Sir Thomas Watson as having noticed that it was often produced in a child by its sitting or sleeping in a room newly washed, and consequently that in Edinburgh cases of this disease occurred with especial frequency on Saturday night. Cheyne said that in all but three of the cases of croup which he saw there had been exposure to the weather; and of the exceptions one occurred in a child who had got up out of bed on the previous night, and stood for some time in a cold passage, snow being on the ground at the time; while another was the case of an infant, thirteen months old, who had been confined to the house on account of the weather for above a week, and was seized with the disease after a very cold damp day. This accords with our experience in Guy's Hospital, where there has not in the course of many years been an instance of a child being attacked with croup while an inmate of any of the wards. And Dr Langdon Down, writing to the Committee of the Royal Medical and Chirurgical Society in 1876, mentioned that croup was unknown among the children at the Earlswood Asylum, whereas there had been many mild epidemics of diphtheria.

It is stated that croup is more frequent in the winter than in the summer months. One cannot draw any conclusions from so small a number of cases as that which I recorded in the 'Guy's Hospital Reports' for 1877, but I may say that no such difference is observable among them; except, indeed, among those instances in which no false membranes were proved to be present. Perhaps, like acute pneumonia, the disease attacks those who are exposed to chills in the evening hours of a hot day, or during the cold weather which in our climate may occur at any period of the year.

The *age* at which a child is most apt to be attacked with croup is between two and seven years. The complaint is rarely seen in infants at the breast; indeed both Home and Cheyne thought that children weaned early were especially liable to it. In adults croup is unknown.

All writers agree in stating that boys are more often affected by it than girls. This is a point of some importance, because it may aid in fixing the boundary line between croup and diphtheria, a disease which (as might be ex-

pected) is equally frequent in both sexes. Indeed, I am not sure that the figures given by different observers, if taken in conjunction with their opinions about the two affections, may not suggest an inference with regard to the prevalence of croup and diphtheria respectively in different parts of Europe. Thus Sanné says that out of 1575 cases of diphtheria admitted into the wards of M. Barthez (where the number of beds for males and females is equal), 813 occurred in boys, 762 in girls. The difference might fairly be attributed to the inclusion of a few cases of membranous croup, which is not regarded by French physicians as distinct from diphtheria; but does not the fact that the difference is not larger indicate that in Paris membranous croup is rare? On the other hand, in 101 cases of croup occurring at Prague, Steiner found that seventy-seven occurred in males, twenty-four only in females. He defines croup as an inflammation of the air-passages attended with the formation of false membranes, and seems to make no attempt to exclude cases due to the contagion of diphtheria. Do not his figures show that the latter disease must be relatively infrequent in the Bohemian capital? The experience of a single institution like Guy's Hospital is too limited to afford a secure basis for a comparison of the numbers of the two sexes, but I am bound to state that they show no considerable preponderance of males among the cases of membranous laryngitis, while they do show a marked preponderance of males among those cases in which it is probable that no false membranes were found. This fact, taken with the difference in the frequency of the two forms at certain periods of the year, is undoubtedly an argument against the view which I have maintained as regards the connection between membranous and non-membranous croup.

Histology.—The false membrane possesses a beautifully laminated structure, being formed of layers of a homogeneous fibrillated substance which alternates with other layers consisting chiefly of leucocytes. It adheres very firmly to the epiglottis and the vocal cords; and Dr West says that when it is detached the mucous membrane of these parts is often found to be slightly eroded or ulcerated. But lower down it lies loose in the channel of the trachea—if it extends so far. Sometimes it is only in very small quantity, being represented by a few little shreds or fibres, embedded in muco-purulent matter, which then lines the larynx. In any case, such a muco-purulent matter is present wherever the membrane ceases in the parts of the air-passages immediately adjacent. Thus transitions between the two kinds of inflammatory product exist in every instance of croup; and we have every reason for expecting that unless the disease reaches a certain pitch of intensity, no false membranes will be found. But this is very seldom the case when it is severe enough to prove fatal.

The colour of the membranes in croup is commonly whitish, or whitish yellow; but sometimes they are grey or brownish, from admixture of blood. Home relates, at second hand, a case in which the patient expectorated a piece "which had a mortified appearance, like black shaggy silk."

All observers now believe that the larynx is ordinarily the starting-point of the morbid process in croup. The earlier writers supposed that it began in the trachea, their reason for this opinion being the absence of soreness during deglutition. Cullen accordingly called the disease Cynanche Trachealis. Steiner, however, says that he has seen four well-marked cases of "ascending" croup. In each instance the disease began with slight febrile symptoms, a rather painful cough and dyspnœa. After four to six

days, while the voice was still sonorous, membranes were expectorated. A week or more passed before the child became hoarse, and signs of laryngeal obstruction showed themselves still later, which in three cases proved fatal.

Extension of the false membrane into the lower air-passages is of very frequent occurrence. Steiner says that out of fifty-five autopsies of children (among which, however, many were doubtless cases of laryngeal diphtheria) there were thirty-one in which it reached the bronchi, with casts even in the smaller tubes; in nineteen it affected the trachea but did not pass lower, and in five it was present only in the throat and larynx. He remarks that "in England, during recent epidemics, implication of the bronchi has been strikingly rare," but I do not know on what evidence this statement rests. Among seventeen cases occurring in Guy's Hospital, in which the fauces were unaffected, there were seven in which the bronchial tubes were lined with casts. Some observers have thought it possible to detect clinically such an extension of the morbid process, the symptoms supposed to indicate it being feebleness of the vesicular murmur, urgent dyspnœa, and marked inspiratory depression of the epigastrium. But I agree with Steiner that these signs are not to be relied on, at any rate before tracheotomy has been performed. The sounds produced in the larynx in cases of croup are so loud that auscultation of the lungs is very unsatisfactory in its results.

According to Steiner pneumonia is of much less frequent occurrence than has generally been supposed. In seventy-two autopsies he found it in the lobular form eight times, in the lobar form only six times. Its recognition during life is difficult. Steiner and Peter have each met with cases in which there was dulness on percussion, with great weakness of the breath-sounds over one lung, before an opening was made into the windpipe, but in which after the operation these signs quickly disappeared. Probably they depended upon atelectasis of the pulmonary tissue, which is a frequent result of obstruction to the entrance of air, the base of the lung, or even the greater part of its posterior surface, becoming purplish-red, dry, and airless.

Diagnosis.—The principal points involved have already been mentioned in the discussion of the relations of croup to the "spurious" affection and to diphtheria. One must never forget that in reality none of the special symptoms do more than indicate the existence of laryngeal obstruction. The distinction between croup and other diseases of the larynx is based upon the acute character of the attack and upon the age of the patient rather than upon anything in the symptoms themselves; but even children are liable to different affections in which the breathing may be no less stridulous.

A post-pharyngeal abscess, for example, sometimes gives rise to "croupy" dyspnœa and cough, although it is not obvious why this should be the case. One day when the author was visiting his patients at the Infirmary for Children, the house surgeon reported that he had just been called to see a case of supposed croup, in which he had felt an abscess at the back of the fauces with his finger, and that relief was afforded as soon as the matter was let out. In a little child, eighteen months old, a patient of Dr Hudson, at Waltham Abbey, the respiration was rather of a snoring character than croupy, but there was a brassy cough. The abscess seemed to have commenced in a suppurative affection of the cervical glands, which had made its way inwards, instead of pointing externally. According to Steiner post-pharyngeal abscess occurs chiefly in infants at the breast, except when it

is dependent upon disease of the spine. He also says that it develops itself more insidiously than croup.

Sir William Jenner speaks of abscess at the side of the larynx in the connective tissue as another affection which may cause great distress in breathing by compressing the tube and pushing it aside, and as not being always easy of recognition. The possible presence of a foreign body in the upper air-passages must never be left out of mind. Laryngeal papillomata are perhaps always too slow in their clinical course to be mistaken for croup.

The *prognosis* of true croup is always grave. The mortality, under the most favourable circumstances, amounts to 60 or 70 per cent. of the children who are attacked by it, and the younger the patient the less is the chance of recovery. It is only when the case is one of the spurious affections that we can speak confidently of a happy issue.

Treatment of spurious croup.—Spasmodic laryngitis requires no active treatment, but it is usual to give an emetic of two to five grains of powdered ipecacuanha, with or without one sixth of a grain of tartarised antimony, repeated at intervals of ten minutes until free vomiting takes place. Jenner says that besides removing from the stomach any source of reflex irritation, and relaxing spasm by the nausea and faintness to which they give rise, these medicines also promote secretion from the laryngeal and bronchial mucous membrane and so relieve the catarrh. He has observed that cases left to themselves last much longer, going on for two or three days, whereas as soon as an emetic has acted the child generally falls asleep at once. However, he follows it up with a dose of calomel and jalap. Another plan of treating spurious croup is that recommended by Graves, of squeezing a sponge out of hot water—as hot as the hand can bear—and applying it beneath the chin, changing it as often as it gets cool, for ten or twenty minutes, until the skin becomes vividly reddened. One must always warn the relatives of a child who has had one attack of spurious croup that it is likely to have others if it is exposed to cold or wet weather and allowed to get chilled. Such children must therefore have special care taken of them, particularly as regards the warmth of their clothing, but they should be accustomed to have the neck and chest sponged over every day with cold water, and Steiner suggests that they should gargle the throat with it several times a day. A child who has repeatedly suffered from this affection is sometimes left with a permanent hoarseness of voice.

Treatment of membranous croup.—When a child is attacked with "spurious" croup the parents commonly send for a medical man at once. On the other hand, in a case of "genuine" croup (except in that rare form which begins quite suddenly and proves very quickly fatal) advice is very seldom sought until the disease has already existed for some hours, and often not for two or three days. The consequence is that one is very likely to deceive oneself as to the results of treatment if one is not well acquainted with the distinctive features of the two complaints. Cheyne seems to have fallen into this error when he formed the opinion that "if medical assistance were procured early enough, croup would scarcely ever be a fatal disease."

For, when the affection is really of a grave or dangerous character, it seems to be very doubtful whether the morbid process can ever be cut short by emetics or by the administration of tartarised antimony in nauseant doses, as has been recommended by so many writers. I have repeatedly followed this practice, but with no marked success, and it is obvious that one can draw no conclusion from the occasional subsidence of the disease under

whatever plan of treatment, since the same result might have occurred spontaneously.

Whether any benefit is derivable from the administration of calomel in croup is admitted on all hands to be very doubtful. Niemeyer, however, was in the habit of prescribing it in doses of a quarter to half a grain every two hours, and he speaks of it as being at any rate more useful than the tartar emetic. This writer also advocates in decided terms the application to the outside of the throat of cold compresses frequently changed.

In very young children one cannot attempt in any way to reach the interior of the larynx with topical remedies. It seems to me absurd to apply nitrate of silver to the epiglottis. Older boys and girls may be made to breathe the steam of boiling water, or to employ a spray apparatus; lime-water is said by Steiner to be preferable to any other liquid for inhalation, on account of its power of dissolving false membranes.

Even if emetics are useless at the commencement of the disease, they often do good service later on by bringing about the expulsion of pieces of the membrane. Sulphate of copper should then be preferred to antimony or ipecacuanha; from two to five grains are dissolved in an ounce of water, and a teaspoonful is given every ten minutes until the stomach rejects its contents. If this should be followed by a decided decrease in the dyspnœa, the emetic may be repeated a few hours later, when a fresh aggravation of the symptoms occurs. But not infrequently such medicines fail in their action at this stage, in consequence (as is supposed) of the carbonic acid poisoning, which renders the pneumogastric nerve insensitive.* Niemeyer recommends that the little patient should then be placed in a warm bath, and have some cold water poured over its head and shoulders. This, he says, almost always brings it round and makes it cough more strongly, and may even lead to the ejection of bits of false membrane. If no such result should follow there can be no doubt of the necessity for the immediate performance of tracheotomy; and after the operation the stomach often rejects the emetics which had been retained.

But of late years it has become more and more the practice to open the trachea early. If it is certain that the case is not one of spurious croup, the operation should very seldom be postponed beyond the time when the chest walls begin to be much sucked in during the act of breathing. Very often, however, a great deal of time is lost from the parents refusing their consent until it becomes apparent that there is no other hope of recovery. And it must be admitted that no period is too late for the possibility of success from tracheotomy: children, actually moribund, have sometimes been saved by it. There is, however, a much better chance of a favourable issue when it is performed at an early stage, before the lungs become congested, with some parts of their tissue entirely collapsed and the rest intensely emphysematous. Moreover, the child may thus be saved from the terrible distress and suffering which accompany the gradual development of asphyxia.

The method of performing tracheotomy is a surgical question. But with respect to the after-treatment—at Guy's Hospital we place the child

* The statement in the text is based upon the authority of other writers. I think I have seen cases of croup in which vomiting could not be excited by emetics, although there were no indications of asphyxia, the disease being at an early period of its course; and Dr Yeats mentions an instance of this kind in which (although the disease was diphtheria) the strength was well maintained, and the child, after two weeks' illness, recovered.—C. H. F.

near a fire; we make a kind of tent, within which its crib is placed; and we direct a long metal tube from a kettle into the space so shut off from the rest of the ward. Of late years, however, many writers, including Sir William Jenner, have expressed the opinion that such precautions are not only unnecessary but injurious; that physician goes so far as to say that he is sure he has seen cases terminate fatally that would have recovered had they not been thus over-nursed and over-cared for. The plan, introduced by Trousseau, of covering the neck with a large folded piece of muslin, appears to afford sufficient protection against cold; moreover, it keeps the air which enters the trachea moist, giving up to it the aqueous vapour which had been deposited from the breath.

If the operation should not be followed by subsidence of the dyspnœa, it is well to introduce the feathery part of a quill pen into the trachea through the wound or through the cannula, and to twist it round a few times, in the hope that it may entangle and bring away with it some of the false membrane. And when this procedure has succeeded once it may be repeated again and again. A moderately abundant discharge of mucous fluid from the tube is said to be a favourable sign: its remaining perfectly dry is of evil omen.

When a case goes on well after tracheotomy, one can often finally remove the tube on the sixth, seventh, or eighth day. In 87 out of 134 cases collected by Sanné, it was withdrawn between the fourth and the tenth days. Cases in which it has to be retained after the end of the third week are quite exceptional, but it does sometimes happen that the larynx becomes thickened and narrowed by a process of chronic inflammation, and Steiner mentions instances in which its channel was completely closed by adhesions. A practical point of much importance is that when there is partial stenosis the child may appear free from dyspnœa until it goes to sleep. In a case of this kind, the tube was removed one afternoon, four months after tracheotomy for a scald of the throat, and the child seemed to be perfectly comfortable all day, but died unexpectedly in the course of the following night.

Recently the practice of *intubation* of the larynx has been introduced as a substitute for tracheotomy. A short tube is passed through the larynx, and, strange to say, rarely excites cough or distress. Deglutition is performed best with soft solids, for liquids are apt to trickle down the trachea. The practice was first introduced many years ago by Bouchut,* but has lately been extensively carried out by Dr O'Dwyer in New York, and promises to be of practical value.

Membranous laryngitis in the adult.—There is no question, that on the one hand diphtheria with membranous laryngitis is common to all ages, and that the disease to be next described, acute catarrhal laryngitis, is even more common in adults than in children. But whatever opinion be held as to the arguments in the preceding section that children are liable to membranous inflammation of the larynx as an idiopathic sporadic disease apart from diphtheria, it is generally supposed that in adults the mucous lining of the

* This was in 1858, but the results were not satisfactory, and the practice was abandoned in favour of tracheotomy in cases of laryngeal diphtheria, principally in consequence of the report of a Committee of which Trousseau was a member. The method had not become widely known even in France, and was long forgotten, when Dr O'Dwyer began his experiments in 1880. See his paper in the 'New York Med. Journ.,' August 8th, 1885, and one by Dr Inga's, of Chicago, in the 'Journal of the American Med. Assoc.,' February 6th, 1886.

larynx and trachea is incapable of producing a false membrane, except under the specific action of diphtheritic contagion. It is certain that fibrinous membranes are not produced in adults as they are in children, by traumatic inflammation—scalds of the throat, for instance; but, nevertheless, the adult larynx and trachea may become the seat of acute sporadic idiopathic inflammation, which leads, not to pus and catarrhal products, but to a fibrinous exudation forming a false membrane.

This is proved by the case of a pregnant woman who was admitted into Guy's Hospital, in December, 1879, with severe dyspnœa from laryngitis. She was taken ill with shivering, after exposure to cold, spat up membrane on the following day; and two days later a complete cast of the trachea with its bronchial ramifications was got rid of. She died after a week's illness, having previously miscarried; and there was found laryngitis, tracheitis, and bronchitis, with lobular broncho-pneumonia and pleurisy. The pyrexia had been moderate, there was no hæmoptysis, no albuminuria, and, *post mortem*, all the other organs were sound. The fauces had been free throughout. Histologically the false membrane consisted of leucocytes and scanty fibres without blood-discs or epithelium ('Path. Trans.,' vol. xxxi, p. 30).

Catarrhal Laryngitis.—From what has been said in the previous section it will be evident that the affection now to be discussed does not altogether correspond in scope with the name under which it is known; for it would be certainly difficult to prove that in cases of "spasmodic laryngitis" or "spurious croup" (as described at p. 22), catarrh of the larynx is entirely absent. But it is in clinical practice impossible to avoid drawing a broad line of distinction between cases of inflammation of the laryngeal mucous membrane attended with dangerous dyspnœa (whether from swelling or from spasm of the larynx), and those of which the chief symptom is impairment of the voice. The latter are commonly known by the name of catarrhal laryngitis; and this, again, may be either acute or chronic.

1. *Acute catarrhal laryngitis* is a very common affection, but comparatively seldom comes under medical advice. Some persons are very subject to it; whenever they catch a cold they become hoarse or lose their voice, and this condition lasts for several days or even for weeks. It is particularly apt to occur in those who are intemperate and in those who habitually use the voice much, particularly in the open air; the reason being that in such persons the laryngeal mucous membrane is constantly more or less congested. It is common in those who are exposed to smoke and irritating gases, as firemen and workers in chemical factories. It may also arise as a complication of some acute disease, as enteric fever, but especially measles. The chief symptom, beside the impairment of the voice, is expectoration of a little tough mucus, which comes away with a short hawking effort, hardly amounting to a cough. With the laryngoscope, according to Ziemssen, one may find that there is reddening and slight swelling of certain parts of the interior of the larynx, especially the hinder ends of the vocal cords, the inter-arytænoid space, or the false cords. During attempted phonation the cords may leave an oval space between them, exactly as though the internal tensors were paralysed. In somewhat more severe cases Störk speaks of the cords as looking red, dry, and lustreless, or even as being covered with yellowish-green crusts formed of dried-up exudation. They may also become ecchymosed, and sometimes their surface shows superficial excoriations.

The prognosis in acute catarrhal laryngitis is generally favourable. It

must not be forgotten, however, that cases in which the early symptoms were those of mere catarrh sometimes run on into œdematous laryngitis; and, on the other hand, the affection, if neglected, may become chronic, and may then be exceedingly intractable.

In the treatment one of the most important points is that the patient should entirely abstain from using the voice. He should remain in a room of which the temperature is kept uniform. Steam inhalations should be frequently employed. Mackenzie advises the addition of tinct. benzoin. comp., or of succus conii (ʒij with gr. xx of sodæ carb. exsiccat.), or of lupulin (ʒss) to the hot water used for inhalation, the temperature of which should be from 140° to 160°. The patient should be encouraged to drink freely of demulcent liquids, such as barley water, linseed tea, and the decoction of cetraria or of althæa. Warm milk or lac cum sevo is a useful beverage. If there is great irritability of the larynx, evidenced by cough and tickling or pricking sensations in the throat, it should be kept in check by opium or morphia, or (according to Störk) by chloral or by lactucarium. It seems to be doubtful whether the application of either hot poultices or cold compresses over the cront of the larynx is advisable. Störk and others recommend, at the fommencement of the disease, that a strong solution of nitrate of silver should be brushed over the cords, but Mackenzie thinks that this is better left undone.

The best way to overcome the liability to acute laryngeal catarrh, in those who are subject to frequent attacks of it, is to make the patient gradually accustom himself to daily sponging with cold water, and to exposing the throat in the open air without wraps, even in the winter. No one can avoid allowing a draught to play upon the neck from time to time indoors, or in a corridor, or in passing from house to carriage; and the more a person endeavours to keep the part protected the more surely will he suffer when the occasion arises. It is also important to live, especially during the night, in airy, cool, well-ventilated rooms. Warm, light clothing should be worn. A long stay by the seaside in the autumn, or in the bracing air of Scotland or Switzerland, often does a great deal towards diminishing the susceptibility to catarrh in the following winter.

2. *Chronic catarrhal laryngitis* often arises out of the acute affection, especially in persons who, in spite of hoarseness of voice, persist in attempting to continue duties requiring loud speech. Such cases are common in clergymen, schoolmasters, costermongers. Another frequent cause is extension downwards from a "granular pharyngitis." Störk believes that a liability to this form of chronic faucial irritation often passses by inheritance from parent to child, having been originally set up in the former by a course of mercury or of iodine. He also thinks that in other cases inoculation of the nasal cavity with gonorrhœal or leucorrhœal discharge is the starting-point of a catarrh that may last for years. As a rule, chronic catarrhal laryngitis occurs during the middle period of life; it is more common in men than in women.

The principal *symptom* of this affection is hoarseness of voice, which may pass on to complete aphonia. The degree to which the voice is impaired may vary very much at different periods of the day. Mackenzie remarks that it is often greater when the patient first begins to talk after an interval of silence than it is a few minutes later, after he has gone on speaking for a time. The attempt to use the vocal cords often gives rise to

a painful sense of fatigue, and there frequently are also complaints of a feeling of dryness or soreness in the throat, and of a tickling sensation leading to a constant desire to hawk or to cough. The expectoration is scanty, and generally consists of a viscid grey mucus, but sometimes is yellow and puriform.

The laryngoscope shows all gradations, from a slight localised injection and swelling of some part of the mucous membrane of the larynx, up to the most extensive and diffused redness of the whole interior of the organ. Mackenzie remarks that one vocal cord may be of a bright red colour while the other is white; he also says that the congestion may be limited to a small portion of one cord, this being always on the outer or attached side of it. Small pieces of mucus are often seen adhering to the mucous membrane at different points; in the form of whitish threads they may pass across from one cord to the other; in cases of long standing the whole surface of the larynx may be covered with secretion. During vocalisation the cords in many cases fail to meet one another. This may be due to swelling of the inter-arytænoid mucous membrane, which is sometimes so extensive as to form a convex projection even when the arytænoid cartilages are as far apart from one another as possible. But in addition to this a paretic state of the muscles is not seldom present. Ziemssen says that this is most frequently unilateral, in which case the opposite cord may pass across the middle line to meet the affected one, after the manner already described at p. 5.

Sometimes certain parts of the larynx are greatly thickened. Ziemssen says that this is especially apt to be the case with the epiglottis, the arytæno-epiglottidean folds, and the false cords. Lewin maintained that thickening of these folds is a particularly marked feature of "clergyman's sore-throat," but this is disputed by Mackenzie. Störk relates in detail a case in which the whole of the interior of the larynx was affected with an extreme degree of hypertrophy of its mucous membrane, so that there was great dyspnœa, rendering tracheotomy inevitable. The disease had been of fifteen years' duration. From the right false cord there grew a hard, solid, fibrous tumour, of the size of a hazel nut; every part of the interior of the larynx was thrown into enormous folds and ridges. Polypoid excrescences are, indeed, not uncommon results of a chronic laryngeal catarrh. Another affection which has sometimes been observed under similar circumstances is a thickening of the mucous membrane below the glottis, reducing the channel for the passage of air to a narrow chink or ring; in almost every instance it has been necessary to open the trachea. Lastly, the vocal cords themselves sometimes become granular on the surface, a condition which has by Türck been designated "chorditis tuberosa" or "trachoma." It has been supposed to depend upon a partial dermoid change in the epithelium; but in one case Wedl found microscopically only connective tissue and nuclear overgrowth.

Enlargement of the mucous glands is not infrequent in chronic laryngeal catarrh. Dr Mackenzie speaks of seeing their enlarged orifices upon the epiglottis and upon the posterior parts of the cords in some cases, either as pale specks on a congested surface, or as small red circles on a pale surface. Another morbid appearance, which Ziemssen regards as an accidental complication of catarrh, is a dilatation of the veins of the mucous membrane, especially upon the epiglottis or upon the cords.

There has been some difference of opinion among writers as to whether

chronic catarrhal laryngitis is or is not apt to cause erosion or ulceration of the laryngeal mucous membrane. Störk says that such a result is not infrequent, and that when an ulcer forms over the *processus vocalis* it may give rise to expectoration of blood in sufficient quantity to suggest the presence of tubercular disease of the lungs.

This observer lays stress upon the occurrence of a vertical fissure in the inter-arytænoid mucous membrane. It is not peculiar to cases of chronic catarrh. Störk speaks of it as being extremely frequent, occurring in as many as 50 per cent. of those who attend his out-patient practice. The fissure, as the result of the traction upon its sides, assumes a rhombic form; so that the upper part of it, which is alone visible in the laryngeal mirror, appears triangular. Its detection is often very difficult; the patient must be placed in the position required for inspection of the trachea with the laryngeal mirror. The symptoms are not always very marked. Störk had seen a singer of reputation, whose voice remained perfect after such a fissure had existed for many years. Generally, however, there are symptoms which appear to be identical with those of chronic laryngeal catarrh. And sometimes the subjacent arytænoideus muscle becomes paralysed, in which case the patient's life may be made almost unendurable by the running down of fluid into the larynx whenever he attempts to swallow or even to lie down.

The course of chronic laryngeal catarrh is generally very tedious and protracted, one reason for this being the fact that patients will seldom carry out the necessary treatment with sufficient perseverance, imagining that they ought to be well in two or three weeks, and neglecting all precautions as soon as they begin to improve a little. There are, however, among schoolmasters and clergymen many who go to the opposite extreme, being so nervous and fidgety about their throats that they may almost be classed with hypochondriacs.

A good many cases recover perfectly under careful management. A very important point is that rest should be given to the voice, the patient either using a slate to express his wants, or at least speaking only in a whisper. He must also abstain from smoking, be very moderate in taking alcohol, and avoid all highly-seasoned foods. Locally, if the larynx is irritable, the patient should make use of a spray containing bromide of potassium or carbonate of soda with a little morphia. But the chief therapeutic results are to be expected from the use of astringent sprays containing tannic acid (gr. j—v ad ℥j) or alum (gr. j—x ad ℥j), or from the systematic application of astringent solutions to the interior of the larynx by means of a brush. Mackenzie says that he generally employs a solution of chloride of zinc (gr. xv ad ℥j); he applies this daily during the first week, on alternate days during the second and third weeks, and afterwards less frequently. Ziemssen lays great stress on the value of the topical use of solid nitrate of silver fused upon the end of a laryngeal probe; this he repeats at intervals of a week or a fortnight; it causes violent spasm, which, however, is at once relieved by filling the throat with cold water. When there is an inter-arytænoid mucous fissure, the application of solid caustic is the best treatment; this must, however, be done with great exactitude, for if the surrounding healthy mucous membrane is touched instead of the sore, the patient's sufferings will be aggravated instead of being relieved.

In many cases of chronic catarrh, electricity applied locally aids in

restoring the voice; and some patients find benefit from a course of the waters at Aix-les-Bains or at Ems.

When great thickening of the squamous epithelium from long-standing laryngitis is present, the case becomes what Virchow has described as pachydermia laryngis.* There is a flat diffused swelling of the membrane towards the back of the vocal chords (cf. p. 53, *infra*).

Tubercular Disease of the Larynx and Trachea.—It is in the medical literature of the end of the last century that the earliest allusions are to be found to what was then, and commonly is now, termed *laryngeal phthisis*. This name, however, is not a good one, because it is apt to suggest the idea that an affection of the larynx may give rise to wasting and to other symptoms resembling those of ordinary pulmonary phthisis, without there being any lesion of the lungs. That such is sometimes the case has, indeed, been asserted by Trousseau and by some other writers. But no pathological proof of it has yet been brought forward. The experience of all pathologists is that when a tubercular affection of the larynx is found after death, the lungs invariably contain tubercles, and also present destructive changes which have evidently been of long standing, even though there may have been little or no auscultatory evidence of disease during life. There appears to be no recorded instance in which, when death has been due to tuberculosis of other organs, leaving the lungs healthy, the larynx has been found to be affected with tubercular lesions; nor, again, have such lesions ever been found in the bodies of those who have died of acute general tuberculosis, unless there was old as well as recent disease of the lungs. Consequently, in spite of the *a priori* probability that tubercles should sometimes form in the larynx earlier than in any other part of the body, the pathological evidence points at present to the conclusion that this is never the case. It may, indeed, be urged, on the other side, that the laryngeal affection almost always lasts for a considerable time before death occurs, and even that its capability of producing a fatal issue by itself is doubtful. But either by interfering with deglutition, or by setting up necrosis of cartilages or œdematous laryngitis, it may sometimes greatly shorten life; and consequently if tubercular disease of the larynx ever arises before similar mischief in the lungs has begun, one ought, sometimes at least, to see in the *post-mortem* room cases in which with an advanced laryngeal affection the lungs are either healthy or in a very early stage of phthisis.

Pathology.—It has been doubted by many pathologists whether in "laryngeal phthisis" local tubercular lesions are really present. The view that the laryngeal affection which accompanies pulmonary phthisis is dependent upon the formation of tubercles was originally advanced by Laennec. It was, however, soon afterwards disputed by Louis, and since that time pathologists have ranged themselves into two camps with regard to this question, some affirming, others as positively denying it. There can, in fact, be no doubt that in the larynx one very seldom sees conspicuous grey or yellow tubercles, such as, for example, are observed with great frequency in the ileum in the very same cases. But the writer has always maintained a belief that the laryngeal affection was really tubercular, basing this opinion not merely on the very large proportion of cases of pulmonary phthisis, especially with tubercular ulceration of the intestine, in which this

* Paper read before the Medical Society of Berlin, July 27th, 1887.

affection occurs, but also on the fact that many laryngeal ulcers have thick caseating edges which appear to be characteristic of a tubercular process. Recently, however, the matter has been taken out of the range of speculation by the careful investigations of Heinze, of Leipzig, who published a monograph on the disease in 1879. The basis of his work was a microscopical examination of the tissues in fifty cases of phthisis, in each of which there was disease of the larynx or of the trachea, or of both together. For it is to be observed that in many instances the morbid process involves not merely the upper but also the lower air-passages, extending sometimes into the bronchial tubes; and for the sake of convenience we will here discuss all such affections together, inasmuch as there are no special symptoms that characterise the tracheal or the bronchial lesions.

Heinze found that in forty among his fifty cases tubercles were plainly recognisable in the larynx; in thirty-nine of these forty there was ulceration, in one there was tubercular infiltration of the mucous membrane without ulceration; in eleven of the forty there was in the trachea tubercular ulceration likewise, in eight the trachea contained ulcers which could not be shown to be tubercular. With regard to the remaining ten cases, he found that in eight there were in the larynx ulcers of which the tubercular nature could not be demonstrated, but in five of these the trachea showed tubercular ulcers; in two the larynx was healthy, but in the trachea there were tubercular ulcers. In other words, there were only three out of the whole fifty cases in which tubercles were not detected either in the larynx or in the trachea, or in both. The tubercles themselves were plainly visible to the naked eye in hardened sections, but they could not be seen in the recent textures. They lay partly in the mucous membrane, partly in the submucous tissue, but always on a plane superficial to the laryngeal mucous glands. They had often undergone more or less complete caseation. With regard to the characters of the laryngeal ulcers which Heinze classifies as non-tubercular, he states that there was nothing in their external appearance to distinguish them from those that were tubercular. In every instance, too, they were very superficial, in fact little more than erosions, and they were generally confined to the vocal cords. It is, therefore, quite open to question whether they were not originally preceded by a formation of tubercles which had softened and been cast off by ulceration, as is believed by Virchow to be very frequently the case when a case of laryngeal phthisis fails to show a definitely tuberculous character. In the trachea, on the other hand, there seems to be no doubt that ulcers occur which are really non-tubercular; they appear as minute depressions of yellow colour surrounding the mouths of the tracheal glands.

In one point only Heinze appears to go too far; this is when he asserts, on the strength of his observations as to the exceedingly small size of laryngeal tubercles in general, that those writers were in error who have maintained that in some exceptional instances they have recognised obvious tubercles in the larynx in making autopsies or even by the laryngoscope during life. Türck, for example, figures a larynx from a dead subject, in which besides tubercular ulcers there is what he describes as miliary tuberculosis plainly visible to the naked eye. And tubercles, both grey and caseating, have several times been seen at Guy's Hospital in the laryngeal tissues. Considering how variable is the size of tubercles in other organs, they may well in the case of the larynx be much larger in some instances than in others.

The fact that tuberculosis of the larynx never occurs independently of pulmonary phthisis, and that probably it never even precedes the lung-affection in order of development, cannot but suggest the idea that the upper air-passages become infected as the result of the passage through them of tuberculous sputum. This idea was originally suggested by Louis, but he thought that the sputum acted merely as an irritant upon those parts with which it came into contact. At the present time one can form a far more definite conception of the way in which tubercle bacilli, settling upon the laryngeal or upon the tracheal mucous membrane, may germinate and invade the tissues. Nor is the possibility of such an occurrence invalidated by the observations of Heinze as to the commencement of laryngeal tuberculosis beneath the intact epithelium.

Ziemssen maintains that a continuous tract of ulceration can sometimes be followed from a vomica in the upper lobe of one lung along the corresponding bronchial tube (which alone of all the bronchial tubes may be affected) through the trachea to the larynx. But, of course, it is quite conceivable that, without there being any vomica, tuberculous sputum from the lung may infect the larynx. And conversely—if laryngeal tuberculosis really does ever precede the pulmonary affection in point in time—one can easily imagine that secretions from the larynx may descend the air-passages and infect the lungs.

Sex and age.—That tubercular disease of the larynx and trachea should be more frequent in men than in women might have been anticipated from the fact that this is the case with pulmonary phthisis, but the preponderance of males over females is far greater; according to Heinze 33·6 per cent. of male phthisical patients have ulceration of the larynx, but only 21·6 per cent. of female phthisical patients.

The age at which tubercular laryngeal affections are absolutely most frequent is between twenty-one and thirty; but among fatal cases of phthisis the proportion in which the larynx is found diseased is relatively larger at a more advanced age, namely, between forty-one and fifty for males, between thirty-one and forty for females. During childhood tubercular disease of the larynx is not common; among nearly 400 cases Heinze found only nine in patients under the age of fourteen; in none of these was there ulceration of the trachea; one was an infant of eleven months. The nature of a man's occupation seems to have no marked influence in modifying his liability to have his larynx affected, assuming him to become phthisical. Heinze found that the proportion of cases in which laryngeal complications occurred was nearly as high among consumptive butchers and tailors as among open-air singers and others whose throats were exposed to local irritation in various ways.

The *symptoms* of tubercular disease of the larynx vary with its exact seat. When the epiglottis is affected or any other part of the upper orifice of the larynx, there is often extreme dysphagia, the attempt to swallow even fluids causing great pain and distress. But Heinze relates one case in which this symptom remained entirely absent, although the epiglottis was greatly swollen and thickened. The voice becomes hoarse and weak and ultimately it is often reduced to a whisper. In some cases in which the true cords are entirely destroyed by ulceration, it is believed that the false cords may vibrate so as to produce harsh deep tones. The effort to speak is often painful, producing a feeling of soreness in the throat. The cough becomes weak and hoarse and toneless.

It must not be supposed, however, that marked subjective symptoms occur in every case of phthisis in which one subsequently in the *post-mortem* room finds that the larynx is affected with tubercular disease, even when it has advanced to ulceration. A few isolated ulcers are often found when no laryngeal affection had been suspected during life; and experience in the deadhouse leads to a decided impression that extensive superficial affection of the mucous membrane possesses clinical significance far more constantly than does the presence of localised ulcers, however deeply they may penetrate into the laryngeal structures. On the other hand there are many cases of phthisis which are attended with hoarseness of voice or even with aphonia, but in which no tubercular affection of the larynx can be detected, whether during life or in the dead body. In 'Virchow's Archiv' for 1877, Fränkel, of Hamburg, endeavoured to find an explanation of such cases in the occurrence of lesions in the laryngeal muscles, the fibres of which he showed to have undergone a granular change ending in complete absorption of their substance and emptying of the sarcolemma, while at the same time the nuclei of the internal perimysium underwent increase. It does not appear that the patients who furnished the material for his observations had had any definite paralysis, and indeed the lesions which he detected were distributed equally over all the muscles. But paretic states of the thyro-arytænoideus internus are said to be not uncommonly present in phthisis, and Gerhardt has described the occurrence of paralysis in the right recurrent nerve as the result of its having become involved in a thickened mass formed by the pleura covering the apex of the right lung. In the laryngeal mirror what chiefly characterises cases of phthisis attended with impairment of voice, when there is no local tubercular disease, is the extremely anæmic state of the mucous membrane of the larynx.

Diagnosis.—The laryngoscopic appearances which indicate tubercular disease of the larynx differ greatly in different cases. Heinze lays great stress upon the recognition of *tubercular infiltration* of the mucous membranes, which (he asys) is quite peculiar to this disease, and which was present in twenty-one of his forty cases. Its most frequent seats were the false cords and the arytæno-epiglottidean folds. He speaks of it as forming in the dead body a smooth tense swelling, of a greyish-white or greyish-yellow colour, often presenting on its surface the appearance of fine pale yellow granules, or spotted with points of hæmorrhage. Over the summits of the arytænoid cartilages tubercular infiltration gives rise to swellings which by Heinze are compared to two rounded sugar-loaves; Mackenzie describes them as "pyriform." The epiglottis, when it is affected, appears rounded and thickened, or "turban-shaped." The false cords become greatly swollen, so that they lie in the same vertical plane with the true cords or even overhang them, while the entrances into the ventricles of Morgagni are greatly narrowed, or seem to be entirely obliterated. Thickening of the inter-arytænoid mucous membrane gives rise to a local bulging or excrescence at the back of the glottis, to which Störk draws attention as characteristic of tubercular disease, it being all the more so because neither polypi nor other new growths are ever seen in that position. The cords themselves very rarely exhibit a true tubercular infiltration, but they may become swollen and rounded, a change which Heinze found to be dependent upon the presence of numbers of small round-cells between their fibres. In the trachea, it is only in the posterior membranous part that tubercular infiltration occurs.

But in a great many cases of tubercular disease the diagnosis, so far as the laryngoscope is concerned, is based mainly upon the presence of more or less numerous *ulcers*. According to Heinze their most frequent seat is upon the vocal cords; among his fifty cases ulceration of the cords was present in no fewer than forty; on both sides in twenty-seven, on one or the other side separately in ten, at the anterior commissure in three. Sometimes the ulceration was limited to a small part of the cord, sometimes it affected the whole length; in eleven cases one or both of the cords was completely destroyed. In the laryngeal mirror an ulcer upon one of the cords may appear either as a narrow linear fissure, or as an actual excavation of its edge, situated upon a more or less reddened surface. When the process of ulceration is further advanced it often happens that the cord looks as if it were split up into two or three separate longitudinal bands, with very irregular edges, arranged one above the other like a short flight of steps. The false cords are comparatively seldom ulcerated, among Heinze's cases only in fourteen; but in eight of these the affection was bilateral. Over the arytænoid cartilages ulcers were present in twenty-three of Heinze's cases, sometimes towards the bases of the cartilages, sometimes upon their summits or even towards the pharyngeal surface. Those which lie towards the bases of the cartilages are almost always bilateral. They are seldom, if ever, visible in the laryngeal mirror. They have a peculiar tendency to penetrate deeply into the tissues, so that they often reach the perichondrium and lead to necrosis of the cartilages. In the dead body they are seen to have their outline irregular, their edges smooth or fringed with papillary outgrowths, and their surface uneven, perhaps of a dirty grey colour. The epiglottis was ulcerated in twenty-six of Heinze's cases, generally upon its laryngeal surface, sometimes upon its border, never on its lingual surface. The appearance of epiglottic ulcers varies in different cases; often there are great numbers of small round shallow sores; sometimes they have run together into a large irregular excavation. The surface upon which they lie is often but little reddened. In some cases the substance of the epiglottis itself becomes eaten away from its margins inwards so that it may present one or more deep fissures, with pointed processes between them; or it may even be reduced to a mere stump. In such cases the ulceration of course affects the lingual surface as well as the laryngeal; indeed it often spreads for some distance upon the base of the tongue. In such cases, too (and indeed in many others), the whole surface of the larynx is often covered with ulcers varying in shape, in size, and in depth.

It must not be imagined that the detection of ulcers by means of the laryngeal mirror is always an easy matter, even when they come fully within the field of observation. Störk remarks that they are often recognised, not so much by the presence of an obvious depression in the mucous membrane, as by a change in its colour and a deficiency in the lustre naturally belonging to its epithelium. Unskilled observers are often misled by patches of puriform mucus lying upon the surface of some part of the larynx, which they take for ulcers.

Even when the presence of ulceration of the larynx is established, it still remains to be considered whether the disease is tubercular. We have seen that in catarrhal inflammation ulcers are seldom found. And practically the diagnosis generally lies between "laryngeal phthisis" and syphilis; the points of difference between the two affections will be discussed under the latter head (p. 45).

It must not, however, be supposed that, in the *post-mortem* room at any rate, ulceration of the larynx is of rare occurrence, apart from either tubercle or syphilis. In acute pneumonia ulcers over the arytænoid cartilages are not infrequent; and ulceration has been seen there, or upon the vocal cords, in two cases at Guy's Hospital in which there was double pleurisy with pericarditis, in two cases of erysipelas, in one case (probably pyæmic) in which there were abscesses both in the liver and in the brain, and in two cases of Bright's disease. One of these last-mentioned cases occurred in a man, aged twenty-four, who had been hoarse for three months before his death; all that was observed by the laryngoscope was that his cords were at one time reddened, but afterwards pale; at the autopsy a narrow linear ulcer extended over each cord for a quarter of its length. Heinze gives details as to eight cases in which ulcers were found in the larynx after death from various diseases.

Prognosis and treatment.—The author has met with three cases of phthisis in which the larynx after death presented what appeared to be the cicatrices of ulcers that had healed; and both Ziemssen and Heinze have recorded similar instances, some of which occurred in patients who had been the subjects of repeated laryngoscopical examinations. One of Heinze's cases is that of a man whose right cord was much ulcerated; two or three years later this was found to have healed and there were also cicatricial bands on the left cord, which must have become affected in the interval. Both this patient and another appeared to owe their recovery to residence in a southern health resort. Of course, however, it remains a question whether the ulcers in either case were actually tuberculous in a strict sense of the term. At the meeting of the International Congress in London in 1881 Rossbach and some other observers spoke very positively with regard to the occasional "curability" of "laryngeal phthisis," while Krishaber virtually denied it. The former speaker attributed considerable value, at an early stage of the disease, to the inhalation of antiseptic agents; the latter declared that no such treatment was of the slightest use. Ziemssen lays stress upon the importance of giving absolute rest to the voice by maintaining perfect silence, or speaking only in a whisper, for months together. Mackenzie speaks of the local application of perchloride of iron, in strong solution, as sometimes greatly diminishing the irritability of the mucous membrane and so relieving the troublesome cough. Nothing, however, is so serviceable in this disease as the local insufflation of morphia as a powder, in a dose of $\frac{1}{8}$—$\frac{1}{2}$ a grain mixed with half a grain of starch. A special instrument called an "insufflator" is made for the purpose; it consists of a hollow vulcanite tube, one end of which is bent downwards so that it can be directed towards the orifice of the larynx; in the middle of the tube is a hole, through which the powder is introduced, and which is then closed by a moveable covering; the other end of the tube is connected with a piece of elastic tubing. In using this instrument the operator places the elastic tubing in his own mouth, and introduces the vulcanite tube into the mouth of the patient. He then blows the powder down into the larynx, just at the moment when the patient is drawing his breath. Mackenzie says that as the greatest diminution of the sensitiveness of the affected parts occurs in rather less than an hour, it is well, when there is much pain in swallowing, to introduce the morphia at about that interval before the time of taking food. It may be repeated twice daily. If distress is caused by liquids entering the air-passages, all fluid nourishment should

be thickened with arrowroot or cornflour; and the patient is more likely to swallow well in taking off a good draught than in sipping. Sometimes it is necessary to administer food through an œsophageal tube for weeks together. Dr Wolfenden recommends the patient, in extreme cases, to drink only when lying in the prone position, or supporting himself on his hands and knees. Cocaine lozenges taken before food are of the utmost value in these cases, and proved so particularly in a severe one lately in Guy's Hospital, when all other means failed. Tracheotomy is now and then required when there is extreme dyspnœa; but this operation seems in no way to retard the further progress of the laryngeal affection.

The duration of life in cases of tubercular disease of the larynx is seldom long. Mackenzie gives a list of 100 cases, of which seventy-nine ended fatally in from six months to two and a half years after "throat symptoms had become troublesome;" in nine only did death occur within six months; twelve were prolonged over a period of from thirty to forty-nine months. Ziemssen, however, seems to maintain that the quickly fatal course of "laryngeal phthisis" depends rather upon the coexistent lung mischief developing itself with great rapidity than upon any marked tendency on the part of the affection of the larynx to shorten the patient's life. According to this writer, when the pulmonary disease assumes a chronic form, laryngeal ulcers may exist for years.

Lupus of the Larynx.—Within the last few years a small number of cases have been recorded, in which lupus of the skin has been associated with a like disease of the epiglottis and even of the interior of the larynx. The affection is characterised by the presence of nodules which may be as large as peas, and of ulcers some of which have undergone partial cicatrisation. It is stated that the ulcerated epiglottis often looks as though a heart-shaped piece were taken out of the middle of its free edge. Otherwise there is nothing in the laryngoscopic appearances which could distinguish lupus of the larynx from the effects of syphilis; nor are there any peculiarities in the symptoms, which consist of hoarsenses, sore-throat, dysphagia, and perhaps dyspnœa. Consequently, the diagnosis of a case which Ziemssen gives as one of laryngeal lupus, in a girl of twelve, whose skin was free, depends chiefly upon the fact that treatment with iodide of potassium proved a complete failure. What is recommended for this disease is the systematic administration of cod-liver oil for a length of time, with energetic cauterisation by means of nitrate of silver. An arrest of its further progress may perhaps be hoped for, but hardly a cure.

A remarkable case of fatal chronic laryngitis which was regarded by the editor as lupus occurred in a young man who was long under his care in Philip Ward. There were neither signs nor history of syphilis, but the epiglottis was seen to be ulcerated and there was no appearance of tubercular ulceration of the cords nor of phthisis. He was therefore put under iodide of potassium and mercurial inunction, but with no good result. Emaciation gradually came on and he died after about a year's illness. Deglutition had been only possible for some time by the local use of cucaine, and the immediate cause of death was gangrenous pneumonia from particles of food gaining entrance to the air-passages. There was no proof of syphilis discovered after death, and only a trace of obsolete phthisis at the apex of one lung. The epiglottis was entirely destroyed, the cords much less affected. The appearance of the larynx was quite unlike that of tubercular disease.

Syphilitic Disease of the Larynx.—Laryngeal affections resulting from syphilis are by no means rare, whether relatively to other effects of the venereal poison, or relatively to other diseases of the larynx. It seems probable that their occurrence is to some extent determined by the existence of local causes of irritation. Thus Ziemssen speaks of them as being especially frequent in persons whose throats are exposed to cold, and who abuse the voice, as, for example, in costermongers. And Mackenzie found that whether in the earlier or in the later stages of syphilis the larynx was far more apt to suffer during the winter than during the summer. The majority of patients are, as might be expected, between the ages of twenty and forty, but among those who suffer from larygeal affections as remote sequelæ of syphilis it is not uncommon to find persons up to the age of fifty or sixty, or even seventy.

Some of the earlier laryngeal manifestations of acquired syphilis may occur within two or three months after infection; its more remote effects may appear five, ten, twenty, or even thirty years afterwards, when all other indications of the disease have long since disappeared, and when the patient has almost forgotten that he ever contracted it. However, perusal of some twenty cases that have at different times come under observation in the *post-mortem* room at Guy's Hospital leaves a decided impression that severe lesions of the larynx are much more often associated with cutaneous eruptions and other obvious signs of syphilis than are other visceral syphilitic affections, as, for example, those of the brain or of the liver.

In some cases, especially those of recent infection, the larynx shows nothing more than a mere superficial catarrh. In this there is nothing characteristic; Ziemssen warns his readers against supposing that a livid-red or dirty-brown injection of the mucous membrane justifies a diagnosis of syphilis. Nor does the protracted duration nor the obstinate recurrence of a laryngeal catarrh afford grounds for concluding that it is not simply inflammatory, whereas in the case of the pharynx such points sometimes possess a marked significance. Störk speaks of syphilitic catarrh of the larynx as so transitory that patients seldom come under treatment for it.

Next in order of development come "mucous patches," or "flat condylomata" (vol. i, p. 317). As to their frequency widely different statements are made by different writers. Some almost deny that they ever occur in the larynx, others say that they are often to be seen by the laryngoscope. Ziemssen speaks of their chief seats as being the cords, the posterior laryngeal wall, and the false cords. They may also appear on either surface of the epiglottis. According to Mackenzie they differ from pharyngeal condylomata in being yellow rather than white, and in being generally accompanied by less marked congestion of the surrounding mucous membrane; he also says that they are less apt to undergo superficial ulceration, and that they generally disappear quickly even without treatment.

At a later period of the disease gummata are of not infrequent occurrence in the laryngeal mucous membrane and submucous tissue. They are described as generally forming small rounded elevations, from the size of a pin's head to that of a small pea, of the same colour with the rest of the laryngeal surface, isolated or collected together into masses of considerable size. They may be seated upon the epiglottis, the posterior wall of the larynx, the false cords, or the surface below the glottis. In 1874 Mr

Norton showed to the Pathological Society a gumma larger than a pigeon's egg, which occupied the right arytæno-epiglottidean fold, and reduced the air-passage to a mere chink, so that it caused suffocation. Laryngeal gummata often ulcerate, but sometimes they remain stationary for a long time, and finally disappear by absorption.

Syphilitic *ulcers* in the larynx are often observed. During the earlier stages of the disease they are generally superficial; at a more advanced stage they are apt to eat their way deeply into the tissues, causing great destruction. It has been much discussed whether syphilitic ulcers present any characters by which they can be distinguished from the non-syphilitic. Türck maintained that some of these cases can be recognised at the first glance by their more or less circular form, by their excavated surface coated with a whitish-yellow material, by their edges, which are sharp, sometimes much raised, and surrounded by an inflammatory areola. It is especially from tubercular ulcers that the diagnosis has to be made. A point of great importance is the comparatively rapid development of syphilitic ulcers. Störk remarks that a patient with extensive destruction of the epiglottis as the result of syphilis may still retain a fresh, healthy appearance, which is never the case where such disease is of tubercular origin. Tubercular ulcers are smaller than syphilitic, except when several have coalesced together; they are often numerous, affecting both sides of the larynx at once, whereas syphilitic ulcers are usually solitary; they are generally seated upon a surface which is pale and anæmic instead of being reddened. But ulcers of the larynx sometimes occur in persons who, having had syphilis, are also affected with phthisis; it may then be quite impossible to declare positively what is the nature of the laryngeal affection.

In some difficult cases great help is afforded by the presence of ulceration of the pharynx, or of the base of the tongue, which in tubercular disease of the larynx is very infrequent. Indeed, Ziemssen remarks that in most cases destructive ulceration of the larynx is preceded by a like affection of the fauces, which passes by continuity of surface from the pharyngeal wall to the side of the epiglottis. At the seat of the earlier lesions cicatrices may very often be observed to have already developed themselves, and this is conclusive as to the syphilitic character of the affection, for in tubercular disease such partial cicatrisation is never seen.

Cicatrices, indeed, frequently form within the larynx itself, and produce an extraordinary amount of deformity. Sometimes a web is formed between the cords, as in several cases recorded by Elsberg, of New York. In other cases the epiglottis is dragged down and fixed to the side of the pharynx, or the parts forming the entrance of the larynx may be puckered together, so as to reduce it to a small round hole. Papillary outgrowths of considerable size are occasionally developed in the neighbourhood of syphilitic cicatrices, and may play an important part in increasing the obstruction to the passage of air.

The *symptoms* of syphilitic disease of the larynx generally include hoarseness of voice, which may pass on to complete aphonia. Pain is often entirely absent; but if the epiglottis or some other structure at the entrance of the larynx is affected, swallowing may be exquisitely painful, although even in that case there may be an entire freedom from pain at all other times. It is wonderful how well some patients manage to swallow, even when they have lost a large part of the epiglottis; the base of the tongue is

carried backwards, and keeps even fluid from passing into the interior of the larynx. Cough is often troublesome, and if there is extensive ulceration, muco-purulent exudation and blood may be expectorated in considerable quantity. In one case at Guy's Hospital hæmoptysis occurred to such an extent that the patient was thought to have phthisis; and Türck has recorded an instance of fatal hæmorrhage from an extensive ulcer of the left side of the interior of the larynx, exposing a necrosed piece of the cricoid cartilage. Otherwise it is chiefly by the supervention of œdematous laryngitis, or perichondritis of the larger cartilages, that syphilitic disease of the larynx tends directly to destroy life. But Türck and Ziemssen point out that, in the case of the arytænoid cartilages, an incurable necrosis is far less apt to follow deep ulceration of the mucous membrane covering them when the disease is syphilitic than when it is tubercular. In some instances that have occurred at Guy's Hospital a fatal termination has resulted from pneumonia, which was probably set up by the entrance of purulent matter from the affected parts into the bronchial tubes.

The *treatment* of syphilitic disease of the larynx should generally consist mainly in the administration of mercury, and inunction is perhaps to be preferred to other methods, as it is often important to produce an effect quickly. Spray inhalations with a weak solution of bichloride of mercury are said to be sometimes very serviceable. Sometimes it is advisable to brush over the affected parts with dilute tincture of iodine, or with a mixture of two parts of iodine, two of iodide of potassium, and ten of glycerine. When mercury has already been freely used, full doses of iodide of potassium should be prescribed internally. Tracheotomy is sometimes necessary, and cicatrices may have to be cut through with endo-laryngeal instruments. Mechanical dilatation by means of tubes, without cutting, has been lately much employed, and with excellent results, as in a recent case of Mr Symond's, in a boy at Guy's Hospital.

Congenital syphilitic lanyngitis is not uncommon, but is usually of only symptomatic importance, by causing aphonia. Cases, however, have occurred of deep and severe ulceration leading to death. Two in brothers, aged between six and three, are recorded by Dr Semon in the 'Pathological Transactions' for 1880, and a third in a girl eleven months old by Dr Thomas Barlow, in the same volume.

Fränkel has recorded an instance in which a syphilitic infant, less than three months old, became the victim of fatal laryngeal stenosis, as the result of perichondritis with exfoliation of the cricoid and of the left arytænoid cartilages.

Inflammation of the deeper Laryngeal Structures.—The inflammatory affections of the deeper laryngeal structures fall into two groups, those of the submucous tissue, and those of the cartilages. The former may be described under the name of "Œdematous" or "Phlegmonous Laryngitis," the latter under that of "Laryngeal Perichondritis."

Œdematous or phlegmonous laryngitis.—This is the affection which is commonly but inaccurately termed *œdema glottidis*, that name having been originally given to it early in the present century by Bayle. The inaccuracy is twofold; on the one hand, the part diseased is not the *glottis*, or space bounded by the vocal cords, for (except in very rare instances) they remain free from swelling, which really affects the entrance of the larynx above

them; on the other hand, the morbid process is by no means merely *œdema*, but assumes any gradation, from exudation of serum loaded with leucocytes into the submucous tissue up to diffuse purulent infiltration, or even the formation of an abscess. Consequently there are some cases to which the designation œdematous laryngitis, others to which that of phlegmonous laryngitis is more applicable. It is, however, necessary to use one name rather than the other in the present chapter, and perhaps the former is to be preferred as less narrow in its scope. Perhaps a better name than either would be "submucous laryngitis."

Œdema of the larynx in the sense of non-inflammatory dropsy is frequently seen in the bodies of those who have died of Bright's disease, or of heart disease, when the epiglottidean folds forming watery swellings are often of considerable size; such cases do not appear to present special symptoms during life, so that mere dropsy of the larynx has no clinical significance.

Very different is the state of affairs in a true œdematous laryngitis, which is one of the most rapidly fatal of all diseases. The appearances, whether at the bedside or in the *post-mortem* room, vary somewhat with the exact locality of the inflammation. When the epiglottis is involved, it forms a turgid round mass, perhaps as large as the end of one's thumb, and often consists of two lateral rounded halves pressed closely together, so as to leave only a narrow gap between them. This may be either felt by the finger passed into the fauces or seen in the laryngeal mirror; occasionally it may be directly visible when the tongue is depressed with a spatula. Sometimes the tissues in front of the epiglottis are included in the swelling. In other cases the parts most affected are the arytæno-epiglottidean folds, which may be converted into two large globular masses, tense and resisting, so that they feel like swollen tonsils. The mucous membrane covering the cornicula laryngis and that between the arytænoid cartilages may also share in the morbid process, in which case the movements of the cartilages are greatly impeded. Within the larynx the inflammation usually affects the false cords, which bulge downwards as well as inwards, so as to overhang and conceal the true cords. The latter are themselves very seldom involved in the swelling, but Risch has recorded ('Berl. klin. Woch.,' 1866) a case in which, having actually removed the larynx within ten minutes of the patient's death, he found the true cords swollen to the breadth of half a centimetre and pressed against one another, so as completely to close the glottis. A similar instance occurred at Guy's Hospital in 1873; the patient was a woman who was found moaning on the ground in the street, and who died before she could be brought into the ward. In some cases the effusion is limited to the structures below the cords, constituting what Gibb termed a "subglottic œdema." Mackenzie speaks of such cases as generally chronic rather than acute, but Ziemssen cites five instances, one of them observed by himself, in each of which the affection in question was clearly recognised by the laryngoscope, the symptoms being of a grave and urgent character and very rapidly developed.

The colour of the affected parts, as seen during life, is generally a bright red. After death they look much paler, the arytæno-epiglottidean folds in particular appearing gelatinous, and having often a yellowish-green colour from infiltration of pus into their tissue. When they are incised in the *post-mortem* room, however, it is often found that no fluid escapes from them, even under gentle pressure. Not uncommonly the

inflammation extends to the laryngeal muscles, which may be full of suppurating points.

Among the *symptoms* of œdematous laryngitis the most important is dyspnœa, which may increase with extraordinary rapidity until it destroys life by suffocation. Inspiration is commonly attended with a loud whistling sound. There is some pain in the throat, increased by speaking, and the larynx is tender when handled in the neck. The voice is not always much altered, but as a rule it becomes first hoarse and then extinct. The cough is hollow or sometimes toneless. If the voice, as is sometimes the case, becomes affected before the breathing, the explanation seems to be that the interarytænoid mucous membrane has been the first to become swollen. Another sign that this part is attacked is, according to Störk, an incessant dry jerking cough. If the epiglottis or other parts bounding the entrance to the larynx are inflamed at the beginning, there is intense pain in swallowing; and in all cases much distress is occasioned by the accumulation of buccal and pharyngeal secretions, which he is unable to get rid of.

Störk lays great stress upon the significance of dysphagia, as the earliest indication of commencing laryngitis in some cases, and upon the importance of using the laryngoscope, whenever there is difficulty of swallowing, not obviously accounted for by an affection of the fauces. A thorough examination with the finger often at once clears up all doubt as to the nature of the disease. But this must be done with some caution, for it is very apt to set up an attack of suffocative dyspnœa.

Ætiology.—Submucous laryngitis occurs chiefly in young adults between the ages of eighteen and thirty-five; in males more often than in females. It is rarely seen in children.

As a primary affection, it appears to be generally due to some septic influence, such as is commonly but vaguely described as "blood-poisoning." Mackenzie says he has met with it in hospital physicians, in medical students, in nurses, as well as in persons exposed to emanations from bad drains. Sometimes it appears to be directly dependent upon exposure to cold, as in Trousseau's case of a man who, having drunk too freely at a wine-shop, was turned out into the street on a cold night, and fell asleep there, to wake with a violent sore-throat, which in an hour or two became attended with the most extreme dyspnœa. Sometimes it arises by direct extension from faucial erysipelas, and sometimes it occurs in the course of smallpox, or of enteric fever. It is frequently the immediate cause of death in the *angina Ludovici*, a diffuse inflammation of the connective tissue of the neck, with brawny infiltration, which may or may not pass on to suppuration. In a case that occurred at Guy's Hospital in 1863 it was secondary to chronic suppuration in the fibrous tissues about the hyoid bone. It is often developed by extension from perichondritis of the laryngeal cartilages, or follows chronic tubercular affections of the mucous membrane. Other frequent causes of submucous laryngitis are scalds of the throat, the entrance of a foreign body into the larynx, and the swallowing of mineral acids or other corrosive poisons.

Some writers describe it as being occasionally the immediate cause of death in cases of Bright's disease. Fauvel, indeed, maintained that it may be the earliest symptom of that disease. A recent case at Guy's Hospital in which acute inflammation of the submucous tissue of the larynx occurred as a secondary complication, was that of a man, aged twenty-seven, who died in 1878 of epileptiform convulsions. He had com-

plained of sore-throat and of shortness of breath, but no symptoms had been observed indicative of laryngeal mischief. Both epiglottidean folds were found infiltrated with pus, but especially the left one. There was also much exudation of puriform lymph round the pharynx and at the base of the tongue. In the following year, a man, aged thirty-four, who was lying in the hospital with cirrhosis of the liver, was attacked one day at 11 a.m. with sore-throat, followed at 4 p.m. by severe laryngeal symptoms, and by rapidly fatal dyspnœa at 10 p.m. After death the left epiglottidean fold was found moderately swollen with an effusion of serum, the right one smooth and shining, and greatly enlarged by infiltration with a semisolid purulent substance. In a case of Bright's disease which terminated fatally by dyspnœa in 1866, the autopsy showed that the cause was not ordinary œdematous laryngitis, but the exudation of a shreddy lymph-like material upon the mucous membrane of the larynx below the cords, extending down to about the eighth ring of the trachea.

It is surprising how rapidly the symptoms of œdematous laryngitis are sometimes developed. Ziemssen relates the case of a young man who was attacked with extreme distress of breathing after eating some bread, and who ran off to the surgeon, thinking there must have been a needle in it, which was sticking in his throat. The laryngoscope showed that the right sinus pyriformis (outside the larynx) contained a pointed splinter of wood which was at once removed with a pair of forceps. Only a quarter of an hour altogether had passed, yet there was considerable œdema of the right arytæno-epiglottidean fold and the entrance of the larynx generally was much injected.

In cases of poisoning by corrosive liquids, laryngeal symptoms sometimes, after setting in suddenly and quickly reaching an alarming height, so that the necessity for tracheotomy appears imminent, subside with no less rapidity.

In *treating* a case of œdematous laryngitis, it is often well, at the commencement, to apply leeches to the neck over the sides of the larynx. Dr Mackenzie recommends that bromide of potassium should be given freely and that the patient should constantly suck ice. Trousseau speaks highly of spray inhalations impregnated with tannin or alum. If, however, the entrance of the larynx is found to be very greatly swollen, the best thing to do is to scarify the tissues thoroughly, so as to give the exudation an opportunity of escaping. A number of shallow parallel incisions should be made, the best instrument for the purpose being a "laryngeal lancet" or small double-edged knife mounted on a curved stem; but in an emergency an ordinary bistoury, covered up with adhesive plaster to within a quarter of an inch of its extremity, answers very well. If relief does not speedily follow tracheotomy must not be delayed. Above all the patient must not be left, even for a few minutes, until an opening into the trachea has been made, for a paroxysm of dyspnœa may set in at any moment, and may end fatally before there is time to fetch a surgeon.

Laryngeal perichondritis.—Several writers in the latter part of the eighteenth or in the earlier part of the present century recorded cases attended with suppuration round, and destruction of, one or more of the laryngeal cartilages. Until a comparatively recent period, the usual opinion was that the disease began in the cartilages themselves, which (it was supposed) first became ossified and then necrosed. But now it seems

to be generally admitted that the starting-point of such cases is rather in the perichondrium. Indeed, as far back as 1850, Dittrich, a very keen observer, pointed out in an interesting article in the 'Prager Vierteljahrschrift,' that in a young subject it is not uncommon to find, when a small portion of the cricoid, happening to lie bare in an abscess-cavity, is converted into a dirty-yellow calcified mass, that the rest of it and all the other laryngeal cartilages are in a perfectly normal state. And the inference which is suggested by such observations is confirmed by a case at Guy's Hospital in 1859, in which the right ala of the thyroid and the right half of the cricoid were alike necrosed, whereas the left halves of their cartilages had escaped. Moreover, ossification of the affected cartilages is not in reality always present, even in adult life. In a remarkable case of a child, eighteen months old, who died with "croupy" symptoms, we once found that part of the left half of the cricoid, which was bathed in pus, had undergone absorption, so that there was a gap in it, with thin smooth edges of perfectly natural appearance. And a year previously, in 1874, in the body of a man, aged thirty-three, the back part of the cricoid was necrosed, lying loose in an abscess-cavity, while its anterior part was represented by a narrow edge of healthy cartilage, thinning off into fibrous tissue. Lastly, there is every reason to believe that disease of laryngeal cartilages is often not merely dependent upon an affection of the perichondrium, but secondary to ulceration which began in the mucous membrane. Probably this is the correct explanation of many of the cases in which such disease arises in the course of phthisis, enteric fever, smallpox, or syphilis. And Dittrich suggested, in the paper already referred to, that in certain cases, occurring in persons confined to bed, necrosis of the cricoid is an indirect result of the pressure of this body, especially when ossified, against the vertebral column. His idea was that the pressure first caused ulceration and sloughing in the two opposed surfaces of the pharynx, and that then the affection of the anterior pharyngeal wall spread to the perichondrium. The morbid process would thus be strictly comparable with that which is concerned in the formation of ordinary bedsores; and Dittrich gave one case in which, the patient being a phthisical man, aged thirty-one, numerous bedsores were actually present at the time of death. He also recorded in detail two out of several cases in which, in bedridden patients, he had found that both surfaces of the pharynx showed local patches of ulceration, without the cricoid cartilage having as yet become involved in the disease. Störk satisfied himself that in severe enteric fever necrosis of the thyroid cartilage may arise in a similar way from pressure against the spinal column. And Ziemssen says that in old people, in whom the cricoid is ossified, that cartilage may be affected with perichondritis as a consequence of the repeated introduction of œsophageal bougies. Sometimes such disease is produced by direct injury, as in a case, recorded by Störk, of a man who was struck in the right side of the neck by a piece of wood, which flew up while he was attending to a circular saw. In some cases, perhaps, it is the result of exposure to cold. In others no cause can be discovered.

Laryngeal perichondritis is much more common in males than in females. An analysis of twenty cases, collected from the pathological records of the Berlin School, showed that the period of life at which it most often occurred was between twenty and thirty years of age. But probably this was dependent upon the circumstance that eighteen of the twenty patients either

were tuberculous or died of enteric fever. For at Guy's Hospital the disease, as a primary affection, has been more frequent in persons from thirty to fifty than in those who were younger. One case occurred in a girl under two years old, one in a boy of nine, and one in an old man of sixty-three.

Hitherto we have spoken of perichondritis as though its necessary result were to produce suppuration and destructive changes in the subjacent cartilage. But there is every reason to believe that this is not always the case. It was remarked above (pp. 5, 6) that ancylosis of the crico-arytænoid joints may probably be caused by development of fibrous tissue as the result of perichondritis. Ziemssen records the case of a young man, in whom, during the course of enteric fever, a dark red flat projection appeared over one processus vocalis, causing hoarseness and severe pain. During convalescence this gradually diminished, and under favourable circumstances it might possibly have entirely subsided; but he insisted on going out, and after three days' exposure to weather and indulgence in alcohol, returned in a state of such severe distress that tracheotomy had to be performed.

When necrosis does occur, the cartilage sometimes remains *in situ*, sometimes it is extruded from the abscess-cavity. An arytænoid is often expectorated entire; the larger cartilages commonly break up into fragments, which come away one by one. Störk speaks of having seen cases in which suppuration went on for years. If, however, the necrosed material is completely got rid of, the cavity may become closed up by fibrous tissue.

The *symptoms* of laryngeal perichondritis vary a good deal with the exact seat of the affection. At first, however, there is little to distinguish them from those of other subacute or chronic diseases of the larynx. The patient usually complains of hoarseness of voice or of aphonia; there may be dysphagia, cough, more or less definitely localised pain and tenderness; presently dyspnœa sets in, which may rapidly increase until it threatens suffocation. Sometimes the spontaneous evacuation of the contents of an abscess-cavity affords great relief to this symptom. In some cases of perichondritis enlargement of the cervical glands is a marked feature. In one case at Guy's Hospital they were found at the autopsy to be as large as plums. The putrid discharge which is formed in some cases is probably a direct cause of danger to the patient's life, by dropping into the air-passages and setting up pneumonia that may rapidly pass on into gangrene: two patients in Guy's Hospital died from this cause.

Perichondritis of the *thyroid* cartilage sometimes shows itself on the outer, sometimes on the inner surface of the cartilage. In the former case there is swelling, œdema, and at length fluctuation over one of the alæ or over the pomum Adami; the affected part is very tender when pressed upon. In the latter case a swelling usually appears in the position of one sinus pyriformis within the arytæno-epiglottidean fold on one side, or even below the vocal cord, as in an instance recorded by Störk, in which it was mistaken for a polypus. Not infrequently both surfaces of the thyroid are affected in succession, so that when the abscesses have discharged themselves, milk or any coloured fluid can be injected through a sinus in the neck and run into the larynx, or a probe can be passed from without inwards until it is visible in the laryngeal mirror.

Perichondritis of the *cricoid* cartilage usually affects its posterior rather than its anterior wall. It causes marked dysphagia. Another effect to

which it sometimes gives rise is paralysis of the crico-arytænoidei postici muscles, so that the cords appear fixed near the median line. In a case that occurred at Guy's Hospital in 1861 it is noted that the voice remained clear, although there was extreme dyspnœa. Sometimes the symptoms develop themselves with extreme rapidity. Ziemssen cites a case of Pitha's which ended fatally in a week from its commencement. Where suppuration occurs, the abscess may discharge itself into the pharynx, into the larynx, or into both canals at once. In some cases a swelling can be seen in the laryngeal mirror, bulging below one of the vocal cords; such a swelling has been mistaken for a solid new growth.

Perichondritis of an *arytænoid* cartilage leads to swelling and œdema of the surrounding soft parts, which may of course be visible in the laryngeal mirror. The mobility of the corresponding vocal cord is more or less interfered with, and the voice may be much impaired. Experience in the *post-mortem* room has impressed the author with the conviction that neither aphonia nor any marked alteration of the voice is nearly so constant a symptom of disease of an arytænoid cartilage as seems to be generally supposed. Cases of phthisis, in which complete exfoliation had occurred, had sometimes been free from laryngeal symptoms during life. In such cases there is a good deal of indurated fibrous tissue in the place of the cartilage, which seems to have fixed the cord and enabled the muscles to act upon it sufficiently to maintain its functions. Laryngoscopically, when an arytænoid has been exfoliated, there is often an obvious falling in of the soft structures around.

It is of course to be understood that more than one of the laryngeal cartilages are not infrequently affected at once; one or both of the arytænoids, for example, together with a part or with the whole of the cricoid.

The *treatment* of perichondritis, if the disease is detected early enough, may sometimes be begun with leeches, the application of an ice-bag to the throat, and other antiphlogistic measures. Whenever an abscess is recognised, whether outside or inside the larynx, it ought at once to be incised. Störk relates a capital case in which, having punctured a swelling below one of the cords and let out a quantity of pus, he subsequently brought the cavity to close by the systematic application of nitrate of silver to its interior. In almost all cases, however, tracheotomy is required sooner or later; and when dyspnœa has once set in there is great risk in delaying it. The immediate result is almost always successful, but it rarely happens that the swelling of the laryngeal structures afterwards subsides sufficiently to allow of the removal of the cannula. Schröter has recently had much success in the treatment of such cases by mechanical dilatation, at first with vulcanite tubes, and afterwards with pewter plugs, about an inch and a quarter in length, which can be left in the larynx for several hours at a time. Having been introduced through the mouth, the plug is held *in situ* by being bolted into the convex surface of the cannula which the patient is wearing.

Laryngeal Tumours.—New growths within the larynx are by no means very rare. From a clinical point of view it will be convenient to describe first those which are benign, and afterwards those which are malignant in character.

With regard to the *causes* of benign growths in the larynx, almost the only fact hitherto ascertained is that they seem often to arise out of the irritation connected with chronic catarrh of the laryngeal mucous membrane.

They are most frequently seen in persons who use the voice a great deal; this might be a sufficient explanation of the fact that they are far more common in males than in females, were it not that, according to Causit, a similar preponderance of boys over girls is observed among children affected with laryngeal growths.

Papilloma.—This, which is sometimes more accurately designated as *fibroma papillare*, is the commonest of all laryngeal tumours. It consists of a series of pointed or bulbous papillary excrescences, sometimes of small size, sometimes forming a large mass like a cauliflower, which may almost fill the cavity of the larynx. Their most frequent starting-point is from one or both of the cords, especially near their anterior extremities, or from the angle between the cords. But sometimes they arise from the false cords, or even from the epiglottis, seldom or never from the mucous membrane covering the arytænoid cartilages or the parts adjacent to them. Their colour may be either whitish, or pink, or red. They cause more or less alteration of voice, or even complete aphonia; cough, which may torment the patient greatly, and which may be of a "croupy" character; dyspnœa which sometimes ends in actual suffocation. It now and then happens that the fragments of papillary growths become detached in the act of coughing and are expectorated. Otherwise it is only with the aid of the laryngeal mirror that their presence can be accurately diagnosed. They not infrequently occur in young children. When removed by operation they are very apt to return, sometimes within a few months. Störk relates a case which came again and again under his observation during a period of thirteen years.

In cases of chronic laryngitis with papillary growths, Virchow finds that the lesion is usually situated towards the anterior ends of the cords. This *pachydermia verrucosa* is apt to be recurrent, but is not malignant. He looks on all the cases as simple, local, and only superficially hyperplastic, in which he finds a sharp line of distinction at the base of the epithelial growth separating it from the fibrous tissue beneath. Where, however, there is any trace of epithelium in the fibrous tissue, he considers the case suspicious. Neither the sessile nor the papillary swellings ought to show anything of an epithelial character below the border-line between the laryngeal epithelium and connective tissue. If there is no epithelium beneath this border-line, then, notwithstanding papillary outgrowths, he considers the disease to be local and benign.

Dr Percy Kidd has described a case of papillary tubercular tumours growing from the interarytænoid fold of mucous membrane in a man aged fifty, who died of phthisis with subsequent tubercular ulceration of the larynx and also of the colon. No giant-cells were discovered, but the other histological characters were those of tubercular growths, and characteristic bacilli were found abundantly ('Clin. Trans.,' 1884, vol. xvii, p. 156). Dr Kidd quotes only one other case of a young man from whose larynx several tubercular tumours were successfully removed by Schnitzler ('Wiener Med. Presse,' April 8, 1883).

Fibroma, or fibrous polypus of the larynx.—This forms a round or pear-shaped swelling, generally pedunculated but sometimes sessile, smooth or more or less lobulated, hard or more rarely soft in consistence, whitish or bright red in colour, varying in size up to that of a hazel-nut or even larger still. It is a solitary growth, its development is exceedingly slow, and it never recurs when it has once been removed by operation. Its most frequent

starting-point is from one of the vocal cords, but sometimes it is attached to some other part of the larynx. Ziemssen figures one, as large as a walnut, which arose from the mucous membrane covering the posterior surface of the cricoid cartilage. Growths of this kind most frequently occur in adult or middle-aged patients. Störk speaks of them as sometimes becoming ulcerated on the surface, so that they bleed. In some few cases a fibrous polypus has become detached spontaneously and has been expectorated. With the laryngoscope the existence and the seat of this sort of tumour are generally easily recognised. Almost the only thing that can cause a mistake in diagnosis is the occurrence of eversion of the sacculus laryngis. Such a specimen, taken from the body of a man who had had no laryngeal symptoms, was shown to the Pathological Society in 1868 by Dr Moxon, and is now in the museum of Guy's Hospital; it appeared like a semi-elliptical tumour hanging down in front of one of the cords, and could easily be replaced. More recently Dr Lefferts, of New York, has diagnosed this affection in the living subject.

The symptoms produced by a fibroma of the larynx vary with its seat. Unless it is at a distance from the glottis the voice is almost always more or less affected, one reason for this being that even if the growth does not actually interfere with the apposition of the cords the surrounding mucous membrane is sure to be affected to a greater or less extent with catarrh. When a polypus has a pedicle of some length it may rise between the cords during phonation, and rest upon their upper surface, whereas during inspiration it falls down between them. The occurrence of dyspnœa is very uncertain. Dr Mackenzie had a patient who invariably slept with her hand resting under the neck, and who would immediately wake up with distress of breathing whenever by chance her hand slipped away. In a case recorded by Lieutaud about a century ago, the patient died of sudden suffocation as the result of stooping out of bed to pick up a book which had fallen on to the floor. He had been conscious some time of the presence in the larynx of something which he could not get rid of by coughing.

Mucous cyst.—This is sometimes found upon the epiglottis, as in a case which occurred in 1863 to Mr Durham, who has recorded it in vol. xlvii of the 'Med.-Chir. Trans.' The patient was a boy, aged eleven, who had suffered for some months from dysphagia, from hoarseness and feebleness of voice, and from attacks of dyspnœa which came on especially during sleep. The cyst, which was situated upon the laryngeal surface of the epiglottis, was incised, and gave exit to a glairy, thick, muco-purulent matter; it is therefore evident that the cyst was inflamed, and indeed, the epiglottidean folds themselves were swollen and œdematous. In other cases a similar cyst has been found in the ventricle of Morgagni. Dr Edis has recorded an instance in which there was a cyst of the size of a hazel-nut in the larynx of an infant, who died of suffocation thirty-seven hours after birth.

Dr Abercrombie showed to the Pathological Society in 1881, a remarkable case of congenital cyst of the crico-thyroid membrane which blocked the glottis so much that the child (a female infant who died on the fourteenth day after birth) had never been able to cry or to breathe properly ('Path. Trans.,' xxxii, p. 33).

In some rare cases a laryngeal tumour has been a *myxoma*, a *lipoma*, an

angioma (Mackenzie), or an outgrowth of the thyroid body penetrating the crico-thyroid membrane.

The only *treatment* for benign tumours of the larynx is their removal by surgical operation. Mackenzie, however, advises that small growths on the epiglottis or on the false cords should be left alone if they give rise to no inconvenience; he has observed several cases in which small "warts," after reaching a certain size, have ceased to undergo further growth. Various instruments have been devised for the purpose of removing laryngeal tumours through the natural passages or (as it is termed) by the "endo-laryngeal method"—knives (guarded or unguarded), cutting forceps, crushing forceps, guillotines, écraseurs, the galvano-cautery, have all found their advocates. It is needless to enter into details with regard to them, because it is not likely that any medical man would attempt to use them without having had special training, nor without consulting the works of those who have devoted themselves to the study of laryngeal affections. In choosing an instrument for a particular case, the degree of hardness of the growth and the character of its pedicle form important considerations; they must be determined as far as possible by the use of a laryngeal probe. It is not advisable to use an anæsthetic unless tracheotomy has previously been performed, but a few whiffs of chloroform may sometimes be given with advantage. The local use of cocaine in solution or as spray has, however, now superseded all other attempts to produce anæsthesia.

A point which must be remembered is that, *cæteris paribus*, more skill is required in the removal of a very small laryngeal growth than of one which is larger. Both in this country and abroad an extraordinary degree of skill has now been attained in the performance of endo-laryngeal operations. The immediate result of the introduction of laryngeal forceps, or of any other instrument, is the production of a violent spasm, with a feeling as of impending suffocation, but this quickly passes off. When there are a large number of papillomata in the larynx, repeated endo-laryngeal operations are of course necessary, which may run over a period of several weeks.

In cases in which it is difficult or impossible to operate through the natural passages, the question arises whether recourse should be had to "thyrotomy," or the division of the thyroid cartilage in the median line, with separation of its halves, enabling the surgeon to seize the growth or growths and to clear out the whole cavity of the larynx on a single occasion. This procedure, which had been adopted for the removal of foreign bodies nearly a century ago, was vigorously advocated by Mr Durham in a paper read before the Royal Medical and Chirurgical Society in 1871. But subsequent experience seems to have greatly limited the range of cases within which alone its performance can be justified. At the International Congress in 1881 opinions were almost unanimous with regard to this question. It was urged that the operation is attended with considerable danger to life from hæmorrhage, or from other consecutive evils, among which pneumonia and necrosis of cartilages with suppuration perhaps take the principal places. Further, it was shown that a permanent impairment or loss of voice is a not infrequent result of thyrotomy, though, on the other hand, there are many recorded cases in which the voice has been perfectly restored. Lastly, it was pointed out that in some patients there is great difficulty in getting the alæ of the thyroid cartilage wide enough apart to enable the operation to be

successfully completed, and that experience does not at all confirm the expectation that the risk of recurrence of multiple papillomata is diminished by the adoption of this procedure as contrasted with endo-laryngeal methods. Most of those who attended the Congress thought that even in young children (in whom multiple papillomata are so common) thyrotomy is seldom necessary. Krischaber related the case of a child, aged six, in whom he succeeded in rapidly removing a number of tumours without a laryngoscope by sliding a pair of forceps along his index finger into the larynx. One criticism it is fair to make upon the speeches delivered at the Congress; it is that laryngologists, not being so much accustomed to ordinary cutting operations, have probably in the performance of thyrotomy met with greater difficulties and obtained less satisfactory results than might occur to hospital surgeons in the like cases.

A mucous cyst in the larynx requires only to be incised and to have its interior rubbed with caustic. Contrary to what might have been expected, it seems seldom or never to fill again.

Malignant growths in the larynx are sometimes *Sarcomata*, generally of the spindle-cell kind. Ziemssen speaks of there being more than twenty recorded instances of such an affection; its seat is usually on or near one of the vocal cords. Mackenzie figures a sarcoma which he describes as growing from the posterior surface of the cricoid cartilage; it had a papillomatous character.

Carcinomata of the larynx usually belong to the keratoid variety, such as are commonly called epitheliomata. They must be rare in comparison with cancers of other parts, for in the *post-mortem* room of Guy's Hospital only some four examples have been met with between 1854 and 1883. All of them occurred in patients between the ages of fifty-eight and sixty-five. According to Ziemssen, however, they are not uncommon relatively to other laryngeal growths; he speaks of having collected 147 cases, of which thirteen had come under his own observation. Among the patients there were many more men than women. In one curious case the development of the disease was preceded, at an interval of some months, by a fracture of the thyroid cartilage, the result of an attempt at strangulation.

The larynx sometimes becomes affected with cancer by extension from the pharynx or from the base of the tongue. But in the cases now under consideration the starting-point of the affection is in the laryngeal mucous membrane, its original seat being generally one of the cords, one of the ventricles of Morgagni, or one of the false cords. In a case that occurred at Guy's Hospital in 1875, the amount of the growth at the time of the patient's death was remarkably small; the left ary-epiglottidean fold showed a whitish thickening, with puckering, as of a healed ulcer, two or three lines in diameter; the thickening extended down to the false cord on that side; until the microscope revealed the structure of a carcinoma, it was doubtful whether a new growth was present. In another case, observed in 1862, there was a raised patch, somewhat papillary in character, growing from the left cord and the parts around. But in many instances, as the disease spreads, extensive ulceration occurs. The structures outside the larynx become infiltrated with the growth, which may protrude into the pharynx, or form an obvious tumour in the neck. The ulcerated surface within the larynx may pour out an abundant ichorous discharge mixed with blood, or may even be the seat of copious hæmorrhage. In such cases the breath

becomes horribly fœtid. Perichondritis, leading to suppuration, and to necrosis of cartilages, often occurs as a complication. Death may be due to œdematous laryngitis, or (as in two out of four cases at Guy's Hospital) to pleuro-pneumonia and empyema.

The laryngoscopic diagnosis of carcinoma of the larynx is by no means always easy. At an early stage, when there is little beyond a diffuse infiltration of the mucous membrane, the case may be taken for one of perichondritus; and at a later period, when an ulcer has formed, an affection really syphilitic may be supposed to be cancerous, as in a very remarkable case recorded by Ziemssen, in which he fortunately gave iodide of potassium with rapid and complete success, the patient being an old man of sixty-eight. There is, of course, nothing very characteristic in the other symptoms. Of these, hoarseness of voice, seldom amounting to complete aphonia, is the most constant and generally the earliest. According to Ziemssen, indeed, it is almost the rule that there is a "prodromal hoarseness," lasting a year or two; and in several of the cases which he collected this was prolonged during three, four, or five years, and once even during twenty-six years. It is certainly difficult to suppose that the affection had a definitely malignant character throughout such long periods; and indeed Ziemssen's statements with regard to the duration of laryngeal cancer appear scarcely consistent with what one knows of the rate of progress of a similar affection of other parts; he speaks of several cases which lasted three or four years, and of some which lasted even six, ten, or fifteen years. Next to hoarseness, *pain*, which may either be seated in some one spot within the larynx, or referred deeply to the pharynx, is the most conspicuous symptom. And Ziemssen lays stress on the frequent radiation of pain into one or other ear; this pain in the ear he associates with the auricular branch of the vagus; he found it present in five out of thirteen cases in which inquiries were made about it; and sometimes when there was no pain in the larynx itself. As a rule, dyspnœa occurs sooner or later; it may be especially marked when the patient is lying down. There may also be dysphagia. In all cases of suspected carcinoma of the larynx careful search must of course be made for enlarged cervical glands; but Ziemssen says that they can seldom be detected within the first six months, and often not for a year or even longer. He also says that cancerous infection of the viscera is of very rare occurrence. However, in a case of Mr Durham's, in 1879, which was yet in an early stage, there were already two flat subcutaneous nodules, one near the right clavicle, the other on the edge of the left sterno-mastoid muscle.

The *treatment* of carcinoma of the larynx can often be only palliative; but Ziemssen's case, already referred to, shows that whenever there can be a doubt as to the nature of the disease the patient should have the benefit of the chance afforded by a course of iodide of potassium. Indeed, at the London International Congress, in 1881, Dr Semon spoke of having several times seen this salt produce in cases of cancer a subjective improvement, and even apparently a brief temporary arrest of the progress of the disease. Tracheotomy is generally required sooner or later; the average duration of life after the performance of this operation is said to be not more than a year.

It is chiefly in cases of malignant tumour that the question has to be considered of the "total extirpation" of the larynx. This operation, originally performed for syphilitic stenosis by Dr Watson, of Edinburgh, in 1866, was first introduced to the profession in 1873 by Billroth, who in

that year carried it out in a case of cancer. In a paper read in 1881 by Dr Foulis, of Glasgow, before the International Congress, reports of thirty-two cases are collected, in twenty-five of which the disease was a carcinoma. In fourteen out of the twenty-five death occurred within sixteen days after the operation; and in not one of the remainder was life known to have been prolonged more than nine months, the only patients who were stated to be alive when the paper was read being two who had been operated on three months previously. It is difficult not to agree with Dr Semon, who took part in the discussion which followed the reading of Dr Foulis's paper, and who evidently was of opinion that the operation had not yet been shown to be justifiable. However, Bottini, of Turin, had one very successful case, in which the larynx was extirpated for sarcoma; in 1881, six years after the operation, the patient was well, and had been able to work in the fields and to act as a postman. This evidently suggests the possibility of a like success in cases of cancer, if they could be operated upon at a sufficiently early stage. But on the other hand it is important to note that the condition of those patients who have survived for any length of time has generally been very miserable, there being great difficulty in deglutition, in consequence of the large opening in the neck, which could not be closed. The voice, however, can be restored by the use of an artificial vocal apparatus, such as was originally invented by Störk.

Malformations of the Larynx.—This will be the most convenient place to mention certain congenital abnormalities of the larynx that occasionally give rise to clinical symptoms. One such appears to be almost confined to female infants, and causes the act of inspiration to be attended with a loud crowing noise, which is nearly constant, continuing even during sleep and after the administration of chloroform, though it is louder during the day. It is sometimes increased by exposure of the body to cold, or in other ways. The noisy state of the breathing is present from the time of birth, but disappears entirely at the end of about a year. Dr Lees has had an opportunity of making an autopsy in a case of this kind, in which death was due to diphtheria; and he found ('Path. Trans.,' 1883) that the epiglottis was folded on itself, like a leaf on its midrib, the arytæno-epiglottic folds being almost in contact. A similar state of things had been seen in the laryngeal mirror during life. The affection is probably not uncommon, for it was the fourth case that came under Dr Lees' notice, and others have been observed by Dr Gee and by Dr Barlow.

Another and a much more serious malformation, described by Mackenzie, consists in a longitudinal bifurcation of the epiglottis, forming two flaps which (in a case that he saw) fell into the larynx, caused constant symptoms of laryngismus from the first week, and death at the end of four months.

Lastly, a congenital band of mucous membrane sometimes connects together the anterior parts of the cords. Mackenzie has recorded such an instance in vol. xxv of the 'Pathological Transactions.' The patient was a young lady, aged twenty-three, who had had complete aphonia from birth, never having cried even as an infant. There seems to have been no dyspnœa. Laryngoscopically the web was seen as a flat membrane during inspiration, but on attempted vocalisation it became folded up, and protruded so as to resemble a tumour, of red colour, and of about the size of a haricot bean. It was excised, and the patient immediately afterwards

spoke, and soon acquired a perfectly natural voice. In a case of Dr Poore's, exhibited at the International Congress in 1881, the patient, a girl of thirteen, could speak, but with a peculiar falsetto tone of voice; she had been liable to attacks of dyspnœa from infancy.

Foreign Bodies in the Larynx.—Not an uncommon cause of severe laryngeal symptoms is the entrance into the upper air-passages of foreign bodies of various kinds. As a rule, such bodies are sucked down within the larynx during a deep inspiration, as the result of coughing, laughing, sneezing, or talking while there is something in the mouth. In children the accident sometimes occurs during the night, in consequence of the foolish habit of sucking a toy before going off to sleep; and it may even happen to an adult who wears false teeth, unless he is always careful to take them out at bedtime. It is surprising what large things will sometimes enter the larynx. Mackenzie relates the case of a boy, aged six, who went to sleep with a toy-engine in his mouth; during the night it was drawn into the air-passages, and tracheotomy had to be performed. The cause of the sudden attack of dyspnœa which had occurred was not discovered at the time, but some months later it was found that the toy was impacted in the subglottic region, whence it had to be removed by thyrotomy.

The symptoms produced by the entrance of a foreign body into the larynx are generally at first very violent; there is a most distressing sense of suffocation, the face becomes cyanotic, the inspiration is prolonged and whistling, a cold sweat breaks out, the patient tears at his throat with the hands, under an irresistible impulse to try to relieve himself of the cause of his sufferings. Such a case may end fatally within a minute or two by asphyxia. But it is an important point that if the air-passages are completely closed there is sometimes no obvious trouble with the breathing; the patient falls dead at once, and it may be only at the autopsy that the real cause is discovered of what had seemed to be an attack of syncope. In the act of vomiting, for example, it may happen that there is inhaled into the larynx a soft, pulpy mass, which entirely fills it.

In many cases a foreign body, having passed into the larynx, at once falls through into the trachea; the early indications of laryngeal irritation then, of course, soon subside, and are followed by a fresh set of symptoms, which will be discussed in a subsequent section (*infra*, p. 64). In other cases, again, the foreign body is quickly coughed out into the mouth, after which it perhaps is swallowed, and ultimately passed through by the bowels. It may thus happen that laryngeal symptoms—brassy cough, more or less dyspnœa, alteration of voice—which were present for some little time, entirely disappear; and it is then difficult to decide whether there is still something in the air-passages or not. Another class of cases in which a diagnosis is not always easy occurs in hysterical women; such persons seek advice for tickling or pricking sensations in the throat, which they declare to be due to the presence of a needle or a pin, or a bristle, but which are really "paræsthesiæ" of neurotic origin.

Finally, when a foreign body is of large size, or when it has pointed ends, it generally becomes fixed in the larynx, and may remain there, as in a case already alluded to, for a great length of time. At any period œdematous laryngitis may set in, attended with severe dyspnœa. But if the foreign body is impacted in the ventricle of Morgagni, there may be merely pain

and cough, with perhaps some degree of hoarseness, so that both the patient and his friends are apt to think it impossible that any such cause for his symptoms can still be present. In such cases the diagnosis rests entirely upon the results of careful laryngoscopic examinations.

When there is a foreign body in the larynx it has, of course, to be removed in one way or another, and generally by surgical interference. In a large proportion of cases tracheotomy is required as a preliminary measure.

Obstruction of the Trachea.—The trachea and the main bronchi are liable to but few affections except such as they have in common either with the larynx alone or with the tubes within the lungs below. And of such affections the clinical importance attaches itself always to the narrower rather than to the wider parts of the air-passages. Hence there is no need to give a separate account of tracheitis; the plastic form of it has been described under croup; the tubercular with tubercular laryngitis, and the catarrhal form will be dealt with under bronchitis.

But there are a variety of diseases which at some point may narrow the calibre of the trachea or of the bronchi, with the result of producing a definite and characteristic group of symptoms. Of these diseases, some have their seat outside the walls of the air-passages; others originally affect the walls of the air-passages themselves; and others, as in the case of foreign bodies, obstruct their channel from within.

Since it is often an accident whether in a given case the part narrowed is the lower end of the trachea, or one or both of the bronchi, it is useless to attempt to separate the affections of these several parts from one another, and the more so inasmuch as two, or even all three of them, are often involved at the same time. All that is possible is to describe the special symptoms which in certain cases indicate that the obstruction is altogether limited to one of the bronchi, leaving the trachea free. The general designation, *obstruction of the trachea and bronchi*, includes the whole group.

A. Of the diseases which, starting *from outside*, may obstruct the air-passages and so cause what may be termed a *compression-stenosis*, the following are the most important:

1. *Tumour of the thyroid.*—It is well known that a bronchocele may compress the trachea in the neck, flattening it usually from side to side, so that its outline comes to resemble that of a scabbard, but also often pushing it out of the straight line or bending it. And it is by no means the largest goitres which are most apt to have this effect; much depends upon the exact situation of the growth, and upon the condition of the overlying muscles, which ordinarily oppose resistance to its extension outwards, but which were in one case found by Virchow to be in a state of complete fatty degeneration. Another point of great importance, for a knowledge of which we are also mainly indebted to this writer, is that the middle lobe of the thyroid when it becomes enlarged, sometimes passes down behind the sternum so as to compress the trachea backwards against the spine. He even maintains that such a "substernal goitre" may be present without there being any obvious swelling of the thyroid in the neck. This, however, seems to be doubtful. Ross, of Zürich, in a very able paper in vol. xxii of the 'Arch. f. klin. Chirurgie,' has drawn attention to a peculiar change in the tracheal cartilages which occurs as the result of the presence of

a goitre, and renders them soft and flaccid. The way in which he recognises this after death is by dissecting off all other structures from the larynx and trachea and then placing them upright; the tube collapses at some one point, bending sharply so that its channel becomes completely closed. A like collapse is believed by him to be the cause of the supervention of sudden fatal dyspnœa as the result of goitre; he supposes that patients instinctively have to maintain the head in such a position as to avoid this occurrence, but that the muscles become relaxed during fainting, or sleep, or chloroform necrosis, or as the result of weakness. Dr Bristowe, for example, relates, in vol. iii of the 'St Thomas's Hospital Reports,' the case of a woman who was admitted for feverish symptoms, but who was one day suddenly attacked with intense difficulty of breathing, followed in a minute or two by blackness of face and insensibility. Fortunately he was close at hand, and finding that she had a tumour in the front of the neck, part of which was evidently cystic, he had this punctured, with the result that two or three ounces of a reddish-brown fluid were removed, and that she was quickly restored to health. In other cases enlargement of the thyroid is due not to a mere overgrowth of its tissues, but to the presence of a hydatid or to the development in it of a malignant new growth, which may then perforate the trachea and protrude into its channel; of this a well-marked instance occurred at Guy's Hospital in 1873.

2. *Thoracic aneurysm.*—Among twenty-seven cases of aneurysm taken without selection from the *post-mortem* records of Guy's Hospital, there was interference with the trachea or with one of the main bronchi in every one (cf. vol. i, p. 991). In fourteen of them the sac arose from the arch and pressed straight backwards upon the lower end of the trachea itself, flattening it, and often adhering very intimately with its walls. Probably in several of these cases the pressure extended also to one or both of the bronchi. But what is surprising is that in no fewer than seven cases the aneurysm seems (from the description given in the case-books) to have pressed solely upon the *left* bronchus; in three of these the sac arose from the summit of the arch on its left side, and pressed mainly upon the upper or upon the anterior surface of the tube; in the other four it came from the descending part of the arch and pressed forwards upon the posterior surface of the air-tube. On the other hand, there were only two cases in which the sac, having its origin in the right side of the arch, compressed only the *right* bronchus. The remaining four cases were examples of what is commonly termed aneurysm of the innominate artery; in them the sac pressed upon that part of the trachea which lies behind the upper part of the sternum or in the root of the neck.

It is difficult to say how many of the twenty-seven cases were characterised by other symptoms which actually did indicate, or might have indicated, the real nature of the disease during life. But in three instances the sac was of very moderate size. One, which flattened the trachea, was a round pouch "of the size of a walnut" (as seen at the autopsy) arising by a definite orifice from the posterior walls of an aorta severely affected with arteritis deformans. Another, which compressed and opened into the left bronchus, was "no bigger than a marble." The third, which likewise interfered with the left bronchus, was "of the size of a small plum."

3. *Mediastinal tumour.*—In the period during which the twenty-seven cases of aneurysm were observed in the deadhouse at Guy's Hospital, there occurred nearly an equal number of cases in which the great air-passages were narrowed by mediastinal new growths; and among twenty-four of them in which details are given as to the exact seat of the lesion there appear to have been eight in which the obstruction affected the lower end of the trachea or both bronchi (sometimes with a great preponderance on one side rather than in the other), six in which it was limited to the right bronchus, ten in which it was limited to the left bronchus. In every instance the new growth invaded the walls of the air-passages, thickening them, and not merely pressing upon them from without. Indeed, there are two other cases besides those already mentioned, in each of which it is expressly reported that although the bronchus on one side was penetrated by the tumour there was no narrowing of its calibre. Among the whole number of cases there seems to have been hardly one in which, if marked symptoms of stenosis were present, there were not also observed other symptoms and physical signs sufficient to show that the obstruction was due to disease beginning outside the air-passages. The pathological reports seem to justify the inference that mediastinal growths seldom invade the trachea or the bronchi at an early period in their development. It must, however, be remembered that they are not likely to be seen in the deadhouse at this stage, since, unlike aneurysms, they do not commonly destroy life suddenly and unexpectedly by hæmorrhage. In an interesting case of lymphosarcoma of the mediastinal glands, recorded by Weil in the 'Deutsches Archiv' for 1874, all the symptoms and signs of tracheal obstruction disappeared suddenly eight days before death; at the autopsy it was found that this was due to the giving way of the softened mass, which must have poured its substance into the air-passages, although the sputa had shown no fresh appearance even under the microscope.

4. *Mediastinal abscess.*—Abscesses of various origin may compress the trachea or a bronchus. A striking case is recorded by Schnitzler, in the 'Wiener Klinik' for 1877. The patient was four years old; an abscess as large as a child's fist pushed forwards and to the right; its starting-point was caries of the second and third dorsal vertebræ.

5. *Caseous disease of the bronchial glands.*—This is commonly given as one of the causes of obstruction of the trachea or of a bronchus, especially in children. Vogel, however, says that although there may be slight flattening or indentation it does not go on to actual stenosis. On the other hand, Widerhofer, in Gerhardt's 'Handbuch,' describes this occurrence, and also cites instances in which after prolonged dyspnœa abscesses dependent on disease of the bronchial glands discharged into the air-passages with relief to the urgent symptoms.

6. *Carcinoma of the œsophagus* is mentioned by Riegel and other writers as an occasional cause of stenosis of the trachea. But although it frequently invades the air-passages, it is rare for it to produce symptoms indicating interference with the entrance of air. As already remarked (pp. 6, 9), it may cause a bilateral paralysis of the abductors of the glottis, and so render the performance of tracheotomy necessary. In all probability the emaciation which is so marked a symptom of œsophageal cancer is attended with a great diminution in the activity of the pulmonary functions.

7. *A dilated left auricle*, secondary to mitral stenosis, may compress the

left bronchus, as was first pointed out by Mr Wilkinson King in 1838, and as may be still seen by his preparations in the museum of Guy's Hospital. Friedreich has recorded an instance in which pressure on the bronchus from this cause was actually diagnosed by physical signs four years before the patient's death; at the autopsy, made by Virchow, it was found that only a very narrow channel was left.

B. Of the diseases which, starting *in the walls of the trachea* or of the main bronchi, may narrow the calibre of the air-passages, some are exceedingly rare. Demarquay, for example, is cited by Riegel as having observed a case in which such an affection arose from ulceration set up by the poison of glanders. Langhans, in vol. liii of 'Virchow's Archiv' (pl. xiii), recorded in 1871 an instance of primary carcinoma, having its origin in the mucous glands of the lower end of the trachea and right bronchus, which destroyed the life of the patient, a man of forty; it appeared as a warty affection of the lining membrane, extending also by infiltration into the muscular and fibrous external coats. Whether a simple local inflammatory process is capable of thickening the walls of the lower air-passages, so as to obstruct their calibre, is doubtful. Andral and Wilks are quoted by Riegel as having reported such cases; but the observations of Wilks, at any rate, refer to syphilitic stenosis only.

Syphilis is, indeed, by far the most important cause of obstruction of the lower air-passages, if the diseases producing compression from without be excluded. Gerhardt, in vol. ii of the 'Deutsches Archiv,' alluded to twenty-two examples of it, of which he had made an analysis; and seven instances presented themselves in the *post-mortem* room of Guy's Hospital between 1861 and 1874. Sometimes the disease is limited to a single spot in the trachea, as in a specimen, taken from a patient of Dr Bright's, which is contained in the museum of Guy's Hospital, and in which opposite the second ring there is a contraction, like that which might have been produced by a ligature. Much more often it extends for a considerable distance along the tube, and it may involve its whole length, and may even be prolonged into one or both of the bronchi.

The bronchi are seldom affected when the trachea escapes. But Wilks, in the 'Guy's Hospital Reports' for 1863, relates a case in which the right bronchus alone was stenosed; and in another case, observed at the hospital in 1875, the lesion was found to have attacked only the left bronchus and the upper branch of the right. Both bronchi were narrowed, with the trachea free, in a third case ('Path. Trans.,' vol. xxviii, p. 336). The mucous membrane is commonly raised into a series of irregular bands and ridges, which Wilks has taught us to regard as the cicatrices of former ulcers. Gerhardt, indeed, has reported a case in which at the time when death occurred from a form of chronic pneumonia there was simply an unhealthy ulcer with raised edges, occupying the right bronchus and one of its branches for about an inch, and exposing the bronchial cartilages. But the view taken by German pathologists generally is that the fundamental lesion is a diffused thickening of the whole tracheal wall, raising its lining membrane into folds and prominences. They describe ulceration, more or less extensive, as of not infrequent occurrence, but they regard this as secondary. It may spread deeply, setting up a perichondritis, and so leading to ossification and necrosis of the tracheal or bronchial cartilages, which may even be exfoliated and discovered by the patient in his expectoration. Or it may penetrate to the

tissues outside the air-passages, forming an external abscess. In a case that occurred at Guy's Hospital in 1865 there was perforation of the aorta, which happened to be highly atheromatous, so that the patient died of sudden hæmorrhage. In other instances, the tracheal rings, instead of being exposed and detached, become atrophied and bent on themselves or dragged one over the other. Whether syphilitic stenosis affecting a bronchus ever leads to its complete obliteration is doubtful. When such a condition has been found it has been regarded as congenital. Thus Ratjen, in vol. xxxviii of 'Virchow's Archiv,' described a case occurring in a man aged forty-nine, whose left bronchus was converted into a fibrous cord for an inch and a half of its length, the corresponding lung being quite airless, while the right lung was enormously enlarged and apparently in a state of true hypertrophy, its air-cells being of normal size. But, as Cohnheim observes of this case, the presence of pigment in good quantity in the collapsed left lung is clear proof that it had at one time been in a functionally active state.

With regard to the time of life at which syphilitic stenosis of the trachea proves fatal, it is perhaps worth noting that the large majority of cases at Guy's Hospital have been in persons between forty and fifty years old, and it has occurred in men far more often than in women. Among the cases collected by Gerhardt there was a far wider range of ages; one was in a patient under ten, and another in a patient under twenty; probably these are the two cases to which he alludes as having been apparently instances of inherited syphilis. Two instances of syphilitic stenosis in children twelve years old are given by Widerhofer.

c. Obstruction of the lower air-passages may be due to a *foreign body*. As already remarked (p. 59), a foreign body which enters the larynx through its upper orifice rarely remains fixed there, unless it is either very large or pointed in shape. Beans, peas, nut-shells, pebbles, small coins, fragments of bone, commonly fall into the trachea. Sometimes they remain free for a time, moving up and down as the patient coughs. One may then be able to feel the impact of the foreign body against the side of the trachea with the fingers placed outside the patient's neck, as was observed by Mr Lucas in the case of a little child with a pebble in its air-passages ('Clin. Soc. Trans.,' xv). Even in that instance there were physical signs which rendered it probable that the pebble lay in the right bronchus in the intervals between the fits of coughing.

As a rule, such bodies soon become fixed in the right bronchus or in one of its main divisions; the reason why they enter it rather than the left bronchus being that the fork between the two is slightly to the left of the middle line, so that the opening into the right bronchus is rather the more direct. Sometimes, however, the left bronchus is the one into which a foreign body passes; and sometimes each bronchus in turn, the body becoming dislodged by cough and falling now into one, now into the other. In certain cases the cause of obstruction is not, strictly speaking, a foreign body at all; it may be a tooth or a fragment of uvula, or a pharyngeal polypus separated by the hand of the surgeon; it may even have found its way into the air-passages by ulceration from the living tissues, as when it is a necrosed laryngeal cartilage, a concretion from a bronchial gland, or an echinococcus vesicle from the liver. An accident which has several times happened after

tracheotomy is that a portion of the tube has become detached from the rest and has dropped into the trachea. Altogether the literature of foreign bodies in the air-passages is very extensive, no fewer than 374 recorded cases having been collected and analysed by Kühn.

As may be supposed, foreign bodies are most frequently found in the air-passages of children and of lunatics. But other patients also, when attacked by sudden and violent symptoms as the result of this accident, may be altogether ignorant of the cause. Hamberger is cited by Riegel, in 'Ziemssen's Handbuch,' as having recorded the case of an old man aged seventy, who fainted after a journey, and was found in a state of dyspnœa, with evident obstruction of the right bronchus. An emetic was given, which led to the expectoration of a green pea swollen to the size of a bean. Subsequently it was learnt that when he was eating peas one day, he had swallowed one the wrong way.

Symptoms of stenosis of the lower air-passages.—Of these the most important is *dyspnœa;* as contrasted with laryngeal stenosis it may be said in the main to be characterised by difficulty of breathing without loss of voice. One must, however, remember that the power of speaking well and even loudly is not in itself proof that the seat of an affection attended with severe distress of breathing is not in the larynx. For in bilateral paralysis of the abductors of the vocal cords, precisely this combination of symptoms is met with, as has already been shown (p. 9). On the other hand, it frequently happens that the voice in cases of tracheal stenosis is weak, thin, and devoid of sonorous quality, from deficiency in the stream of air reaching the larynx from below. A further point to be borne in mind is that syphilitic disease of the larynx is often combined with a like disease of the trachea; a patient may have lost his voice as the result of a syphilitic affection of the larynx, but the dyspnœa from which he suffers may nevertheless be dependent on mischief lower down, so that, if tracheotomy should be performed, the operation may turn out a failure, and afford no relief whatever. Again, in many cases of aneurysm or of mediastinal growth, tracheal stenosis is accompanied by paralysis of laryngeal muscles, as the result of pressure upon one or both of the recurrent laryngeal nerves.

Whether or not the voice is affected, it is therefore essential to make a thorough laryngoscopic examination in all cases of suspected stenosis of the trachea or of the main bronchi. But moreover, it is often possible, especially if the larynx is healthy, to make a direct diagnosis of the nature of a tracheal lesion by examination with the mirror. The lower part of the windpipe, with its bifurcation, and the orifices of the two bronchi, is said to have been first seen in the person of Czermak himself by Elfinger. Türck has pointed out the conditions most favourable to a successful exploration of these parts. The patient should be seated with the body and the neck upright and the head bent slightly forward, the object being to bring the axis of the larynx and that of the trachea into a straight line. The mirror must be placed against the soft palate, rather further forwards than usual, and with its surface nearly horizontal. The observer should sit at a lower level than the patient. The illumination must be very bright, and the light should be thrown into the mouth horizontally, or rather from below. An aneurysm may sometimes be seen bulging into the trachea, as in a case of innominate aneurysm which was examined by Mr Lane, when house physician at Guy's Hospital. It must not, however, be supposed that a

mere slight pulsation of the lower end of the trachea necessarily indicates a morbid condition, for Gerhardt and Schrötter have shown that such pulsation is present in many healthy persons as the result of the pressure of the great arteries coming from the base of the heart.

Local diagnosis.—An important distinction between stenosis of the lower air-passages and that of the larynx was first pointed out by Gerhardt. It is that in the former affection the larynx does not during inspiration make the rapid and extensive movement downwards which occurs when the larynx is itself the seat of obstruction to the entrance of air. According to this observer, if with severe stenosis the range of descent of the larynx is not more than one centimetre, one may confidently assert that the disease is either in the trachea or possibly in both bronchi, but not in the larynx. He also remarks that the position of the patient's head differs in the two sets of cases. When the obstruction is laryngeal, the head is thrown backwards as far as possible. When it is tracheal, the head is stretched forwards, and the chin slightly depressed, so as to relax the trachea. The character of the dyspnœa in stenosis of the lower air-passages is in the main inspiratory, like that in laryngeal stenosis. It is less often extreme in degree, on account of the greater calibre of the trachea as compared with that of the glottis. Consequently, the breathing is not usually greatly reduced in frequency; nor are the lower ribs and the other unsupported parts of the chest walls very much sucked in. But should the disease go on to actual suffocation, all these phenomena may be as marked as they possibly can be in any case whatever. On the other hand, there is generally from an early period the loudest and most noisy stridor, or (as the French term it) *cornage*. It is heard not only through a stethoscope placed over the trachea or over the back of the neck, but also more or less on auscultation over every part of the chest, drowning the normal breath-sounds. Indeed, it is commonly obvious to everyone standing near the patient. According to Gerhardt, the only cases in which any safe conclusion as to the seat of the obstruction can be drawn from observations as to the spot at which this sound is heard loudest through the stethoscope, are those in which this spot is directly over the trachea in the neck. When there is stenosis of the lower part of the trachea, it often happens that the sound is audible with greater intensity over the larynx than over the sternum. Sometimes a râle is constantly discoverable over some particular point in the trachea. A sign to which Demme has drawn attention is that in prolonged cases of constriction of the lower air-passages the circumference of the upper part of the thorax becomes lessened.

In most cases of stenosis of the trachea, the dyspnœa undergoes aggravation from time to time, there being paroxysms of the most extreme distress which are attended with cyanosis, and one of which generally at length proves fatal. It was formerly supposed that the cause of such attacks was paralysis, or perhaps spasm, of the vocal cords from implication of one or both of the recurrent laryngeal nerves, but Dr Bristowe, in an admirable paper in vol. iii of the 'St Thomas's Hospital Reports,' showed the incorrectness of this opinion. They are probably due either to acute swelling of the mucous membrane at the seat of pressure, or to an accumulation of mucus there which cannot be dislodged, or perhaps in part to spasm of the muscular tissue of the trachea itself. It is important to notice that no relief is to be expected from the performance of tracheotomy.

The patient commonly complains more or less of subjective sensations of

oppression of the chest, of soreness behind the sternum, or of actual pain. There may or may not be cough, with expectoration of mucus, perhaps tinged with blood, according to the nature of the disease which produces the stenosis.

In contrasting the physical signs of obstruction of one bronchus with those of stenosis of the entire lower air-passages the first point to be remarked is that much depends upon whether the obstruction is complete or partial. In the former case there is absence of vesicular murmur over the corresponding side of the chest, with impaired movement of the ribs and of the diaphragm, deficient vocal fremitus, and a normal percussion sound. After a time the side may actually be found to have fallen in, and to measure less than the other side. In the latter case a snoring, whistling, or humming sound may be heard over the root of the lung between the scapula and the vertebræ, or there may be moist sounds there. A thrill may sometimes be felt with the hand placed upon the surface of the chest.

One clinical peculiarity of the obstruction of a main bronchus caused by a foreign body is that it is far more sudden, as well as more complete, than that due to any other cause. Consequently its effects may be supposed to approximate more closely than those of any other morbid condition likely to be observed in man, with those of the plugging of a bronchus by wedges of laminaria which were studied by Lichtheim in a series of experiments on rabbits recorded in vol. x of the 'Arch. f. exp. Pathologie.' The opposite lung in these experiments became enormously distended. Very often it gave way, so that pneumothorax resulted; even when this did not occur the animal usually died within twenty-four hours. What proved that the rapidly fatal issue was immediately dependent upon the state of this lung rather than of the one which was deprived of air, was that no such consequences followed when the pleura was laid open on the side of the obstructed bronchus. A bean or a pea is capable of swelling, like the laminaria plugs used by Lichtheim, although more slowly; and his results are worth bearing in mind, because it may be that in the failure of all attempts to extract a foreign body from a bronchus, to admit air into the pleura might sometimes be a justifiable procedure; even if it did not prolong life it might greatly relieve the dyspnœa.

Sequelæ.—Every form of disease producing obstruction of the lower air-passages is liable to be attended with inflammatory changes in the pulmonary tissue, as well as in the walls of the air-passages themselves. Thus when an aneurysm has pressed upon the trachea, or upon a bronchus, I have repeatedly seen the mucous membrane ulcerated and some of the cartilages exposed and partially detached, even though there may have been no indication of an approaching rupture of the sac. Stenosis of a bronchus, from whatever cause, is not infrequently accompanied by dilatation of its branches within the lung. Purulent fluid is apt to accumulate in them, and the result is the occurrence of more or less extensive pneumonia, which often goes on to gangrene. A foreign body fixed in a bronchus often sets up ulceration and sloughing of the part of the tube against which it presses. Sometimes this ends in perforation of the pleura, with pneumothorax, and the foreign body itself may become loosened and fall into the serous cavity. Sometimes it leads to a pneumonia which may spread from the root of the lung far into its substance. The occurrence of fœtid expectoration, and the development of the appropriate physical signs may reveal these various changes, but in some cases they are first detected in the *post-mortem* room

there having been no suspicion of them during life. Even after expulsion or removal of the foreign body, it sometimes happens that the case nevertheless ends fatally as a consequence of the pneumonia that had been set up; but happily this sometimes subsides and the patient makes a permanent recovery.

With regard to the *diagnosis* from one another of the several affections that may cause obstruction of the lower air-passages, it is worth remembering that the two diseases in the course of which stenosis of the trachea is most apt to occur without the presence of any other symptoms are syphilis and thoracic aneurysm. When the obstruction is limited to a bronchus, aneurysm is still probable, especially perhaps on the left side; a mediastinal growth is a more likely cause than a syphilitic stricture. The possible presence of a foreign body must never be disregarded, especially if the symptoms have come on suddenly.

The *duration* of syphilitic disease of the trachea after symptoms have set in ranges, according to Gerhardt, from two months to four years. That of a compression-stenosis, from whatever cause, would probably be found to be confined within comparatively narrow limits of time. Foreign bodies sometimes remain for a very long period—for months, even for years—in the lower air-passages, and yet are after all expectorated.

As to the *treatment* of the various affections that may cause stenosis of the trachea or of the bronchi, there is little to be said. Whenever there is a possibility that it may be due to syphilis, mercury and iodide of potassium should be actively employed. Gerhardt relates, in vol. ii. of the 'Deutsches Archiv,' the case of a man, aged thirty-six, who had had constitutional symptoms after a hard chancre eight years before, and who consulted him on account of cough with scanty, muco-purulent expectoration, a tickling sensation behind the sternum, a little alteration of voice, and slight interference with the breathing. These symptoms had been present for about six months. The patient had lost flesh to some extent; his face was somewhat puffy and livid. Nothing could be discovered with the laryngoscope, and only râles behind the manubrium with the stethoscope. A permanent cure was effected by the administration of full doses of iodide of potassium during several weeks. Unfortunately, however, the cases which are usually seen, and in which the affection has already led to the formation of cicatricial bands and ridges, appear not to be amenable to anti-syphilitic remedies.

When there is a foreign body in the air-passages the only proper course is to perform tracheotomy at once. Until this has been done it is not safe to place the patient head downwards on the chance that the body may fall out through the glottis, as happened (but after tracheotomy) in the case of Mr Brunel, which is so graphically told in Watson's 'Lectures.' Nor does it appear to be prudent to administer an emetic, on account of the risk that the body, if dislodged from its position in a bronchus, may become impacted in the larynx and cause suffocation.

DISEASES OF THE LUNGS

SYMPTOMS AND PHYSICAL SIGNS

COMMON SYMPTOMS—*Dyspnœa—Varieties—Phrenic dyspnœa—Cough—Bronchial, faucial, gastric, and cerebral—Pleurodynia.*
PERCUSSION—*History—Methods—Terminology—Physical theory—Tympanitic resonance—Amphoric and cracked-pot sounds—Significance.*
AUSCULTATION—*History—Methods—The respiratory murmur—Bronchial and tubular breathing—Physical explanation—Râles and Rhonchi—The vocal resonance in health—Bronchophony—Pectoriloquy—Ægophony.*
Palpation—Tactile vibration—Inspection and measurement of the chest.

IN passing on to consider the diseases of the lungs, with their bronchial tubes and pleural investment, there are certain symptoms common to many of these morbid conditions which demand notice, and particularly the physical signs elicited by the methods of percussion and of auscultation.

DYSPNŒA.—The use of this term is commonly limited to cases in which a sensation of "shortness of breath" is experienced, with more or less discomfort or distress. But it is better to follow the physiological use of the term, and to understand by it that the respiratory movements are deeper than natural, or more frequent, or both deeper and more frequent, without regard to whether the patient is or is not conscious of any disturbance of his breathing.

It is a remarkable fact that persons affected with extensive disease of the lungs, provided that such disease develops itself gradually, may continue to breathe as slowly as in health, and with no more effort, so long as they are at rest. The amount of oxygen supplied to the blood is no doubt considerably reduced under such circumstances; but they manage to make it suffice for the wants of the system. It might be imagined that a deficiency of oxygen would probably interfere with the due completion of the chemical changes in which this element is concerned; that sugar, for example, would be likely to appear in the urine, and that urea would to a greater or less extent be replaced by less perfectly oxidised bodies, such as uric acid. But a series of experiments on animals recorded by Senator in 'Virchow's Archiv' for 1868 appears to show that this is not the case; and his conclusions are quite in accordance with clinical experience. In reality, the body adjusts its requirements to the necessities of its condition. In all probability one reason why persons affected with chronic bronchitis or other pulmonary disease almost always grow thin is that they instinctively learn to take very little food. But a far more important method of adjustment seems to be the avoidance of all bodily effort. So soon as such a patient begins to walk, especially on rising ground, dyspnœa sets in. Muscular exertion at once involves a demand for more oxygen than is contained in

his arterial blood. So, again, the supervention of pyrexia in a case of this kind necessarily leads to a disproportionate increase in the rapidity and in the depth of the breathing. In illustration of this principle Cohnheim instances the remarkable subsidence of dyspnœa which often occurs immediately after the crisis in acute pneumonia, before the affected lung has even begun to recover itself.

Sometimes, however, dyspnœa of a very marked kind arises without reason to suppose that the oxydation of the blood is at all defective. This is the case, for example, in *diabetic coma.* And a distressing shortness of breath may be the earliest symptom of which the patient is conscious in the course of chronic *Bright's disease.* Of this a very striking instance came under the author's notice some years ago. He was one day seeing his out-patients at Guy's Hospital, when the attendant nurse asked him to listen to her chest, because her breathing had become so difficult and laboured that she felt unfit for any exertion. After the most careful investigation, nothing amiss with either the lungs or the heart could be detected. Then, as she said she was thirsty, her urine was examined for sugar, and the result being still negative, it was tested with nitric acid, which brought down a large quantity of albumen. A few months later dropsy set in, and her case soon ended fatally.

Another form of dyspnœa, independent of any disease of the thoracic organs, appears to be of *nervous* origin. It is often ascribed to hysteria; but, according to Walshe, it is not always accompanied by other signs of that disease, although he has never seen it except in the female sex.

This will be the most convenient place for a description of a peculiar form of dyspnœa which depends upon *paralysis of the diaphragm.* The characters of this affection were first recognised by Duchenne, and they deserve careful study, because its true nature is very likely to be overlooked. So long as the patient is at rest his breathing is perfectly easy. But the slightest effort at once begins to distress him and to increase the frequency of his respirations; when he walks he experiences a sense of suffocation as soon as he has made a few steps; in mounting a staircase, and even in speaking, he is obliged to stop every instant to take breath. When he sighs he feels as though the abdominal organs were being drawn up into his chest. The act of defæcation is much embarrassed. His voice is weak; there is more or less difficulty in coughing and sneezing, because he cannot take the deep full inspiration which is a necessary preliminary; so that even a slight attack of bronchitis is attended with great danger. If one looks at the surface of his body while he breathes, the characteristic indication of paralysis of the diaphragm is generally at once apparent. During inspiration, when the ribs rise and the chest expands, the epigastrium and the hypochondriac regions become drawn in; during expiration they are pushed forwards. In other words, their relation to the thoracic movements is exactly the reverse of what it normally should be. Sometimes it is not so easy to see the alteration as to feel it with the two hands placed just below the cartilages of the ribs. If only one side of the diaphragm is paralysed, as is sometimes the case, the corresponding hypochondrium may be drawn in while the other one protrudes in the natural manner.

Among the examples of this affection recorded by Duchenne, there are some in which it appeared at an advanced stage of progressive muscular atrophy, others in which it was associated with paralysis of many other muscles as the result of lead-poisoning, and one in which it was hysterical. Walshe says that he has seen it in a well-marked form as a sequel of diph-

theria. Erb cites Oppolzer as having observed it at the age of puberty without any cause being discoverable; he also says that it may be due to the influence of cold upon either the phrenic nerve or the substance of the muscle itself. Another cause which is mentioned by Duchenne on the authority of Aran, is the extension of inflammation from the peritoneum or from the pleura. And he gives a case of empyema in which the muscular tissue of the corresponding side of the diaphragm was of an orange-yellow colour, and in which the fibres microscopically had undergone complete fatty degeneration.

It appears hazardous to diagnose paralysis of the diaphragm in every case of thoracic disease in which one or both of the hypochondriac regions are drawn in during the act of inspiration. That is no infrequent occurrence in a great variety of circumstances, and is often due to a mere inaction of the muscle, which surely ought to be distinguished from paralysis. The application of electricity seems not to be likely to help in clearing up the difficulty, for in all cases in which the diaphragm has been observed to be paralysed, it appears to have retained its power of responding to faradic stimulation of the phrenic nerves. The best method of stimulating these nerves is, according to Duchenne, as follows :—by two fingers, placed just outside the edge of one sterno-clido-mastoid muscle, the skin is first drawn slightly inwards; they are then separated, leaving between them an interval, upon which a small conical metal rheophore is pressed, so as to be just over the spot where the phrenic nerve lies upon the scalenus anticus. The rheophore is now given to an assistant to hold, and the same procedure is repeated on the opposite side of the neck. When both rheophores are fixed the operator takes one in each hand. He passes through them an interrupted current, which should instantly give rise to a contraction of the diaphragm, shown by the abdominal walls being pushed forwards, while the lower ribs are separated from one another. Sometimes, however, the platysma interferes with this result, contracting with such force as to jerk the rheophores out of position. And sometimes it is necessary to shift them a little from spot to spot before one can succeed in acting on the phrenic nerves. Erb recommends a different method; he places one pole upon the neck, and the other over the attachment of the diaphragm to the costal cartilages.

Whatever position may be adopted for the rheophores, the stimulus should be so used that the resulting contraction of the diaphragm may fall in with the natural respiratory movements. The current should be stopped as soon as the muscle has acted, and a few seconds alter it should be re-applied.

This procedure seems to possess considerable therapeutical value. By means of it Duchenne succeeded in completely restoring the functions of the diaphragm in a man named Bonnard, who had advanced progressive atrophy of other muscles, but in whom the paralysis of the muscle in question was as yet recent and incomplete, as was shown by the hypochondriac regions receding only when he breathed deeply. After a few weeks of treatment he became able to ascend stairs and to walk long distances without discomfort.

Cough, as is well known, is produced in the following manner :—A deep inspiration is first taken, the glottis is then closed, and, a sudden expiratory effort being made, the glottis is allowed to open, causing a loud sound and allowing a blast of air to pass out, which may carry with it any secretion or other substance present in the air-passages. In describing laryngeal diseases we have already seen how they may modify the cha-

racters of cough, giving it a hoarse, or rough or metallic quality, or rendering it almost noiseless. But in its ordinary forms cough is a symptom of affections of other parts rather than of the larynx itself, and, indeed, may almost be taken as an indication that the larynx is healthy.

The nervous mechanism by which cough is effected is reflex in its action. As a rule, the irritation which gives rise to it starts from the respiratory mucous membrane, as is evidenced by the consequent expulsion of mucus or pus in greater or less quantity. But sometimes the most violent and repeated efforts of coughing bring away nothing. The cough is then said to be *dry ;* and in the last century the distinction between a "dry" and a "humid" cough seems to have been regarded as one of the most fundamental points in regard to chest complaints. It is, however, quite possible for the air-passages to contain mucus which is too viscid and too firmly adherent to be expectorated ; and probably what is still more frequent is that some part of the respiratory mucous membrane is affected with slight catarrh, and that this condition either itself constitutes an "irritation" or else renders the surface sensitive to the passage of air over it, or to the disturbance produced by the laryngeal movements in breathing or speaking. But, on the other hand, there is no doubt that the starting-point of cough is sometimes altogether outside the air-passages, and, as may well be supposed, the recognition of this fact is of great importance in medical practice. The question has been worked out in experiments upon animals by several physiologists, one of whom was Kohts, of Strasburg, whose observations appeared in 'Virchow's Archiv' for 1874. In regard to cough, as to all other reflex phenomena, although positive experimental results are of great clinical value, negative results prove very little. For under morbid conditions afferent nerves may transmit impressions with more than usual energy, or reflex centres may be unduly excitable, so as to be stimulated by impressions which normally should not disturb them.

The following appear to be the chief varieties of cough which have to be recognised, apart from affections of the respiratory organs :

1. *Throat cough.*—Kohts found, both in animals and in man, that irritation of the pharynx had the effect of producing cough in many individuals, but not in all. There is therefore no theoretical difficulty in admitting that catarrh of the fauces may be attended with cough, without there being a corresponding affection of the larynx ; but the parts being continuous it must always be difficult, if not impossible, to say that this is actually the case, especially as Kohts showed that the glosso- and arytæno-epiglottic folds and the lateral edges of the epiglottis were among the most sensitive structures of all, so far as the production of cough is concerned. It is a somewhat different question whether a relaxed and elongated uvula frequently gives rise to cough by coming into contact with the parts behind the base of the tongue. Dr Mackenzie speaks of this as giving rise to a "distressing tickling cough continuing all day," and some surgeons have adopted the practice of snipping off the uvula whenever a patient has complained of such a cough, for which no other cause could be discovered. But while this treatment sometimes succeeds (as, for instance, in cases recorded by Dr Garrett, of Hastings, in the 'Lancet' for 1872), its failures are at least as frequent.

2. *Ear cough.*—That cough can be excited by irritation of the external auditory meatus had been known long ago, but it was generally forgotten until Dr Cornelius Fox drew attention to it in the 'Lancet' for 1867. He examined a number of persons, and found that this peculiarity

ex sted in about one among every five or six. He mentions the case of a gentleman who experienced a feeling of irritation of the larynx and had a violent suffocating cough, whenever he introduced a toothpick into the left ear; in him, too, a somewhat similar action was capable of being exerted in the reverse direction, for long-continued singing would cause him pain in the ear. Dr Fox shows that the ear may sometimes be the starting-point of a cough under such circumstances that the relation may be overlooked. Thus a healthy-looking woman, aged fifty, had for eighteen months had a most distressing cough. As she was deaf in the right ear the meatus was examined, and was found to contain a hard plug of cerumen, and to have a small ulcer in its floor. Almost immediate relief to the cough followed extraction of the wax and the application of nitrate of silver to heal the ulcer. In a patient of Mr Toynbee's a cough was cured by the removal of a piece of necrosed bone from the external ear. It is obvious that a foreign body, such as a bead, might have a similar effect. Dr Fox is no doubt right in maintaining that the afferent nerve in all such cases is the auriculo-temporal branch of the fifth, and not (as had been suggested) the minute auricular twig of the vagus.

3. *Tooth cough.*—Dr Fox incidentally mentions that it is well known to dentists that the stump of a tooth may be the starting-point of a cough, and he also refers to cough in infants during the first dentition as ceasing when the gum lancet is used. In investigating an obscure case, therefore, one must not fail to examine the teeth.

4. *Stomach cough.*—In the last century it was a favourite dogma that dry cough, and even humid cough, are very often produced by disorder of the digestive organs. The most recent exposition of such a view is to be found in 'Copland's Dictionary.' But, as so often happens in like cases, what has long since ceased to be taught by the faculty has become an article of faith among the public. Thus mothers still commonly refer to the stomach coughs in their children which are really due to catarrh of the upper air-passages. Or, committing a fatal error, they set down to the same cause the dry cough of early phthisis, attended (as it often is) with nausea and loss of appetite and pain in the side. In his lectures on the pneumogastric nerve Dr Habershon takes up only this side of the question, and no one is more likely to have met with examples of stomach cough, if it were really of frequent occurrence. It has been stated that the characteristic sign of a cough due to gastric irritation is either that it comes on when the stomach is loaded with a full meal, and disappears after the completion of digestion, or else that it occurs chiefly when the patient is in bed at night. The second of these criteria corresponds well with the fact that persons in whom intermission of the pulse and palpitation of the heart are caused by irritation of the stomach experience these symptoms when they lie down more than when they are sitting or standing. Kohts, however, in his experiments, failed altogether to excite cough by irritating the stomach. He cites from 'Brücke's Physiology' a case in which a boy coughed day and night with the utmost violence and obstinacy until he vomited, whereupon the cough at once ceased; but he adds that Brücke, who himself made the observation, believed the starting-point of the affection to have been, after all, something in the air-passages, which became dislodged when the stomach expelled its contents. Another instance, quoted from Professor Leyden, is that of a patient who had repeated attacks of biliary colic, and who every time became affected with dry cough and with pain in the right hypochondrium twenty-four hours before

the jaundice set in. Walshe says that he has known the trifling irritation due to the presence of an *Ascaris lumbricoides* keep up reflex cough for several weeks.

5. *Centric cough*—Kohts found that he could sometimes excite cough in animals by mechanical or electrical stimulation of the floor of the fourth ventricle, and he thinks that the centre for this reflex act is situated rather above that for respiration. In hysteria, as is well known, a hard, dry, barking cough is common, and this may be supposed to be concentric in its origin. A remarkable instance of this was recorded by Dr John Harley in the 'Med. Times and Gazette' for 1863. The patient, a girl aged fourteen, uttered a short bark seventy times a minute without intermission, so that, according to calculation, she must have coughed 40,000 or 50,000 times in the course of the day. She had had the cough a fortnight when she came under observation. She was treated with valerianate of zinc and with a cold douche and frictions to the spine, and in three days the cough ceased. A very similar case, in a child aged eight, was described by Dr Whytt more than a century ago under the name of nervous cough. A remarkable feature in each of these cases was that the cough ceased instantly when the patient lay down. Dr Whytt made an elaborate series of investigations into the effects of posture upon his patient, finding (for example) that the cough did not return when she sat up in bed so long as the feet were extended straight out, but that as soon as they were inclined at an angle she began to cough. He also observed that putting the feet in hot water at once arrested the cough.

PAIN is a symptom of various thoracic diseases, but it may also occur in the same places when it is the sole indication that anything is the matter with the patient, and when therefore it can only be regarded as a substantive affection.

Sometimes, perhaps, the seat to which pain is referred is the interior of the lung itself. Walshe speaks of "pains deeply felt within the chest, and shooting in the direction of the pulmonary branches of the vagi and sympathetic," as existing "independently of any other deviation from health not only local but general." He also refers to "various anomalous and more or less painful sensations, felt deeply within the chest by phthisical patients."

But in the large majority of cases thoracic pain is referred to the chest walls, and especially to one or both of the infra-mammary or infra-axillary regions. Various names are given to pain in these situations, according to the views held with regard to its nature. Walshe describes in succession three separate affections, which he terms "pleurodynia," "thoracic myalgia," and "intercostal neuralgia." The distinctions which he would draw between them seem to be chiefly in reference to the intensity and duration of the pain, to its being accompanied by superficial tenderness, and to the presence or the absence of the "points douloureux" of Valleix. But in discussing neuralgia in general, we have found reason to doubt the value of any such differences (vol. i, p. 418). Walshe limits the term *pleurodynia* to attacks of pain of extreme severity, generally setting in suddenly and lasting only a short time. Of it he says first that it is "an actual rheumatism of the walls of the chest, affecting their muscular and fibrous textures," and then, a little further on, that "nerve-fibres are implicated, and that rheumatic neuralgia of the intercostal nerves forms an element of it." Is it not clear that the distinction is only arbitrary? The term "pain in the side," *pleurodynia*, is probably best used as a comprehensive name.

Another difficulty, generally ignored by systematic writers, but of great importance in practice, is to determine whether the respiratory organs are concerned in the production of a pain in the side, or whether it may not depend upon disorder of the heart or of the stomach. The stomach seems to be very frequently its starting-point when it is on the left side, which (according to Walshe) is the case in the majority of instances in pleurodynia; and another frequent cause is ovarian irritation, especially in women who are hysterical. The spine, too, must be thought of, even when the pain is unilateral; and we must remember that a pain in the side, if recent, may be the precursor of an attack of shingles.

Again, it is necessary to bear in mind the possible presence of disease or injury of a rib. In July, 1877, the author was consulted by a lady, the wife of an old schoolfellow, who told him that, having had a cough all the previous winter, she had one night felt something crack in her left side while she was coughing. Ever since then she had suffered from a continuous gnawing pain there. On examining the side there was considerable enlargement of one of the lower ribs, which seemed to be clearly the callus of a fracture. Under suitable treatment she got well, but for as long as six months afterwards she still experienced some pain in coughing, which, however, was no longer limited to one spot and extended as high as the shoulder. She also said that she sometimes felt pain in the side towards night when she was fatigued, and that changes of weather seemed to increase it. Probably the fracture of a rib in coughing happens very rarely indeed. In a lecture reported in the 'Lancet' for 1882, Mr Marshall relates the case of a woman, aged thirty-five, who in the severe weather of the spring of 1881, owing to exposure to draughts, caught cold, shivered, and was attacked first with pain in the left side and then, a month later, with equally severe pain in the right side. In the previous year she had had acute rheumatism, and this had also been attended with pains in each side alternately. Her case was regarded as neuralgic, and powerful remedies were administered, but with only temporary benefit. At length she came to Mr Marshall, who found two firm oblong swellings, one along the lower border of the right fifth rib, and the other at a corresponding spot upon the eighth rib. When they were pressed upon she experienced very acute pains shooting through to her back. They gradually softened into abscesses and were opened, when parts of each rib were found to be eroded and softened. Ultimately some pieces of dead bone came away and she did perfectly well. In another instance, also recorded by Mr Marshall, an abscess, evidently connected with disease of a rib, arose in a patient who had phthisis. Syphilitic periostitis is another affection that must be borne in mind, although it is much less common in the case of the ribs than of the sternum. Mr Marshall speaks of it as occurring nearly always in women.

Methods of Physical Examination of the Chest.—Even now, when more than half a century has passed since the introduction of percussion and auscultation into England, the history of the discovery of these methods of diagnosis is full of interest. For one cannot help feeling that, beside directly revolutionising the then existing knowledge of diseases of the chest, they also gave so powerful an impulse to the scientific spirit among medical men that they have indirectly brought about changes scarcely less important in every other department of medicine.

Percussion.—This method was discovered in the middle of the last century by an Austrian physician, Auenbrügger, who published in 1761 his 'Inventum novum ex percussione thoracis humani abstrusos interni pectoris morbos detegendi.' This method, however, seems to have been adopted by Stoll alone among contemporary physicians of eminence, and it had passed into complete oblivion when Corvisart in 1808 brought out in Paris a translation of Auenbrügger's work, with commentaries of his own, based upon extensive practice at the Hôpital de la Charité. It was introduced into this country in 1825 by Sir John Forbes.

The way in which Auenbrügger performed percussion was by striking the chest directly with the fingers "brought close together and stretched out straight." He also directed that a glove should be worn, and that the patient should be clothed in a shirt drawn tightly over the chest. Even now the whole of the fingers of one hand, with their tips brought to a level, are sometimes used when one wishes to ascertain roughly and rapidly what is the state of the back of the lungs, but for more delicate percussion such a method is altogether inadequate.

In 1828 Piorry, who was himself afterwards physician in the Charité at Paris, published a work upon what he termed "mediate percussion." This he described as consisting in the use of a thin plate of ivory, called a *plessimètre*, to be held by the left hand in contact with the surface of the chest, while a gentle blow is given to it with the tip of the right forefinger, or with the tips of the fore and middle fingers. Since that time a hammer, which is known as a *plessor*, has also been introduced. On the Continent these instruments are more or less employed and also in England for teaching a class. But in this country the usual practice is to simplify Piorry's method by using the left fore or middle finger in the place of a plessimeter. It is curious that in his original work Piorry himself speaks of that plan as having been already adopted by certain English and American physicians who had attended his lectures.

It must not be supposed that percussion in this simple form is very easy. On the contrary, a great deal of practice is necessary to enable one to obtain with certainty correct results. Clinical clerks commonly continue to make blunders in percussing long after they have mastered the difficulties of auscultation. It is very important that the blow should come from the wrist, that it should be sharp and sudden, so as not to damp the sound which is produced, and that the fingers at the moment of striking should be as nearly vertical as possible. The amount of force that should be employed, and the extent to which the finger should be raised before striking, vary with the thickness of the soft tissues over the part of the chest which is to be struck, and every physician, even without being aware of it, modifies his manner of percussing in different patients and upon different regions of the chest according to what experience has taught him to be necessary in order to elicit the best possible sound under various conditions. As a rule, percussion can be practised by a skilled observer without causing any sensation that is complained of by the patient as being painful, but in delicate women, and even in some very thin men, the sternum and the ribs may be so exquisitely tender that a satisfactory sound can be elicited only with great difficulty. Sometimes cough is excited by every attempt at percussion, and a forcible blow may even lead to blood-spitting, so that it is well to abstain as far as possible from this method of physical examination when there has been recent hæmorrhage from the lung.

We must not imagine that in different persons one can always elicit the same sound by percussion of the chest, if the organs are healthy; nor,

again, that the sound ought to be the same over different parts of the chest of any one individual. But, in health, the range of possible sounds is almost limited to a simple series. One extreme is heard where a thick mass of solid tissue lies behind the ribs, as over the centre of the heart just below the fourth left rib, or over the liver at about the right seventh and eighth ribs. This sound is said to be "dull."* The other extreme is heard where the ribs cover a thick substance of lung, as in front below the clavicles or behind below the scapulæ. This sound is commonly called "clear" or "resonant."† Both sounds are difficult to describe satisfactorily, but they are easily recognised in practice. With regard to the names for them, it is important to bear in mind that "resonance on percussion" means altogether a different thing from what is called *resonance* in works on acoustics. Such a technical and conventional way of using the term would no doubt have been better avoided, since it is apt to engender confusion; but in England it has been universal, and perhaps it is not worth attempting to alter it. Between the extremes of dulness and resonance there are all gradations, for which the expressions "partial dulness," "incomplete resonance," "muffled resonance" are employed. These varieties of percussion-sounds are heard at the borders of a resonant or dull area, especially where the lung overlaps a solid organ with a thin edge. At such points many different sounds are obtained according to the amount of force used in striking. A gentle blow elicits a sound only from the parts immediately below the spot which is struck; a more forcible blow affects deeper parts as well. Thus it is usual to speak of "superficial" and of "deep percussion." But it must be remembered that in "deep percussion" the sound is also modified by the structures which are laterally adjacent, so that a resonant area interferes with the sound yielded by a dull area on forcible percussion, although a dull area does not interfere with the sound yielded by a resonant one. For example, by "deep percussion" over the heart one can often elicit a more or less resonant sound even when no lung covers the heart; but on deep percussion over the lung it would not be possible to detect any degree of dulness, however slight, as the result of the presence of the heart or of the liver by the side of the lung. When a solid organ is overlapped by lung, it is often essential to employ deep percussion as the only means of detecting the distance to which the solid organ extends. But under other circumstances superficial percussion should be used when the object is to map out the relative positions of the viscera. It is otherwise when one wishes to determine the presence or absence of disease of the pulmonary tissue. In such cases one has to ascertain by repeated trials what amount of force is necessary to bring out an altered percussion-sound most distinctly.

As a rule, it is by comparing the two sides of the chest together in the person under examination, rather than by an absolute standard, that one judges of the results obtained by percussion. But if both sides are diseased this method may altogether fail. And even when only one side is diseased it is necessary to have some kind of standard, since without it one could not tell, by percussion alone, on which side the disease lay. Now, as already stated, the percussion-sound in health is different in different individuals. The thinner a man is, the more "resonant" his chest is likely to be; in thick-set, very muscular, or very fat persons it is sometimes by no means easy to elicit a clear sound anywhere, particularly over the backs of the

* Dulness or flatness on percussion: *Fr.* Matité. *Germ.* Dämpfung.

† Resonance on percussion: *Fr.* Son Clair. *Germ.* Sonorer oder heller Schall.

lungs. The differences in pecussion-sound at different parts of the chest in health depend upon obvious anatomical conditions, but they nevertheless require careful study in actual practice. In front the sound is modified by the position of the liver and of the heart on the right and left sides respectively; and immediately beneath the clavicles it is more resonant near the sternum than it is further outwards. Behind, in the suprascapular regions, one sometimes has to use a good deal of force in order to elicit anything but a dull sound; and even between the scapulæ the sound generally becomes gradually more resonant as one passes downwards. Below the scapulæ the sound is generally little less clear than in front; the resonant area on the left side extends about a finger's breadth lower than on the right side. While the back is being percussed the patient should have his shoulders drawn forwards as much as possible, crossing his arms over the chest. In this way the "interscapular region" is increased and the "scapular" regions are much diminished.

In a former paragraph the variations of the percussion-sound in health were said to be *almost* limited to a simple series. The qualification was necessary for two reasons. 1. When the blow is struck upon the clavicle or the sternum or upon one of the ribs or rib-cartilages, a high-pitched tone is added to the sound, giving it what is termed an "osteal" character. 2. In a very young child, especially when it is screaming, one sometimes elicits what will presently be described as a "cracked-pot sound."

With regard to the theoretical interpretation of the sounds generated by percussion there have been great differences of opinion. Even now scarcely any two writers seem to express the same views. Nothing is more confusing than to read in succession three or four works, and to attempt to compare together the statements contained in them, even as to some matters of fact, such as the pitch of particular sounds. And the variations in nomenclature are absolutely bewildering. Dr Walshe insists that the use of the terms "dull" and "resonant" or "clear" is inaccurate, because they represent conditions which, instead of being "simple, are in reality made up of several elements, capable of separate analysis;" and of course it is true that writers on acoustics, in enumerating the properties of sound, make no such distinction. But the analysis which Dr Walshe himself has attempted throws less light on the subject than we might expect. The view which appears the most satisfactory—if we may assume its theoretical accuracy, as to which few are competent to form an opinion—is that enunciated by Dr Gee. He declares that the terms in question are perfectly capable of scientific definition. According to him, dulness means *absence of tone;* a part is dull when the sound which it yields on percussion is a mere noise, without any regular succession of impulses such as constitutes tone. On the other hand, varying degrees of clearness or resonance correspond with the admixture, in different proportions, of noises and tones. No percussion-note is ever perfectly pure; and thus beyond the resonance yielded by the healthy chest of even the thinnest person, there are "hyper-resonant" sounds which may accompany certain diseases.

Another question upon which writers differ is as to the seat of the vibrations causing the tone which is elicited by percussion over the healthy chest. Dr Gee refers this tone to the middle-sized and largest bronchia; following Wintrich, he thinks that the pulmonary vesicles and the bronchioles are too small to yield it. But it certainly seems to be a great obstacle in the way of the acceptance of that view that in bronchitis resonance is seldom, if ever, impaired, however completely the tubes become filled up with pus or mucus. I suppose that Dr Gee would attribute the *noise* or toneless part of every

percussion-sound to the thoracic walls, seeing that its amount, in proportion to that of the tone, varies with their thickness and with the extent to which they are loaded with fat. But Dr Bristowe believes that the whole of the sound is "mainly due to the vibration of the thoracic walls alone." He assumes that "so much of each half of the thorax as bounds lung tissue vibrates bell-like when any part of that half is struck, and that the impure musical sound which is elicited comprises a fundamental tone due to the vibration of the whole or a large portion of the side, and harmonic tones due to the vibration of aliquot parts of it." It is a strong argument in favour of this doctrine that deformity of the chest, without any apparent thickening of the parietes, may give rise to absolute dulness on percussion, notwithstanding that the lung beneath is quite healthy: when there is lateral curvature of the spine, for example, the rounded projection formed by the ribs on one side of the back generally, if not always, yields a dull sound. Another argument is afforded by a peculiar alteration in the percussion-sound beneath the clavicle, observed when a certain quantity of fluid is effused into the lower part of the corresponding pleural cavity. No explanation seems so satisfactory as that suggested by Dr Bristowe, namely, that the vibrating area is diminished, and that consequently it yields a fundamental tone which is raised in pitch. Now, the subclavicular percussion-sound is not affected in the same way when there is pneumonia of the lower lobe of the lung. One must therefore assume, if one adopts Dr Bristowe's view, that hepatisation of the lung, notwithstanding that it gives rise to a more or less dull sound when percussion is made directly over it, does not, like pleural effusion, completely damp the vibration of the corresponding part of the chest wall and prevent it from joining with the rest of the side in emitting a tone when a distant part is struck. One point, however in which it is less easy to agree with Dr Bristowe is his rejection of the distinction between "superficial" and "deep" percussion. He evidently thinks that his doctrine is incompatible with the admission that a percussion-sound can in any way be modified by the presence of solid matter within the chest, except in so far as it is in direct contact with the inner surface of the chest wall. But about the reality of this distinction there is surely no doubt.

We have already spoken of the chest as being sometimes *over-resonant*; a better term than *tympanitic*, which has often been employed as meaning the same thing. Originally a percussion-sound was called tympanitic when it was such as would be yielded by an abdomen in which the intestines are distended with gas; for tympanites has, since before the days of Hippocrates, been a name for that condition. And, as a matter of fact, the sound generated by percussion over an emphysematous (or over-distended) lung, when the chest walls are rounded and thin, often approaches, if it does not actually reach, a tympanitic quality. But there are other cases in which a tympanitic is far from being identical with a merely over-resonant percussion-sound. We now reach a point which has been a stumbling-block to all writers on percussion, and about which the author always felt the greatest difficulty, until he had carefully read Dr Gee's work. We have seen that, according to this writer, *dulness* means an absence of the tone which would be yielded by percussion over healthy lung, and its replacement by a mere confused noise. The *pitch* of this tone, within certain limits, varies in different individuals. But here comes the point which seems essential to a right understanding of the matter. It is obvious that the absence of the tone yielded by percussion over the healthy chest is by no means incompatible with the

presence of other tones having a different origin. Thus, although dulness and over-resonance are at opposite ends of the scale, it is quite possible for a sound to be at the same time dull and tympanitic, if beside the toneless noise yielded by the chest walls when there is solid matter beneath, it includes a tone due to the vibration of some part of these structures, or of air in a space within the chest.

Dr Gee classifies percussion-sounds containing adventitious tones as follows, according to their pitch. Those which are highest pitched he calls *Osteal* after Piorry, because they are yielded by the hard solid tissues, cartilage, and bone, as has already been mentioned. Those which are somewhat lower were termed by Dr C. J. B. Williams *Tracheal* or *Tubular*, being more or less like the note yielded by the trachea on percussion when the mouth is a little open. Lower still are tones which Dr Gee proposes to call *Sub-tympanitic;* they are such as are usually yielded by percussion over healthy lungs in their natural state of distension. The lowest-pitched tones of all are the *Tympanitic*, and are heard on percussing a distended stomach.

There are still certain modifications of the percussion-sound which are noticed under special circumstances; they are (1) the "amphoric note" or "metallic ring," and (2) the (likewise metallic) "cracked-pot sound."

The *amphoric percussion-note** is described by Dr Gee as consisting in an "overtone existing either apart and alone, or as a harmonic superadded to the fundamental tone, which itself may either be clear or muffled." It is commonly heard when percussion is made over a stomach distended with air. It may also be elicited by giving a sharp fillip to the cheek when it is blown out to a certain point. It is higher in pitch than an ordinary tympanitic note.

The *cracked-pot sound*† is exactly like the chinking of coins, or the sound produced by clasping the hands loosely together and striking them upon the knee. Probably it always depends upon the propulsion of air out of a space through a more or less narrow opening. It was originally noticed by, and received the name of *bruit de pot fêlé* from, Laennec.

Lastly, it must be stated that, besides the sound which is yielded by percussion and which is audible to bystanders as well as to the physician, he is himself conscious of differences in the degree of resistance offered to his fingers as he taps, and that this is often of considerable assistance in enabling him to draw correct inferences from his observations.

As to the *practical significance* of the various modifications of sound elicited by percussion of the thorax, it is not necessary to say much in this place, since the subject must be considered in discussing each of the diseases of the bronchi, of the lung, and of the pleura. But, in general, it may be laid down that *dulness* on percussion may mean either of two things: (1) consolidation of the pulmonary tissue, from whatever cause; (2) compression of it by fluid or otherwise. As a rule, the diagnosis between these various causes is mainly based upon other considerations independent of percussion. But the dulness resulting from the presence of liquid in the pleura has the peculiarity of being more complete or absolute than that which arises in almost any other way; and the sense of resistance is also greater, especially when the quantity of liquid is large. A *tympanitic sound*, if it be too well marked to be due to emphysema of the lung, can hardly depend

* *Fr.* Bruit de pot fêlé. *Germ.* Zischender oder klirrender Schall (Geräusch des gesprungenen Topfes).

† *Fr.* Son argentin, son métallique, son humérique. *Germ.* Metallklang, Amphorischer (d. h. krugartiger) Schall, Metallish klingender Schall.

upon anything except the presence of air in the pleural cavity—though it is perhaps right to mention another very rare affection, namely, diaphragmatic hernia, with escape of the stomach through the diaphragm into the chest. The various combinations of dulness with tympanitic, subtympanitic, tracheal, and osteal tones are met with chiefly when there are cavities or vomicæ in a part of the lung which is separated from the surface by a thickened and adherent pleura or by a layer of consolidated pulmonary tissue, or when air is present in a pleural cavity of which the walls are indurated. The significance of the amphoric or metallic ring and of the cracked-pot sound will be fully considered elsewhere.*

This is the most convenient place to mention an effect of "immediate" percussion, which is not infrequently seen in emaciated persons, namely, the production of contractions in muscles which receive the tap, especially in the pectoralis major. A rounded knot rises up at the spot which is struck, and from this a smaller ridge spreads away, wave-like, along the fibres in both directions. This "idiomuscular contraction" has been supposed to be characteristic of phthisis. But the fact is that it occurs in wasting diseases generally, when the muscles are in a condition of physiological "exhaustion."

AUSCULTATION.—As Dr Gee remarks, it is a very interesting fact that this, a still more important method of physical examination than percussion, arose directly out of Corvisart's researches. Among those who followed the practice of that professor at the Paris Charité were two friends, Bayle and Laennec. In endeavouring to distinguish between active and passive enlargement of the heart when percussion showed its size to be increased, they were in the habit of carefully observing the character of its impulse; and they became accustomed to apply the ear to the cardiac region for this purpose rather than the hand. One day (about 1815) Laennec was consulted by a young woman who had the general symptoms of disease of the heart, but in whom, as she was fat, he was unable to feel the impulse satisfactorily. For reasons of delicacy he was hesitating to put his hand to her chest, when he remembered the fact that by applying the ear to one end of a plank one can hear the scratch of a pin at the other end. So he took a quire of paper, and rolled it very tight. And then, placing one end on the precordial region, and leaning his ear on the other end, he found, to his surprise and pleasure, that he could actually hear the beating of the heart more plainly than when the ear was in immediate contact with the thoracic wall. He soon began to employ the new method of investigation, which he termed *l'auscultation médiate*, in phthisis and in other pulmonary diseases, read a memoir on the

* Before leaving the subject of percussion, it is well to repeat that the student who aims at more than educating his ear to appreciate differences of sound and learning their empirical significance, must remember that "dull," "resonant," and "tympanitic" are conventional terms, best defined, practically, as those elicited by percussion over the forearm, the right axilla, and the stomach respectively; but that each of these sounds, and the many others to be heard by percussion of the healthy as well as of the diseased body, consist of a fundamental tone with endlessly varied harmonic or discordant overtones, and that each note may vary as follows: (1) in loudness or amplitude of vibrations; (2) in pitch or number of vibrations per second; (3) in duration; (4) in quality or *timbre*.

A clear sound (*son clair, heller Klang*) is opposed to a non-resonant or toneless one (*mat, dumpf*), a high or treble to a low-pitched or bass, a tympanitic on the one hand to an osteal or sternal sound which, though resonant, is very short, and on the other hand, to a long but completely toneless one (as explained by Stokes, and more fully by Gee); and lastly (according to Skoda), a loud and long percussion-sound (*voller Schall*) to a short and feeble one of somewhat high pitch (*leer*).

subject in 1818, and published his great work in the following year. In 1826 he died of phthisis, at the age of forty-five, having almost worked out the subject, so far as concerns affections of the lung.

In his examination of the heart Laennec was led astray by imperfect knowledge of its movements and its sounds in health. He failed to appreciate the condition now familiar as regurgitation or incompetence of a valve, and only recognised obstruction. So that Corrigan, Hope, Hodgkin, and Tweedie in this country, Fauvel in France, and Traube in Germany, had much to correct and complete.

But with regard to the auscultation of the lungs, Laennec left little for later observers to do. Appreciating the importance of combined percussion and auscultation, he was also well acquainted with the morbid anatomy of the diseases to be recognised. He showed how to distinguish by physical signs, peripneumony, emphysema, pleuritic effusion, and pneumothorax, and he classified and defined the most important variations of the respiratory murmur and the chief adventitious sounds which he called generally *râles*. He, like other pathologists at that time, was not aware of the importance of bronchitis (*catarrhe pulmonaire*) as a separate disease, and the very term was still unused, but he describes the signs of œdema of the lungs and of phthisis almost as we should describe them now, and points out the existence and significance of the curious sound called "metallic tinkling."

It is remarkable that of the very few references to sounds heard by listening over the chest which have been traced in medical writings of an earlier date, two are in the works of Hippocrates, the father of medicine; namely, the well-known allusions to the leather-like creaking of pleural friction and to the succussion-splash. The only other notice of any sound produced in the lung is, according to Dr Gee, one relating to pneumonic crepitation in van Swieten's 'Commentaries,' published in 1774.

The stethoscope.—In examining the backs of the lungs, we still sometimes apply the ear directly to the surface of the chest, or rather so that only a towel or the patient's nightdress separates one's ear from his skin. In this way one can rapidly judge of the state of a large part of each lung; and it is often more convenient than using an instrument when the person is very ill and has to be lifted up in bed by attendants. But in most cases we employ some form of stethoscope. That which was originally designed by Laennec (*le cylindre*) has long ago been discarded; it was long and heavy, of a clumsy shape and needlessly elaborate in construction. What is now used is generally a light hollow stem of wood or metal, having a flat ear-piece at one end and spread out into a concavity at the other end, which is placed upon the spot to be examined. In applying it one must be careful not to hurt the patient by pressing too hard or by bearing unevenly upon one side of the rim. The ear must always be moved to the stethoscope and not the stethoscope to the ear.

Many years ago a stethoscope was introduced in which the chest piece and an ear-nozzle were connected by a flexible tube, and it is still used by some good auscultators. It is more convenient than the binaural form for auscultation of the sides and back of the chest without raising the patient.

Recently, however, it has become the fashion to employ a stethoscope with a flexible stem and with two long metal and ivory ear-pieces, one of which is introduced into the meatus of each ear, and is held in position by an elastic band. To the use of this binaural stethoscope, as it is called, everyone should accustom himself, because of the facility with which the back and

sides of the chest can be explored by it in persons who are too ill to sit up. Students like the instrument on account of the loudness with which sounds are transmitted through it. But it is not certain that what reaches the ear is always as clear and distinct as when an ordinary stethoscope is employed. And the slightest movement of the flexible part of the binaural stethoscope produces noises which are apt to be perplexing. Other disadvantages of the instrument are, that it is cumbersome to carry about, that the least contact with the patient's dress causes a loud noise, that it is almost useless unless applied directly to the skin, and that the natural sounds, as estimated by immediate auscultation, are exaggerated. Its advantages are its convenience of application to many parts of the chest, the exclusion of other sounds by both ears being occupied, its allowing the auscultator to see while he is listening (not always an advantage), and its making both respiratory and cardiac murmurs easier to bear.

Hence the binaural stethoscope is preferred by many auscultators in the wards of a hospital. By those who are experienced in each method of auscultation, it is used to determine the existence and character of slight or doubtful cardiac murmurs, it is useful for listening to the trachea and vessels of the neck, to the lungs above the scapulæ and to the abdomen, and it or the single flexible stethoscope is by far preferable for listening to the lung of a recumbent patient.

For examining the heart and the front of the chest without removing a patient's underclothing, the ordinary wooden stethoscope is much the most suitable instrument.

For listening to the back of the lungs while the patient is standing or sitting up in bed, no form of stethoscope is so convenient as the application of the ear directly to the patient's chest only covered by a thin towel.

Whatever form of stethoscope may be employed, one must take care that the patient's clothes do not rest against it nor against the surface of the chest near the spot to which it is applied. And another point to be kept in mind is that if the instrument is placed on the hairy part of a man's chest a crackling sound is often produced which may be very like what will be presently described as crepitation. Guttmann observes that all difficulty from this source may be avoided by wetting the surface.

So far as the respiratory organs are concerned, the practice of auscultation falls under two divisions, that of the breathing and that of the voice.

Auscultation of the Breathing.—*The respiratory murmur and its modifications.*—On listening over the lungs of a healthy person, one hears each time he breathes a soft, rustling, breezy sound. Of this no further description is needed, since the only way of really learning to know it is to hear it again and again and in many different individuals. It is commonly called the "*vesicular murmur*," having been so named when the idea that it arose in the air-cells of the lung was accepted without question; but such an association with a theory which is disputable is an objection to the term. No doubt also Dr Walshe is right when he says that it has often led to an erroneous impression with regard to the character of the sound itself, but there seems to be no possibility now of securing formal acceptance for the name "pulmonary respiration sound," which he proposed instead. "The normal *respiratory murmur*," is on the whole the best term.* The greater part of this sound accompanies the act of inspiration; expiration is either

* *Fr.* Souffle respiratoire, souffle vésiculaire—*Germ.* normaler Lungengeräusch.

altogether noiseless or attended with a much shorter and fainter murmur, which, however, is almost continuous with the inspiratory. In some persons the respiratory murmur is much louder than in others. In children it is particularly loud, so that when under morbid conditions an equally intense murmur is heard in an adult, this is sometimes called "puerile breathing." But in thin adults it is often scarcely less loud, even when all parts of the lungs are healthy. On the other hand, there are some people in whom the act of breathing is attended with scarcely any sound, even when one tries to make them breathe deeply. One must therefore listen over different parts of a patient's chest before one draws conclusions from the degree of loudness of the murmur at a single spot. It is naturally louder where the thoracic walls are thin than where they are covered with thick muscular masses, and over the edges of the lung it is less loud than elsewhere; in other words, its intensity in health is generally proportionate to the degree of resonance on percussion at various parts of the chest. In the same way it is feeble when the walls of the chest are thickly covered with fat.

At the bases of the lungs, especially in persons who are confined to bed by whatever cause, the vesicular murmur is sometimes found to be mixed with a crackling sound, which may be mistaken for crepitation, but which disappears when the breath is drawn deeply two or three times in succession, so that it must be due to slight collapse from disease. Of course it has no clinical significance. On the other hand, there are certain spots at which in most healthy individuals the breathing is attended with a sound that differs from the vesicular murmur. They are (1) the spaces between the scapulæ over an area of variable extent, but somewhat lower in situation on the left side than on the right; (2) the region below each sterno-clavicular joint, especially on the right side and in females; (3) the part corresponding to the spines of the seventh cervical and two adjacent vertebræ, extending sometimes a little outwards towards the suprascapular region, especially the right. The sound heard over these parts is called "*bronchial breathing*," because it is transmitted from the main bronchi. It differs from the vesicular murmur in having a blowing character, in the fact that the expiratory part of it is as loud as (if not louder than) the inspiratory, and in there being an interval or pause between them. A similar sound is heard more loudly on auscultation over the trachea, and still more loudly over the larynx; here it is of a more "whiffing" quality and harsher, and is distinguished as "*tubular*," a term, however, which some writers use as synonymous with bronchial.

To be able, in practice, to distinguish a loud or harsh vesicular murmur from bronchial breathing is the most essential step in auscultation. For the latter sound, with certain modifications of it, may be heard, in disease, over any part of the chest, and constitutes one of the chief signs of the most important pulmonary affections. In bronchitis, indeed, bronchial breathing does not occur, but it may accompany any disease in which the lung is either compressed, or consolidated, or hollowed into cavities. In other words, its range is generally conterminous with that of percussion-dulness, though the two phenomena do not always accompany one another in each individual case. The modifications of bronchial breathing concern its quality. A blowing character belongs to them all; each of them consists of an inspiratory part and of an expiratory part, separated by an interval. What distinguishes them is that the blowing sound is more or less hollow. In its most extreme form it has an echoing character and resembles the

sound produced by breathing into a large empty glass bottle; it is then named *amphoric.** When it is whiffing but without a "hollow" quality it is by most writers termed *tubular*, although others, as above mentioned, employ "tubular breathing," and "bronchial breathing indescriminately."†

It must be understood that between these several modifications of bronchial breathing all gradations exist, so that it is often difficult to know whether to call a sound "tubular" or bronchial, tubular, or "amphoric." But there are nevertheless good grounds for maintaining, as far as possible, the distinctions between them; and a point of some importance is that if tubular or amphoric breathing is heard, at whatever part of the chest, there can be no doubt that disease is present, even though the spot should be one in which bronchial breathing is audible in health. It is a good rule to spare the more striking epithets. The usual error of beginners is to mistake a loud or harsh respiratory murmur for bronchial breathing, to call bronchial breathing tubular, and tubular breathing amphoric.

With regard to the physical causes of the vesicular murmur, of bronchial breathing, and of its various modifications up to amphoric, there has been much speculation. Of late the tendency has been to apply to them strictly the theory of the *veine fluide*. According to this theory, a blowing sound is generated whenever a fluid (whether liquid or gas) passes from a narrow space into a wider one. Now, during inspiration, this condition is fulfilled at two points in the respiratory tract: (1) when the air enters the trachea from between the vocal cords; (2) when it emerges from the extremity of each bronchiole into the ampulla formed by the air-sacs around. On the other hand, during expiration, the only point at which a *veine fluide* can be formed is at the upper orifice of the larynx; but since the false vocal cords form a lip on each side, the resulting sound should be audible not only above the spot at which it is formed, but also below. It is obvious that these facts accord perfectly with what has been stated with regard to the characters of the vesicular murmur and of the sound heard on listening over the trachea and primary bronchi. The former belongs mainly to the act of inspiration; the latter is divided into more or less equal parts, of which one attends inspiration and the other expiration. That the vesicular murmur is generated somewhere below the larynx has indeed been demonstrated, in a series of experiments of which an account was given by the author in the 'Med.-Chir. Review' for July, 1873, by certain French observers, especially Bergeon, Chauveau, and Boudet. They cut through the trachea of a horse and drew the lower end of the tube outwards through the wound in the skin so that no *veine fluide* could possibly arise in it; after this operation they found, on listening over the animal's chest, that the vesicular murmur still remained audible and that its intensity was little if at all diminished. On the other hand, by dividing the pneumogastric nerves in another horse, they succeeded in abolishing the vesicular murmur. The explanation of this is supposed to be that the muscular walls of the bronchioles were para-

* A less marked degree of the same quality of sound is often called *cavernous*, because it is commonly heard over vomicæ, which are sometimes spoken of as caverns in the lung. The term is superfluous and misleading. A vomica may often yield consonating râles, not amphoric breathing at all. Moreover, the names of physical signs should refer to their physical characters, and not to the anatomical conditions they denote, especially when their significance is not constant.

† *Fr.* Souffles bronchique, bronchillaire, tubaire, glottique, soufflante, caverneuse, amphorique. *Germ.* Bronchialgeräusch, amphorisches Athemgeräusch, unbestimmte Athemgeräusche (the last answering to the vesiculo-tubular or subtubular breathing of some English and the respiration rude broncho-vésiculaire of some French authors).

lysed, so that they opened into the air-sacs by wide funnel-shaped mouths—which would yield no *veine fluide*. In this last experiment the laryngeal sounds heard over the trachea are of course still persistent.

The theory of the *veine fluide*, however, in the form in which it is stated by the French experimenters, does not account for the fact that in most persons expiration is attended with a murmur, although a faint one. Moreover, in pulmonary emphysema, the expiratory murmur becomes greatly prolonged and very noisy, while the inspiratory murmur is much diminished.*

The French observers whose views have been quoted suppose bronchial breathing, whether in health or in disease, to consist of sounds generated in the larynx and transmitted downwards with more or less modification until they reach the ear of the auscultator. Indeed, they seem to have proved this experimentally. A horse was affected with pneumonia, and an intense *souffle tubaire* was audible. Chauveau and Boudet cut through the trachea, so as to allow air to enter the lungs without passing through the glottis, and the *souffle* at once disappeared. It would be of great interest to observe the same point in man, either in cases of cut throat or after tracheotomy; but the requisite conditions can scarcely ever be satisfactorily fulfilled, for unless the orifice into the trachea is as wide as the calibre of the tube itself a *veine fluide* may always be generated, at least during inspiration. Stokes, however, long ago pointed out that when the larynx is diseased it is often difficult or impossible to determine whether the lungs are or are not healthy. And it certainly seems very probable that extensive ulceration, destroying the attachments of the vocal cords, may prevent the formation of a *veine fluide* in the air which passes the glottis, and so render impossible the development of bronchial breathing in diseased lung below.

As to the question why laryngeal sounds should be transmitted to the surface of the chest better when the lung is consolidated or compressed than when it is healthy, there has been much discussion, and perhaps even now it cannot be answered quite satisfactorily. It will be better, however, to postpone the consideration of it until we come to the subject of bronchophony. Of the modifications of bronchial breathing, from tubular to amphoric, all that need be said in this place is that the more marked the hollow quality the more one is justified in asserting that a space filled with air, of larger size than even a main bronchus, has been formed in the substance of the lung—unless, indeed, the pleural cavity itself should contain air. But even in acute pneumonia, when there has not been the slightest destruction of pulmonary tissue, it is surprising how hollow the bronchial breathing sometimes is. And Dr Walshe speaks positively of having heard sounds to which he would assign the name of "cavernous," in cases, whether of pneumonia or of pleurisy, in which there was no excavation, and in which the lung was only consolidated or compressed over large bronchial tubes.

Adventitious respiratory sounds.—The sounds derived from auscultation hitherto described have been sounds which are identical with, or more or less modified from, those that can be heard on listening over the healthy

* These difficulties may be met as follows. It is a very slight extension of the results obtained by Bergeon, in experimenting with a tube provided with a lip or rim where it was narrowed to assume that a lip, at the orifice of a contracting cavity, would generate a *veine fluide* in the cavity itself. Now, in emphysema it is fair to suppose that the mouth of the bronchiole projects some distance forwards into the space formed by the dilated air-sacs around it; and even under normal conditions it may do so sufficiently to produce the faint expiratory murmur which is heard in healthy persons.—C. H. F.

lungs or air-passages. But there are other sounds which are altogether adventitious, and have no physiological representatives. Thus in pleurisy a *friction sound* is heard, which of course has its origin on the surface of the lungs; this is almost, if not quite, peculiar to that disease, and may therefore with advantage be discussed in the chapter on pleurisy. With regard to the remaining sounds which are found within the lungs themselves there has unfortunately been much confusion of names. Some writers describe them all indifferently, either as "Râles" or as "Rhonchi," the latter term having probably been originally intended as a Latin equivalent for the French word which was introduced by Laennec, and rendered into English as "rattle" by those who introduced his views into this country. According to this use of the words in question, each of them is applicable indifferently to two kinds of sounds, which are very unlike one another, and which may be distinguished by those of the one kind being *continuous* and blowing, whereas those of the other kind are *interrupted*, and "crackling" or "bubbling" in character. Very often the two kinds are spoken of as being respectively "dry" and "moist;" but this is better avoided, for reasons which will presently appear.

There is, however, another meaning of the term *rhonchus*,* which I believe was first given to it by the late Dr Latham, of St Bartholomew's Hospital, and which afterwards was sanctioned by Sir Thomas Watson. In that sense it is limited not merely to continuous or "dry" sounds, but to a single variety of dry sounds. It is best, as far as possible, to have simple substantives for names, and therefore to follow the writers just named in their use of the term *rhonchus*, especially as there is another term, *sibilus*, which is exactly applicable to the only other "dry" sound known to auscultators. Both these sounds belong mainly to bronchitis, and will be again considered in the chapter devoted to that disease.

If the two continuous (or "dry" and blowing) sounds are to have each a name of its own, the term *râle*† may coveniently be confined to interrupted sounds, of which, however, there are several varieties. They occur in almost every disease to which the lungs or the air-passages are liable. They are generally attributed to the disturbance of fluid lying in the bronchial tube or in a vomica, or in the pleural cavity, by air bubbling through it; and on that account they are known as moist sounds. But, as we shall find in the case of the "crepitation" heard in acute pneumonia, there have long been observers who have maintained a different opinion with regard to the origin of some at least of these sounds, and have ascribed them to the sudden separation of surfaces that had been in contact, just as one can make a series of clicking sounds by pulling away the lower lip from the gum several times in rapid succession. Moreover, in 1871, Traube, in the 'Berlin med. Wochenschrift,' applied this view still more widely, referring a great number of the râles found even in the larger tubes to the momentary detachment of portions of viscid mucus from their sides, by the air-current passing into or out of them. It is, indeed, difficult to conceive that in such thick fluids as are ordinarily found in the air-passages bubbles can be made and broken with sufficient frequency to account for the abundant râles that are often heard. And, as Traube remarks, râles are often to be detected in cases of pleuritic effusion or of hydrothorax, when there is not the least

* Rhonchus (ῥόγχος or ῥέγκος, snoring) is Laennec's *râle crépitant sonore; Germ.* Knarren, Schnurren.

† Râle or *rasle* corresponds to the German *Rasseln*, and our *rattle*.

reason to suppose that any fluid is present in the tubes, and when therefore they can only be attributed to the separation of the sides of tubes that had been forced against one another in compressed parts of the lungs. He says that he has several times determined the absence of fluid in such cases at an autopsy. A further point to which he draws attention is that a râle can be produced by pressing gently with the stethoscope upon the surface of the healthy lung of a recently killed animal. Wintrich has shown that a like result may also be brought about by inflating the collapsed lungs after death ('Virchow's Hdbh.,' Bd. v, Abth. 1).

The usual mode of classifying râles is by what may well at first sight appear to be an arbitrary principle, namely, according to the impression which they give as to the size of the bubbles which might be concerned in producing them, or rather perhaps of the spaces in which they are found. This distinction, however, of "fine" or "small râles" from "coarse" or "large râles" is one which is in practice very easily drawn.*

Consonance.—But there is another distinction between different kinds of râles which is of far greater importance than that of their apparent size, and which depends upon whether the tubes in which they are found are surrounded by spongy or by consolidated lung tissue. In the latter case they have a peculiar quality which in the former is wanting, and which the ear easily recognises, although to describe it in words is very difficult. One might say that râles heard when the lung is solid differ from other râles in being "bright," "clear," "ringing," or "metallic." But the name which is commonly given to them is that of "consonating" râles, originally proposed by Skoda; and there is no great objection to the continued use of it, even if we reject the doctrine that their peculiarities depend upon *consonance* in the sense to which the word is strictly limited by writers on acoustics. We have already found ourselves obliged to employ the term "resonance," as regards the results of percussion, with an arbitrary technical meaning. And in the same way we may perhaps adopt consonance in dealing with the phenomena of auscultation.

It must be understood that consonating râles are often associated with bronchial breathing, both in its simple form and in its "hollow" modifications, and also associated with the equivalent vocal sign to be described a little further on as bronchophony.

Not only does the "consonating" quality divide râles into two well-defined groups, but it is essential to the definition of certain names which are applied in clinical practice to particular varieties of these. The largest râles of all, when they possess the "consonating" quality, are often called gurgling, as above mentioned; but few would apply this term to the toneless, large, bubbling sounds heard in the trachea, the râles of approaching dissolution, the "death rattle." Gurgling is particularly used to express the large consonating râles heard in a vomica and transmitted through solidified lung. The sounds which are termed crepitant râles (or crepitation) and subcrepitant râles, are "smaller," *i. e.* the interruptions are more frequent and shorter than in gurgling; but they also differ from toneless or non-consonating râles of the same "size" or degree by their "bright" or metallic quality, and they are only heard through solid lung. One particular kind of râle, almost (if not quite) peculiar to an early stage of acute

* The gradation is sometimes made thus: *fine* (or small)—*submucous*—*mucous* râles and then *gurgling;* but "mucous" is a bad adjective; the fluid causing the râle may be pus or blood. "Small," "medium," and "large" râles are sufficiently descriptive terms.

pneumonia, is termed *fine crepitation.* This sound is also consonating and typically bright or musical, and it also is never perfectly heard except when the lung is hepatised. Whether it is a true râle, *i. e.* "moist" sound, is doubtful. It will more fully be described, and its origin discussed hereafter.

To non-consonating râles, such as belong chiefly to bronchitis, it does not appear to be necessary to apply any special designations but those of size or order. The term "mucous râle," which is now little used, was generally confined to such râles as, besides being of medium size, were devoid of the consonating quality.*

Auscultation of the Voice.—If, while one is listening over the lungs of a healthy man one tells him to speak, one generally hears an indefinite humming or buzzing noise. In a woman the sounds come to the ear much more sharply, but still so that one is unable to distinguish the words that are uttered. Only over the upper part of the interscapular region on each side is there in some persons a space in which one can hear the voice clearly, and in which the separate words are perceived almost as they are when one places the stethoscope over the larynx and trachea, but of course with less loudness. Now, in disease the voice may be carried to any part of the chest thus distinctly, and may be heard far more loudly than is ever the case in health. For this "increased vocal resonance," as it is often termed, Laennec invented two names according to its degree of intensity. When the voice, however distinct, gave the impression of still coming from a distance, he spoke of "*Bronchophony;*" when it appeared as though it were formed within the chest immediately below the spot at which the stethoscope was applied he used the term "*Pectoriloquy.*"†

Bronchophony, as a rule, is associated with bronchial or tubular breathing and with consonating râles; pectoriloquy accompanies amphoric breath-sounds and gurgling. In other words, bronchophony attends those diseases, or those stages of disease, in which the pulmonary tissue is airless, whether solidified by exudation or condensed by pressure; pectoriloquy is heard when the lung is hollowed out into a large and empty cavity, or when the pleural space contains air.

Laennec was originally disposed to lay more stress upon auscultation of

* The statements made in the previous paragraphs must inevitably appear perplexing to the tyro; and it would be easy to draw up a far simpler classification of the adventitious sounds heard on auscultation of the lungs. But to every simple classification that has as yet been proposed there are two insuperable objections. One is that in endeavouring to convey to other persons definite ideas as to the auscultatory phenomena observed in a case of pulmonary disease, or in receiving from them the same kind of information, we are at once baffled by uncertainty as to the sense in which we or they employ different terms, unless we are familiar with the various meanings that are assigned to them by practical men. The other objection is that in studying medical literature, as soon as we pass beyond the scope of a single text-book, we fall into the gravest mistakes if we imagine that the language used by different writers has always the same sense. And, as for the future, all experience goes to show that if the most distinguished physicians of the day were to meet week after week until they adopted a uniform terminology, and agreed to impose it on others, nearly every one of them would within a year forget in his own practice to make use of it. The attempt, however, was made at the International Medical Congress at Copenhagen, with what degree of success remains to be seen.

† "En me livrant à des recherches comparatives relativement à la résonnance de la voix chez plusieurs sujets sains et malades, je fus frappé par un phénomène tout-à-fait singulier . . . . Lorsque, tenant le cylindre appliqué au dessous de la partie moyenne de la clavicule droite, je faisais parler la malade, sa voix semblait sortir directement de la poitrine et passer toute entière par le canal central du cylindre. Ce phénomène n'avait lieu que dans une étendue d'environ un pouce carré. Dans aucun autre point de la poitrine on ne trouvait rien de semblable" ('De l'auscultation Médiate,' tom. i, § 22).

the voice than of the breathing; the first section of his great work is headed "Exploration de la Voix." Pectoriloquy was the first-fruits of his great discovery, for he did not introduce the term bronchophony until after the first edition of his work; hence, as Dr Gee remarks, it is not surprising that he always clung to this sign with peculiar affection. It became his object to define it in such a way that it should become an unerring indication of a vomica. For this purpose he added to the characters of what he termed perfect pectoriloquy that of being limited to a very small part of the chest. But subsequent experience has shown that, like all other "hollow" sounds, the most typical pectoriloquy is capable in some very exceptional instances of being generated when there is no cavity of abnormal size within the thorax. Thus pectoriloquy no longer carries the exaggerated importance which Laennec attached to it; indeed, one modern writer, Guttmann, omits it altogether, and includes all degrees of increased vocal resonance under the term bronchophony.

On the other hand, an attempt has of late been made, while retaining the use of the term pectoriloquy, to assign to it a new meaning. It has long been known that under certain circumstances an increase in the vocal resonance is discovered more readily when the patient whispers than when he speaks aloud, and the term "whispering bronchophony" has been employed to indicate that the sign is elicited in this way. But Dr Bristowe proposes that in future the transmission of the whisper should be regarded as the special characteristic of pectoriloquy. By bronchophony he would understand that tones generated in the larynx—by pectoriloquy that the articulate sounds formed in the cavity of the mouth—are conveyed downwards to the stethoscope with abnormal distinctness. According to this way of using the terms, when the patient speaks aloud, and when the words he utters are clearly perceived by the auscultator, both bronchophony and pectoriloquy are heard at the same time. Dr Bristowe's proposal has the advantage of introducing a real distinction into the meanings of the two terms in question, whereas hitherto the difference has been only one of degree. But its adoption would render useless, except to those who kept themselves well informed of changes in the sense of words, all the literature of the subject during the last sixty years. Moreover, it is not suggested that transmission downwards of the whisper enables any conclusion as to the state of the lung to be drawn which cannot be drawn from ordinary bronchophony. The practical importance of whispered bronchophony lies in the fact that its presence is so easily recognised, and is thus a valuable sign when there is difficulty in detecting increased resonance in the ordinary voice.

The question has been much discussed *why* bronchophony and pectoriloquy should be heard when the lung is solid or when it is compressed rather than when it retains its spongy structure. But the answer to this question involves also the explanation of the occurrence, under similar circumstances, of bronchial breathing, of peculiarly bright and clear râles, and of the loud transmission of the cardiac sounds to distant parts of the chest; and the complete physical explanation has not yet been given.* Nevertheless, the

* Laennec was content to assume that spongy lung substance was a bad conductor of sound. Skoda, however, as the result of direct experiments upon the dead tissues, declared the conductivity of hepatised lung to be actually less than that of the healthy organ; but he could not reproduce the condition under which auscultation is practised during life, and I find it very difficult to believe that a homogeneous material should not convey sound better than one which is full of spaces containing air. Thus almost all recent writers reject Skoda's conclusion. But even if we admit that bronchial breathing and brochophony are

differences in the sounds under discussion are readily appreciable by everyone who takes the trouble needful to educate his ear; they mutually check and confirm one another; and long experience has given us confidence in assigning to them a diagnostic significance on which we can depend.

It is necessary to make special mention of one modification of vocal resonance, that which from the time of Laennec has been known as *ægophony*, on account of its resemblance to the bleating of a goat.* Its characters, and the conditions under which it occurs, will be discussed in the chapter on pleurisy; it is sufficient here to state that the presence of a moderate quantity of fluid effusion, forming a somewhat thin layer and separating the lung from the chest wall, is believed to be essential to the production of this sign.†

PALPATION.—By applying the hand to the chest, we are able to compare the extent of movement in respiration of the two sides and of the upper and lower parts of the same side. This use of the sense of touch corrects or confirms the observations of the eye, and applies no less to the exploration of the cardiac impulse than to that of the lungs.

due merely to increased conduction downwards of sounds produced above, we have still to account for the hollow modifications of the breath-sound and for pectoriloquy. Now, Skoda maintained that *consonance* was the cause of all these phenomena. In acoustics, however, consonance has been understood to mean a power of vibrating in unison with some particular tone, or of producing sounds in harmonic relation to it. And it has always appeared to me that in his work on the subject, Skoda does not really limit his application of the term to what is known as consonance in a strict sense. He makes allusions both to the effect of the sounding-board of a guitar or violin, and to the augmentation of the sound produced by a tuning-fork when it is placed upon a table. But each of them is an example of what writers on acoustics have termed "resonance" rather than "consonance." I observe, however, that in Tyndall's 'Lecture on Sound' nothing is said about consonance, everything being included under resonance. Probably, therefore, there is little or no risk of error resulting from our continuing to speak of "consonating" sounds in auscultation, and we need not intend to imply that we adopt a definite acoustical theory as to their origin. It may be sufficient if we suppose that either bronchial tubes surrounded by solidified lung tissue, or the walls of a cavity, are capable (with the air which they contain) of reverberating sounds conveyed to them from above, so that they are subsequently transmitted to the ear of the auscultator with great loudness, or more or less altered in character besides.—C. H. F.

* "De l'égophonie ou de la pectoriloquie chevrotante.—L'égophonie ressemble à la pectoriloquie . . . . La voix plus aiguë, plus aigre que celle du malade, et en quelque sorte *argentine*, produit seulement une illusion telle qu'il semble que quelqu'un parle dans la poitrine du malade. Elle a, d'ailleurs, un caractère constant d'où j'ai cru devoir tirer le nom du phénomène; elle est en quelque sorte, tremblotante et saccadée comme celle d'une chèvre, et son timbre se rapproche également de celui de la voix du même animal. Ce caractère ne présente que des variétés légères dont on peut se faire une idée exacte en se rappelant l'effet que produit un jeton placé entre les dents et les lèvres d'un homme qui parle, celui de la voix transmise à travers un roseau fêlé, ou le bredouillement nasal des bateleurs qui font parler le fameux personnage de tréteaux connu sous le nom de *polichinelle*" ('Ausc. Méd.,' tome i, § 154).

† Dr Stone has recently given an ingenious explanation of ægophony. In a course of experiments with a pitch-pipe placed between the lips of various patients and made to utter a musical note by their drawing a deep inspiration, Dr Stone found that when the spoken voice, or even the whisper, yielded marked ægophony to a stethoscope placed over the affected part of the chest, there was no transmission of the sound of the pitch-pipe. So, also, if the patient could be made to sing or to intone a good musical note, no ægophony was to be heard. And among spoken words a difference was found to exist as to the degree in which they gave rise to this modification of vocal resonance according to the vowel sounds contained in them. The French A yielded hardly any ægophony; it was more marked with the E, still more so with the I, and most of all with U. Now, as is well known, Helmholtz showed that the different vowel sounds are formed by the addition of certain harmonic overtones in varying degrees of intensity to a fundamental tone which may be the same for all of them. And according to Dr Stone the cause of ægophony is that the fundamental tone is intercepted in its passage through a layer of pleural exudation, while the overtones

But there is another way in which palpation affords a physical sign which is associated not with ocular but with auscultatory information. When the hand is placed upon the bare chest of a healthy man, a tremulous sensation is felt, especially if the voice is powerful and low in pitch. The sensation is more "voluminous" (because felt over a large space) but in quality closely resembles that of the *frémissement cataire* described in the chapter on diseases of the heart (vol. i, p. 963). In a woman or a child it is often not to be perceived, and on the right side of the back it is almost always more distinct than on the left side. The presence of fluid in the pleura, even in moderate quantity, invariably annuls this sensation, whereas it is exaggerated by hepatisation of the lung, the other signs of which so closely resemble those of effusion. The practical importance of loss of tactile vibration seems to have been first pointed out in the 'Journal Hebdomadaire' for 1829 by a French physician, Raynaud, who also discovered pleuritic friction sound.

This normal sensation is known as tactile vibration of the voice or *vocal fremitus*. It is increased when the lung is solidified, and is diminished or abolished when the lung is separated by liquid from the thoracic walls. In other words it is increased under the same conditions as vocal resonance on auscultation and diminished or absent when the vocal resonance and respiratory sounds are rendered feeble or entirely lost. In estimating tactile vocal fremitus the whole hand should be laid flat on the patient's chest and he should be directed to speak loudly, slowly, and in as deep a tone as the compass of his voice will allow. As with other physical signs of the chest, comparison of the two sides is invaluable for bringing out the diminution or exaggeration of tactile vibration.

Inspection.—A rough estimate of the shape, size, and movements of the chest are naturally made when one first glances at the patient after he has stripped. But accurate observations are often postponed until after one has listened to the breathing over different parts of the lungs.

It is, however, better, as a rule, when a complete examination is to be made, to begin by carefully inspecting the chest as the patient sits upright and in as easy a posture as may be before the physician. The light should be made to fall first full, and then obliquely on the surface. The shoulders should be covered by a shawl while the front is examined. The number of respirations in a minute should be counted, their rhythm, and especially the length of expiration and the degree of pause between expansion and contraction noted, and the relative movements of the chest and abdomen (denoting the predominance of thoracic or phrenic respiration) should be observed. Next

are allowed to pass and, being heard by themselves, give the peculiar character to the sound. In some further experiments he succeeded in imitating ægophony. This was effected by transmitting the voice through a wide india-rubber tube, over which was placed a bladder containing water. When a stethoscope was applied to the upper surface of the bladder, an ægophonic twang became exceedingly distinct.

Dr Stone's theory of ægophony seems to me to be fully established. And it is of the more interest because it brings into complete accord with the auscultatory phenomenon another physical sign, which (so far as I am aware) had never been thought of in connection with it, but which has long been known to be one of the chief indications of pleuritic effusion, namely, loss of tactile vibration. When the pleura contains fluid the fundamental tone, according to Dr Stone, is intercepted; and this is the one which would be felt under normal circumstances, whereas the overtones consist of waves too rapid to be perceptible to the touch. I am not sure, however, that loss of tactile vibration is to be detected in every case when the voice has an ægophonic character. When the effusion in the pleura is large, tactile vibration is absent and also ægophony; the harmonic overtones, as well as the fundamental tone, of the voice, are both cut off.—C. H. F.

the general shape of the chest should be scanned, in front, behind, laterally, and by looking down upon it from above the patient's head as he sits, so as not only to measure its relative dimensions, but to compare the movements of the two sides.

It is not necessary in this place to go into details with regard to the varieties of shape that the chest may present under different conditions. But it may be noted that bulging or flattening of one of the infra-clavicular regions is more easily detected by standing behind the patient while he is sitting, so as to look downwards over his shoulders. A general enlargement of one side is often easily appreciable by the eye. But one must make quite sure that the patient is sitting or standing perfectly upright, especially if the case is that of a child or of a young woman with a thin flexible spine. In infants Dr Gee remarks that it is a good plan to grasp the chest with the two hands, placing the thumbs tip to tip upon one of the vertebræ. In adults a measuring tape is often used. But, as the same writer remarks, circumferential measurements are apt to be fallacious, because considerable increase in the sectional area of one side of the chest may leave the length of the periphery unaltered, by "the passage of the elliptical form into the circular." It is this which renders his *cyrtometer* so useful an instrument, by which the outline of the chest in any horizontal plane can be accurately determined. It is made of two long pieces of very narrow metal gas-tubing, of an eighth of an inch in diameter, which are fastened together by a short piece of caoutchouc tube, slipped over their ends. The central caoutchouc piece is placed over the spinous process of a vertebra, and the hollow metal rods are then carefully bent round the patient's body, so as to meet over the sternum. It is now easy to remove them without altering their shape; and by laying them upon a sheet of paper one can obtain an accurate tracing, which shows exactly the configuration of the two sides of the chest, and enables them to be compared. One must not forget that the half circumference of the chest on the right side is in many healthy persons greater than that on the left side, the difference being sometimes as much as an inch.

Various instruments, called *stethometers*, for the measurement of the movements of the chest, have been devised by physicians, one of the best being Dr Arthur Ransome's, of which a description may be found in the 'Medico-Chirurgical Transactions' for 1873. But although they have yielded information as to the exact degree of impairment of mobility of different parts of the chest wall in various diseases, it seems doubtful whether any one of them has been employed in actual practice by other observers than their inventors, the reason being that they are troublesome to use, and that they bring to light no facts that may not be ascertained without them.

Nor does it appear that in clinical practice any results worth speaking of can be attained by the use of an instrument invented by Dr John Hutchinson many years ago for the purpose of measuring the amount of air that can be expelled from the chest by the fullest possible expiration. This instrument, which is called the *spirometer*, may perhaps be of value in the examination of recruits for the army, or of "lives" for insurance, by suggesting doubts as to the advisability of accepting such candidates as, for their height, fall very far short of the standard. But there are great practical difficulties in obtaining correct results; very few persons succeed in "blowing" their full amount of air into the instrument until they have had some practice.

BRONCHITIS

General symptoms: cough, dyspnœa, pain—Physical signs: rhonchus, sibilus, râles—Morbid anatomy.

Acute bronchitis—Capillary form—Its symptoms and prognosis—Pulmonary collapse—Its production and relation to pneumonia.

Chronic Bronchitis—Varieties—Sequelæ—Emphysema—Its anatomy and origin—Atrophic emphysema—Symptoms and signs of emphysema—Bronchiectasis uniform and saccular—Fœtid Bronchitis—Ætiology, prognosis, and treatment of Bronchitis generally.

Plastic Bronchitis—Its rarity—Anatomy, course, and symptoms—Treatment.

THE disease which is termed bronchitis is so common, and it is now so familiar, that probably almost everyone is surprised to learn that medical literature contains no allusion to it by name before the publication of works in 1812 by Peter Frank in Germany and in 1814 by Badham in England. Up to that time it had been known as "catarrh," or "defluxion on the breast," while the more severe forms were often designated by the cumbrous name of "peripneumonia notha," invented by Sydenham. It is generally understood to include inflammations of all parts of the air-passages below the larynx. When the windpipe is very obviously affected, tracheitis is sometimes said to be present in addition; but, as might be expected, the artificial boundary lines recognised by the anatomist find no application in clinical practice. In a large number of cases the morbid action reaches to a greater or less extent above the bifurcation of the trachea, yet we need not speak of the disease as anything more than bronchitis. On the other hand, it is usual to exclude altogether such forms of inflammation of the trachea and bronchi as are attended with plastic exudation, or lead to deep ulceration and cicatrisation. These have been already described (see p. 63).

There are few other affections of which there are so many varieties as of bronchitis; and these differ, both in symptoms and in course, to the most extreme degree. It therefore seems hopeless to attempt to describe them in common. The more convenient plan will be to enumerate such of the symptoms and physical signs as belong to them all alike, and afterwards to give separate accounts of the more important varieties.

Common symptoms.—Foremost among the general symptoms is *cough*. This is never absent, and it is often exceedingly severe, and of a very loud, barking, or ringing character. It may consist of isolated explosions, succeeding one another more or less regularly, and sometimes with extreme frequency. Or it may occur in paroxysms, which sometimes end in retching or actual vomiting. It may be worse when the patient lies down, or it may come on especially when he first gets up in the morning, being excited by an accumulation of mucus or pus in the air-passages during the night. Sometimes the irritation which sets it up is definitely referred to some one spot along the course of the trachea, which is felt to be raw or

tender; sometimes there is a vague tickling sensation, which cannot be localised. The characters of the sputum, if the cough leads to expectoration, differ so widely in different forms of bronchitis that it is useless now to allude to them.

Another symptom which belongs to all but mild cases of bronchitis is *dyspnœa*. It depends, in the main, upon the mechanical obstruction to the entrance of air into the lungs, which we shall presently see to be a natural result of the pathological changes in the mucous membrane of the small tubes. Thus it is especially associated with inflammation of the lower air-passages, as contrasted with that of the trachea and main bronchi, the calibre of which is probably never so much diminished, at least in the kind of inflammation with which we are now concerned, as to interfere with the access of air to the pulmonary tissue.

A curious circumstance, to which Riegel seems to have first drawn attention, is that in all affections of the bronchioles the dyspnœa is expiratory rather than inspiratory. Sometimes the act of inspiration is quite short and easy, while that of expiration is difficult and much prolonged; sometimes they are both alike embarrassed; but inspiration appears never to be alone obstructed in bronchitis, as it so often is in affections of the larynx or trachea. The explanation of this special tendency for the expiration to be interfered with, when the smaller air-tubes are inflamed, is by no means obvious.

Another peculiarity of the breathing, which may often be noticed in children, is that each expiration is instantly followed by an inspiration, the pause in the act of breathing taking place at the end of each inspiration and not at the end of the expiration, as it does normally.

In severe cases there is often complete *orthopnœa*, the patient having to be propped up with pillows all night. The reason why it increases his distress to lie down seems to be partly that the weight of the abdominal viscera is thrown upon the diaphragm and renders its descent less easy, partly that the pectorals and other accessory muscles of respiration cannot be so easily brought into play as when he is upright.

When dyspnœa is considerable there is almost always more or less lividity or *cyanosis*. This shows itself upon the face and hands. In extreme cases the face becomes very turgid, flushed, and bloated. The veins of the neck are dilated and throb. The superficial veins of the body generally are fuller than natural.

Pain is by no means constantly present in bronchitis. Many patients, however, complain of a sore feeling behind the sternum, or in the upper part of the chest on either side. Or, again, the harassing cough may give rise to more or less severe myalgia in some part of the thoracic walls. Not infrequently such a pain, muscular in its origin, is felt at the epigastrium. But another cause of pain in this position is fulness of the liver, resulting from obstruction to the venous circulation.

Physical signs.—The signs of bronchitis are less numerous than those of almost any other disease of the respiratory organs. They are mainly auscultatory, the percussion-sound being quite unaltered unless the case is complicated with pleural effusion or with some affection of the pulmonary tissue, such as emphysema, or collapse, or broncho-pneumonia.

With the stethoscope it is found that the vesicular murmur is more or less altered in character, or that it is replaced by, or has added to it, certain adventitious sounds. In some cases, and those not the least grave, the

change in the vesicular murmur is that it is *faint* and *indistinct;* it may be absent occasionally over a part of the lung, from the corresponding bronchial tube being plugged by mucus. In this case one can usually bring it back by making the patient cough vigorously two or three times.

As a rule in bronchitis the vesicular murmur is for a time *rough* and *harsh* in quality, and the expiration may be accompanied by a very similar sound. There is then sometimes considerable difficulty in drawing a distinction from bronchial breathing. This, however, has a more blowing character; and it is strictly limited to certain parts of the chest, whereas in bronchitis the sound is heard over both lungs alike and very widely; true bronchial breathing, except over the sternum and between the shoulders, is accompanied by more or less dulness on percussion, and more or less increase of the vocal resonance. It is a point which cannot be too strongly impressed on those who are learning the use of the stethoscope that neither bronchial breathing nor any of its modifications occurs in bronchitis except when some complication is present. Nor is the transmission of the voice in any way altered.

The adventitious sounds which occur in bronchitis are those that have been already discussed at p. 87, under the names of "rhonchus," "sibilus," and "râles." *Rhonchus* (or "sonorous rhonchus") is a loud snoring or cooing noise, often audible by the patient himself and by those about him, and due to vibrations that can be felt by the hand placed upon the surface of his chest. It is formed in the larger tubes, and in bronchitis its cause is the presence of a mass of viscid mucus partly obstructing the entrance of air, and producing a *veine fluide.* The proof of this is that it can very generally be made to disappear, at least for a time, by the patient's coughing once or twice, and, indeed, it comes and goes of its own accord, being heard first in one part of the chest and then in another, as mucus happens to accumulate in different branches of the bronchial tree.

Sibilus (or "sibilant rhonchus," as some prefer to term it) is a high-pitched whistling sound. It is formed in the bronchioles, and is therefore of much graver import than rhonchus, inflammation of the smaller air-passages being far more dangerous than of the larger. It seems to be due to the narrowing of the calibre of the affected tubes which results from swelling of their lining membrane. Consequently, it cannot be got rid of by coughing, and it usually remains in the same spot for hours or days together.

Many terms have been employed to denote the particular quality of these continuous "dry sounds." Sometimes they resemble snoring, as the term rhonchus would imply, sometimes the hoarse cooing of a wood-pigeon; often they are high-pitched and musical like a box of pipes, sometimes deep in tone like the notes of certain stops of the organ, and sometimes "wheezing," "squeaking," or "whistling."

The *râles* which accompany bronchitis may be of every variety of size. They are not of "consonating" quality, inasmuch as the lung tissue round the tubes in which they are formed still remains more or less spongy. As a rule this distinction is very obvious. In general, if râles are not universally distributed through the lungs, they are most marked over the lower lobes, and behind rather than in front. Signs that might suggest the presence of bronchitis in the upper lobes only—especially if limited to the upper lobe on one side—should always arouse a strong suspicion of phthisis. A point which is worthy of mention is that it is sometimes impossible to detect any

râles in cases in which the profuse expectoration would certainly have led one to expect them. Only "dry sounds" are audible.

Diagnosis.—The symptoms and physical signs above enumerated are not sufficient in themselves to justify a diagnosis of bronchitis. It is necessary to add to them certain negative points by which the presence of other affections of the air-passages or of the lungs is excluded. Particularly in cases of which the clinical history is such as to render it possible that the pulmonary parenchyma may contain scattered tubercles, whether of acute or of chronic development, great caution should be exercised in forming an opinion. Sometimes, but very rarely, the occurrence of secondary nodules of a malignant new growth in the lungs offers another source of error. As a rule, however, the difficulty is not so much in saying that bronchitis is present as in determining whether it is the principal affection from which the patient is suffering or only a complication. For it is exceedingly apt to arise in the course of a great variety of diseases, among which may be mentioned the exanthemata, enteric fever, rickets, organic lesions of the heart, and Bright's disease of the kidneys.

Morbid anatomy.—In all mucous membranes, and in the skin, the morbid appearances produced by inflammation are far less conspicuous after death than during life; and a reason why such should be pre-eminently the case with the bronchial mucous membrane is afforded by the abundance of elastic fibres in its structure. Indeed, it is sometimes far from easy to determine the presence of bronchitis at an autopsy, even when it has been the principal disease from which the patient suffered. A good method of detecting puriform secretion in the smaller tubes is to slice off the extreme edge of the lung, and then to press the tissue upwards towards the cut surface, when a yellow bead appears at each little orifice. But in many cases there is extreme redness and swelling of the mucous membrane, which may have a velvety appearance, while every part of the air-passages, up to the trachea, may be full of a yellow or brownish opaque fluid.

The histological changes in bronchitis have been in recent times particularly studied by Socoleff ('Virchow's Archiv,' vol. lxix), and by Dr Hamilton ('Practitioner,' 1879). The former set up inflammation in dogs and in rabbits by the insufflation of bichromate of potass or of a weak solution of chromic acid; the latter based his investigations upon cases that presented themselves in the *post-mortem* room of the Edinburgh Royal Infirmary. Both observers are agreed that a very early change is the detachment of the ciliated epithelium, which seems to be thrown off in flakes, and which remains absent during the whole course of the disease, to be regenerated when recovery takes place. In a young man who died of opium-poisoning, in from ten to sixteen hours, the ciliated cells were already to a great extent shed, although Dr Hamilton speaks of the morbid process in that case as having been rather acute congestion than actual inflammation. He says that the cells themselves undergo fatty degeneration, which probably destroys many of them. Others, no doubt, are expectorated; others are inhaled into the finer air-tubes, where they may be seen lying in large detached masses among the other catarrhal products. There is an obvious analogy between this exfoliation of the columnar layer of the bronchial epithelium and the separation of the cuticle in the roofs of blisters or vesicles when inflammation affects the skin. During the further progress of the attack, the basement membrane is covered only by a layer of flat cells, from which there project here and there pyriform or oval corpuscles,

of transitional character, which are covered by a more or less abundant mass of leucocytes, embedded in a mucoid fluid. A point on which Dr Hamilton lays great stress is that the basement membrane itself becomes greatly thickened and swollen, apparently as the result of œdema. Both he and Socoleff are convinced that the leucocytes which appear in such large numbers upon the free surface of the mucous membrane are not derived by emigration from the blood-vessels, but are formed by germination from the flat cells that lie immediately in contact with the basement membrane. Socoleff's chief reason for maintaining this opinion was that in animals killed twenty-four hours after the commencement of the morbid process he found leucocytes on the free surface of the mucous membrane, although its substance was at that time entirely free from them. Dr Hamilton insists especially on the difficulty which leucocytes derived from the blood would have in traversing the thickened basement membrane, and on the fact that in his preparations he could discover no indication that this was taking place. But it is perhaps worthy of notice that Socoleff himself figures ciliated epithelial cells having in their interior red blood-discs, which must have made their way through. And one naturally hesitates to accept observations upon deep-seated tissues as overthrowing the results of investigations made upon the cornea and other superficial structures, for the special purpose of determining the nature of the inflammatory process (cf. vol. i, p. 52, *et seq.*).

In all but very early and very slight cases of bronchitis the mucous or submucous tissues are, in their whole substance, more or less thickly filled with leucocytes, which are collected in lines along the lymph spaces between the fibrous bundles and around the vessels. Dr Hamilton is satisfied that these also are in great part derived by germination from the flat endothelial cells of the lymph spaces or from connective-tissue corpuscles. Another very important change occurs in the mucous glands. They become swollen, so as to be sometimes as large as hemp-seeds, according to Riegel. Their epithelium undergoes very active proliferation, and the newly-formed cells become distended with a mucin-holding fluid, and appear to be the source of the mucus that often forms so large a part of the expectoration. This mucus, however, becomes mixed with serous exudation from the walls of the bronchial tubes themselves, and with leucocytes and cells of "transitional" form, as has already been stated.

When bronchitis has existed for a great length of time before death the changes found *post mortem* are somewhat different. The mucous membrane is often pale and grey, with but few vessels visible. In many cases it presents a number of delicate longitudinal ridges, which Rindfleisch has shown to consist of an overgrowth of connective tissue, containing very numerous cells, and bundles of elastic fibres running in various directions. According to Dr Hamilton the muscular coat is sometimes found to be hypertrophied, sometimes atrophied. The cartilages shrink and disappear, the change in them being exactly the same which occurs in cartilage tissue under so many other conditions, namely, the absorption of the matrix from the periphery inwards, with the formation of "medullary spaces" filled with leucocytes. In many cases the mucous glands also are destroyed. At an earlier period their orifices are widely dilated, giving the mucous membrane a finely-pitted appearance when looked at with a good light. And sometimes they become inflamed, forming minute funnel-shaped ulcers.

Of the various forms of bronchitis some run an *acute*, others a *chronic* course.

Acute Bronchitis.—This often affects mainly the larger air-passages, so that it may fairly be called a "tracheo-bronchitis;" and in such cases the inflammation is sometimes derived by extension from the nose and throat. The cough may then be exceedingly distressing, being especially violent when the patient attempts to lie down. He may complain greatly of a sore sensation along the sternum; and pressure upon the trachea may be painful and may at once excite cough. This form of the disease, however, is not dangerous.

Capillary form.—Very different is the course of acute bronchitis when it attacks the bronchioles throughout the lungs, for this may be one of the most rapidly fatal of all diseases. It is distinguished as "Capillary Bronchitis," and formerly as "Suffocative Catarrh."

It usually sets in with a sensation of chilliness, or even with a rigor; according to Niemeyer its onset differs from that of pneumonia in the circumstance that repeated rigors may occur, whereas in the latter disease there is seldom more than one. The degree of pyrexia varies widely; the temperature may range up to 104°, especially in children; more often it is at a lower level; and it does not run any typical course. The head and the upper part of the body become covered with sweat. The hands and the surface generally feel hot, the face is more or less deeply flushed. The pulse is frequent, sometimes so rapid that it cannot be counted. It is often tense and full, perhaps as the result of the obstruction offered by the systemic arterioles to the passage of blood containing an undue quantity of carbonic acid.

But the most prominent symptom of this form of bronchitis is the dyspnœa. The patient sits up, with chest heaving and with nostrils quivering, unable to utter more than two or three words at a time, using his shoulders and arms in violent efforts to breathe. On carefully inspecting the thoracic movements, one finds that there is a great obstacle to the entrance of air into the lungs. The epigastric and the hypochondriac regions of the abdomen recede at every inspiration; in children all the lower ribs and the lower part of the sternum may be forcibly sucked in. The supra-clavicular and the suprasternal spaces also recede, but, on the other hand, as Seitz has pointed out, the upper ribs often remain almost motionless in a position which is that of a forced inspiration, giving to the corresponding part of the chest a vaulted shape.

The cough of capillary bronchitis is often exceedingly harassing. At first it is usually dry, there being nothing in the air-passages to be expectorated. Afterwards it is accompanied by more or less abundant sputum. The bronchial mucous membrane, indeed, when it is inflamed, goes through stages very similar to those that may be observed in the nostril during the progress of a cold in the head. It begins by being swollen and dry, then it pours out a transparent mucous fluid; after a time this becomes muco-purulent, and finally almost pure pus. The dry stage sometimes lasts several days, or even throughout the whole duration of the disease. Thus Dr Latham in his 'Lectures on Subjects connected with Clinical Medicine,' narrates the case of a boy, seven or eight years old, who for six days remained in a condition of extreme suffering, with shrill sibilus audible all over his chest, and then gradually recovered without expecto-

rating anything. A point, however, which must not be forgotten is that infants and young children commonly swallow whatever they cough out of the air-passages into the mouth. In older patients, when sputum first appears, it is as a rule scanty and dislodged with great difficulty, the patient perhaps coughing a number of times in rapid succession, and until he is purple in the face, before he can get relief by bringing up a little translucent pellet of mucus. But in other cases the spitting-jar becomes filled in a few hours with a considerable quantity of a greyish-white glairy liquid, which has numerous air-bubbles entangled in it, and is so viscid that the vessel may be turned bottom upwards without its escaping. Under the microscope this kind of sputum is found to contain remarkably few formed elements. As already remarked, in bronchitis the tubes cease for the time to be lined with columnar epithelium. It is therefore probable that when a few cells of that type are seen in the matters expectorated (except at the very commencement of the disease) they have been derived from healthy and not from inflamed parts of the air-passages, just as flat epithelial cells are often seen which come from the throat or the mouth. Cells of transitional form, however, and mucous corpuscles are present in small numbers, and as the case advances pus-cells abound more and more until the sputum becomes quite opaque and of a greenish-yellow colour. It now comes freely, and with little effort, so that the cough is said to be "loose."

Event.—In many cases, after acute bronchitis has lasted for some time, the quantity of expectoration begins gradually to diminish from day to day; the other symptoms become less and less severe, and presently the patient recovers entirely from his attack. But in other cases the inflammatory exudation accumulates in such large amount as to threaten death by suffocation. Râles then become audible all over the chest, and are so loud that no trace of vesicular murmur can be anywhere detected. Indeed, they are often heard at a distance from the patient, and by those about him as well as by the physician. Still more important as a warning of danger is the supervention of cyanosis: the flushed cheeks, the lips, and the hands assume first a faint lilac, but finally a leaden colour; the blood is no longer duly aerated, and a condition of asphyxia has begun.

Another very grave symptom is the failure of effort on the part of the respiratory muscles; the breathing gradually becomes more and more shallow, until at last it may be represented only by a slight flickering movement of a few of the ribs, or by a faint jerking contraction of the diaphragm. With this, too, the patient ceases to be conscious of the necessity for active breathing. Instead of remaining upright he sinks down in bed, with his head in any position in which it may happen to be placed. His mind may wander for a time and then he becomes unconscious. Sometimes death is preceded by one or more convulsive seizures.

As a rule, if acute bronchitis is to end fatally, it does so in the course of the first fortnight, and in some rare cases the patient succumbs within twenty-four or forty-eight hours. But it not infrequently happens that when the disease has apparently been subsiding favourably, a relapse occurs which puts an end to all hope of recovery. It need not be said that the patient's muscular strength is one of the most important points that one has to take into account in attempting to form an estimate of the probable issue in a case of acute bronchitis. In very old persons the prognosis is always doubtful; it is so likewise in those who are very fat, or who are already weakened by previous illness, or who have progressive muscular

atrophy affecting the shoulder or trunk muscles, or any considerable deformity of the spine or of the chest.

In infants, the chance of recovery is better in proportion to the age ; while the gravity of the disease is greatly increased by the presence of rickets. We must be cautious, however, in giving an unfavourable prognosis in the case of children. It is surprising how rapid may be both the pulse and the breathing, for two or three days together, in those who ultimately recover completely.

Complications.—In many instances of acute bronchitis the digestive organs are disturbed in a manner that is not readily accounted for, the degree of pyrexia affording no adequate explanation of it. The tongue is often coated with a whitish-yellow fur, of surprising thickness. There may be nausea and vomiting, and the bowels may be obstinately constipated. In children it is sometimes difficult to say whether the symptoms belonging to the abdomen or to the chest constitute the more essential part of the case.

But the most important complications of acute bronchitis, when it attacks the smaller tubes, are those that concern the substance of the lungs themselves. One of them is known as *collapse* of the pulmonary tissue ; another is an inflammatory affection, for which the best name appears to be *broncho-pneumonia*. This is distinct in its origin, pathology, and histology from true or acute pneumonia ; but since, unfortunately, that term is applied to at least three separate diseases, it will be more convenient to consider it in the chapter upon pneumonia proper.

Pulmonary Collapse is identical with a state of lung which is seen in infants as the result of imperfect respiration, and which is nothing else than a persistence of the fœtal condition of the tissue. That, however, is properly termed Apneumatosis or Atelectasis (ἀτελής= imperfect, ἔκτασις = expansion). It affects the whole of both lungs if the child has never breathed at all, or parts of the lungs (especially the anterior edges) if it has breathed incompletely, from having been prematurely born, or being weakly, or having its air-passages obstructed by mucus.

A German writer, Jörg, is generally credited with having been the first to point out, in the year 1832, the real nature of the atelectasis, which before was supposed to be congenital pneumonia. And it is commonly said that the patches of collapsed lung also were up to that time confounded with red hepatisation. But Bright in 1828, writing of the morbid appearance found in the lungs of two children who had died of whooping-cough, showed that he clearly recognised the difference ; and Dr Alderson also is said to have drawn attention to it. The distinctions between these two affections are unmistakable. A collapsed part of the lung is, indeed, reddened, and the colour of its cut surface is reddish brown, or, when covered with pleura, reddish purple or violet. A section of it, however, looks perfectly dry, smooth, and homogeneous ; it has not the dull, lustreless, and granular appearance of hepatisation. Moreover, its surface lies below the level of the adjacent air-containing parts of the lung ; if it reaches the free edge of the organ it forms a notch there. Lastly, inflation from the bronchus will usually restore to it its normal appearance.

Sometimes, however, collapsed pulmonary tissue is at the same time œdematous, and then its characters are less marked, its cut surface being moist and emitting serum when gently squeezed.

Another airless state of lung which has received a separate name is the "carnification" caused by compression by pleural effusion; here the tissue is bloodless, as well as airless, and the colour is slaty or mouse-coloured instead of being reddish brown.

The way in which collapse arises was well illustrated in a case which occurred at Guy's Hospital in 1874. A child, aged two years and two months, died four days after the performance of tracheotomy for a chronic laryngeal affection. Upon the under surface of the left lung there was a narrow red line of collapsed lung tissue. This had running through it a tube which (like all the other tubes in the same part of the organ) happened to have become dilated as the result of the chronic obstruction to the child's breathing. That tube was plugged at its upper part by a piece of sponge, about a quarter of an inch long, which had evidently fallen into the trachea at the time of the operation. The limitation of the collapse to the part of the lung served by the obstructed tube was perfect. As a rule, collapse is secondary to closure of the corresponding tube by viscid mucus or muco-pus.

How this brings about the affection has only lately been well understood. Gairdner, in 1850, suggested that the plug acted like a ball-valve, allowing air to escape during expiration, but preventing its entrance during inspiration. But the explanation always seemed unsatisfactory, and would not account for the complete disappearance of the air, inasmuch as the elastic force of the pulmonary tissue and that of the confined air must soon become inadequate to raise the valve. Lichtheim, of Berne, in an important research recorded in the 'Arch. f. exp. Path.' for 1879, showed that in rabbits collapse follows within twenty-four hours the plugging of a bronchus by a piece of laminaria, which becomes swollen, so as to prevent all passage of air in either direction. It is therefore evident that the affection must depend mainly, if not entirely, upon absorption of the air by the blood which circulates in the walls of the alveoli; and Lichtheim gives reasons for believing that the several gaseous constituents of the atmosphere are taken up with different degrees of rapidity, the carbonic acid and the oxygen first, and afterwards the nitrogen. A further result of his investigations is the proof that the elasticity of the pulmonary tissue is not exhausted until it has become completely devoid of air. For without the aid of this elasticity, absorption by the blood must cease before collapse would become complete.

The state in which a lung is found when there has been slight narrowing of the space in which it lies (whether from pleural effusion or enlargement of the heart or pushing up of the diaphragm) is inexplicable, unless it be admitted, as a general principle: that whenever even a small part of the organ fails to be acted on by the forces which are concerned in inspiration, its elasticity brings about a total collapse of its substance, notwithstanding that the tubes which serve it may be patent. This, it must be admitted, is a hard doctrine to accept; but there seems to be no doubt about its truth. Now, in bronchitis, at least in children, it often happens that large portions of the lower lobes of the lungs are unacted upon by inspiratory forces, for (as we have seen) the lower ribs and even the sternum are commonly drawn inwards, instead of rising, during the act of breathing. And the same thing occurs in croup and in other diseases attended with laryngeal obstruction. It therefore appears probable that collapse of the lower and anterior edges of the lungs, which is so often seen under such circumstances, is generally, if not

always, due directly to the cessation of inspiratory traction upon those parts of the organs. And Bartels, as far back as 1860, showed by dissection that it is in fact often impossible to demonstrate any plugging of the tubes passing to collapsed areas of pulmonary tissue. So, again, in enteric fever, we may refer the collapse of the bases of the lungs, which is so commonly seen in the bodies of those who have died of that disease, to the shallowness and imperfection of the respiratory movements. Even when collapse depends upon obstruction of tubes, an important factor in its production is a deficiency of power in the muscles of the thorax. For, with strong muscles, there is not only the chance that a fit of coughing may expel a plug of mucus, but also that a vigorous inspiratory effort may succeed in drawing air into the tissue in spite of it. Accordingly, in adults collapse scarcely ever occurs as the effect of primary bronchitis. Even in children its development is greatly favoured by a rachitic state of the ribs, and perhaps also by the muscular weakness resulting from measles or any other acute disease. And the younger the child the more likely is it to show collapse of the lungs under a bronchial attack.

The view that inspiratory retraction of the lower part of the chest is generally the cause of collapse of the lungs, when the two things are associated together, is not incompatible with the opinion that an extensive collapse due to obstruction of the corresponding bronchial tubes, may sometimes in its turn lead to a falling in of the thoracic walls. Dr Gee, for example, describes unilateral shrinking of the chest as resulting in some cases from collapse of the whole of a lung in consequence of plugging of its main bronchus. Generally speaking, however, the space in the pleural cavity vacated by a collapsed portion of pulmonary tissue is filled up by over-distension of other parts of the organ. And, if an entire lung should become emptied of air, the opposite lung undergoes enlargement and displaces the mediastinum. This condition is sometimes confounded with emphysema, as we shall see further on.

Some writers believe that pneumonia is apt to arise in collapsed portions of lung tissue, in which case the anatomical distinctions between collapse and hepatisation would of course fail. Lichtheim found in his experiments that acute œdema of the affected lung sometimes occurred, so that, although airless, it was bulky, soft, and moist. The retention of secretion in obstructed tubes is suggested by Jürgensen as a probable cause of inflammatory irritation; and Lichtheim, in some instances in which an animal survived for a considerable time after obstruction of the bronchus, describes the lung as looking almost like a sacculated kidney, full of dilated channels distended with pus. But, as a rule, pulmonary tissue collapsed as the result of disease in the human subject remains uninflamed. If there are patches of broncho-pneumonia in the same organ, their presence is merely a coincidence.

So far as appears, collapse, whether arising from bronchitis or from laryngeal obstruction, is always a temporary condition, the affected parts of the lung again receiving air, if recovery takes place from the primary disease. The author has never seen in the *post-mortem* room any appearances that appeared to be traceable to the occurrence of collapse at a former period.

With the exception of the collapse which occurs at the extreme bases of the lungs as the result of inspiratory retraction of the chest walls, this condition, when secondary to bronchitis, seldom affects more than small

portions of each organ, lobules or groups of lobules scattered here and there, some on the surface, others in the interior. Hence it does not often give rise to very definite physical signs; though, if many patches should exist in close proximity to one another, it is possible that there would be more or less dulness on percussion, deficiency of vesicular murmur, and even perhaps bronchial breathing. As regards symptoms, all that can be said is that collapse aggravates the dyspnœa already produced by the bronchial affection.

Chronic Bronchitis.—This, like the *acute* form, varies widely in its degree of importance and of severity in different cases. Some of its mildest forms are seen in children and in young adults, who from time to time have attacks of what is termed bronchial catarrh, until, as they get older, they perhaps ultimately "grow out" of them. Other cases, chiefly in persons advancing in age, take the form of what is called a winter cough. Year after year, during the cold season, these people become troubled with a cough, which leaves them entirely in the summer, and which is attended with more or less free expectoration of a muco-purulent material. Sometimes it occurs only in the morning, when they rise from bed; sometimes it goes on at intervals throughout the day; sometimes it is very bad at night, disturbing their rest. For a time there is not the least dyspnœa. Gradually, however, they find that in muscular exertion, as in walking uphill, or in mounting stairs quickly, the breath becomes short and hurried. Still it is surprising how little heed is paid to such symptoms, which, among the poorer classes, seem to be taken almost as a matter of course. In making autopsies we have repeatedly found the tubes in the lower parts of the lungs filled with pus, and even dilated, and the lungs themselves markedly emphysematous, in the bodies of patients who had perhaps died in the surgical wards of the hospital, and who had never made any complaint of pulmonary symptoms. But after a few years this cough continues even during the summer; and there is constant dyspnœa, the breathing being hurried and wheezing, especially during any exertion. For a long time there is no loss of flesh; but at length wasting occurs, and it may reach an extreme degree.

It is only during the early stages of chronic bronchitis that it is possible for the patient to recover, so as to remain henceforth free from the liability to its return, when exposed to cold or damp. But even in advanced cases, occurring in persons of easy circumstances, the disorder may often be kept at bay by their avoiding all changes of temperature, and spending every winter in a warm climate, or else remaining indoors throughout that season of the year. In this way life may not infrequently be preserved to its natural term.

When death occurs, it is sometimes as the result of an intercurrent acute attack. Indeed, those who suffer from chronic bronchitis are exceedingly apt subjects of the form of the disease, attended with more or less pyrexia, which always cause more or less anxiety, but which may pass off, leaving behind them only an increased susceptibility for the future.

In other cases, chronic bronchitis ends fatally by the supervention of dropsy, exactly like that which attends disease of the heart. The right chambers of the heart are then dilated and hypertrophied; and the trunk and branches of the pulmonary artery are thickened, just as in mitral stenosis. In one instance the wall of the pulmonary artery was actually

thicker than that of the aorta, and it was also atheromatous. Such changes probably never take place until emphysema of the lungs has developed itself; and the obstruction to the pulmonary circulation is sufficiently explained by the defective aeration of the blood which occurs under such circumstances, and by the diminution of the capillary area in the pulmonary system of vessels. But Traube laid stress upon a third factor, namely, the deficiency of the movements of expansion and retraction of the lungs in breathing, which under normal conditions were supposed by him to further the flow of blood through the pulmonary capillaries. Ultimately the liver becomes "nutmeg," the spleen and the kidneys indurated, and the stomach congested. It is, however, remarkable how rarely these results of long and extreme venous congestion are seen in cases of chronic bronchitis with emphysema, compared with their constancy in cases of mitral disease of the heart. One important element in the bringing about these more remote changes is the occurrence of granular degeneration in the muscular substance of the right side of the heart. Another, which has not yet received its due share of attention, is a like degeneration of the diaphragm, as pointed out by the late Mr Callender in the 'Lancet' for 1857, and by Zahn, in vol. lxxiii of 'Virchow's Archiv.'

Certain varieties of chronic bronchitis demand separate mention. One of them is that which Laennec called *catarrhe sec.* A like form of acute bronchitis has been already mentioned. But the cases now referred to are described by Riegel as having a duration of several months, and as often ending fatally, at least in children. The most prominent symptom is a paroxysmal cough, which is so violent that the face becomes purple and the cervical veins swell out as thick cords. Yet there is no sputum, except perhaps a little tough mucus. Pyrexia is very slight, or altogether absent.

Another variety of chronic bronchitis is attended with a remarkable flow of a thin watery albuminous liquid from the mucous membrane, so that the name of *bronchorrhœa serosa* has been given to it. Laennec relates the case of an old man of seventy, who for ten or twelve years spat up such a fluid to the extent of about four pints every day, and yet remained not ill nourished. But in other instances, as Andral pointed out in his 'Clinque Médicale,' extreme emaciation occurs, with weakness and pallor, almost like what might have been caused by profuse hæmorrhage.

Chronic bronchitis, if it lasts for any length of time, gives rise to certain secondary affections of the pulmonary parenchyma and of the air-passages themselves, which affections add greatly to its gravity. They are known as "emphysema of the lungs," and "bronchiectasis" or "dilatation of the bronchial tubes."

Emphysema of the Lungs—or "emphysema," as it is often called without any addition, when there can be no doubt that a pulmonary affection is intended—must not be confounded with that condition of the subcutaneous and other connective tissues which also bears the name of emphysema, and which depends upon an infiltration of them with air as the result of injury to some air-containing structure. What adds to the confusion is that in the lung itself infiltration of air into the subpleural connective tissue does sometimes, though rarely, occur, and may go on until, passing along the root of the lung, the air diffuses itself through the mediastinum, and reaches even

the superficial fascia of the neck and of the chest; this last affection is technically known as "interlobular" or "interstitial emphysema."

A very complete description of emphysema as regards both its anatomical character and its symptoms was given by Laennec; before then it had almost escaped notice. Its real nature was first pointed out by Rokitansky, who showed that it consists in part of an over-distension of the pulmonary alveoli, but also in part of an atrophy of their walls, causing their cavities to run together into irregular spaces, sometimes of very large size.

Over-distension of the alveolar tissue does not in itself justify the lung being called emphysematous. In children who have died after a few days' illness of laryngeal diphtheria, or croup, or acute bronchitis, it is common to find the lungs very bulky, and looking far more open-textured than usual. One is apt to speak of this condition as emphysema. But it is clear that there has been no time in such cases for the occurrence of atrophy of the alveolar walls, and in all probability if the patients had recovered the lungs would quickly have returned to their normal state. Even when some amount of emphysema seems to be really present, it may happen that immediately after an attack of dyspnœa a great apparent increase of the condition can be made out by physical signs, which yet subsides again within twenty-four hours. Hertz, in 'Ziemssen's Handbuch,' mentions the case of an asthmatic patient, aged thirty, in whom he observed such a transitory over-distension of the lungs on several distinct occasions.

Anatomy.—The presence of emphysema of the lungs in the dead body is recognised partly by touch and partly by sight. The substance has a peculiar soft feel, like that of a pillow of down; it scarcely crepitates, if at all, when squeezed between the finger and thumb; and pressure upon its surface readily causes a deep pit, which remains after the pressure is removed. This last circumstance depends upon the loss of the normal elasticity of the pulmonary tissue. Another effect of the same cause is failure of the lungs to collapse when the chest is opened. They often remain fully distended, and the left lung covers the heart, so that scarcely anything is to be seen of that organ. In a case which was observed at Guy's Hospital in 1868, one lung overlapped the other behind the sternum by an inch and a quarter. A similar condition, which must have been pathological, was found in a body which the writer, when demonstrator of anatomy, froze for the purpose of making a transverse section; a wax model of that preparation, which is now in the museum, shows one lung covering the other for some little distance; there was advanced phthisis, and it is very likely that emphysema also existed, although this was not proved to be the case. In extreme instances the lungs bulge in all directions, both during life and after death, displacing the structures around them. Their apices protrude far above the clavicles; and their bases bulge so that the diaphragm from being arched upwards has a flattened upper surface. In two cases, the pericardial sac was pouched inwards at its lower part, so that Dr Moxon described the heart as resting upon and as being separated from the surface of the diaphragm by cushions of lung. Sometimes there are large bullæ or blebs containing air, of all sizes up to that of a walnut or a pigeon's egg. These are seen chiefly along the anterior borders of the lungs, but sometimes also along their inferior borders or near their roots. Not infrequently the ear-shaped process of the left lung shows a more marked degree of emphysema than any other part. But in some instances, even when the lungs are very highly emphysematous, no large cavities are to be seen. The tissue if closely inspected is found to

be full of spaces of the size of small shot or of millet-seeds. One noticeable appearance is a rounding off of the free edges of the lungs, and their outer surfaces often show marks of the ribs, the intercostal spaces having yielded so as to allow the lungs to bulge outwards. Emphysematous lung tissue is of a grey colour, mottled with spots and lines of pigment; it is soft and inelastic but not friable, and the surface is dry and bloodless.

The earliest change in a lung that is becoming emphysematous is, according to Rindfleisch, a dilatation of the infundibular cavities, into the sides of which the alveoli open; according to Hertz, a nearly uniform dilatation of the infundibula and also of the alveoli themselves. Gradually the alveolar walls waste until nothing is left of them but small ridges, projecting a little way into the interior of an oval or rounded space into which each infundibular cavity and its alveoli have now become resolved. After a time the septa between these spaces in their turn thin away and become perforated. Thus the result is a progressive increase in the size of the spaces with a diminution of their number. An aggregation of fatty granules round the remains of the nuclei of the alveolar epithelium is commonly present, and in the 'Med.-Chir. Transactions' for 1848, Mr Rainey maintained that the morbid process concerned in emphysema was primarily and essentially a fatty degeneration; but there is no reason to suppose that this is the case. There is, of course, an enormous destruction of capillaries when the affection is at all extensive. Rindfleisch speaks of the vessels as collapsing until "only a narrow ribbon-like band is left, which may be recognised as an obliterated vessel by its greater transparency amid a dark, often pigmented, parenchyma, and by its uniting with other bands like itself to form the usual anastomotic network." He goes on to say that some relatively wide communications are opened up between the pulmonary artery on the one hand and the pulmonary and the bronchial veins on the other hand. These anastomoses appear in well-injected lungs as peculiar elongated unbranched vascular arches of the same diameter throughout, contrasting very strikingly with a far more numerous assemblage of extremely tortuous and dilated arteries, for whose contents no such supplemental mode of escape has been provided.

Pathogeny.—With regard to the mode of origin of emphysema of the lungs there have been many different opinions.

Laennec's idea was that the tubes in cases of bronchial catarrh being obstructed by swelling, or by an accumulation of mucus, the air which found its way into the alveoli during inspiration became unable to escape during expiration, inasmuch as the expiratory force was less than the inspiratory. In other words, he thought that emphysema was the result of a process, the exact converse of the ball-valve action which, as we have seen, was supposed by Gairdner to be the cause of collapse. It was objected by Louis that the ordinary seat of catarrh is the base and lower part of the lung, whereas the parts most apt to be affected by emphysema are the apex and the anterior margin.

In 1851 Gairdner formulated a very definite theory of emphysema, which he held to arise solely during inspiration. His view was that collapse or reduction in bulk in one part of a lung is a necessary antecedent to the development of emphysema in another part. During inspiration, when the chest becomes enlarged, if each and all of the lobules cannot expand to fill it, some of them must be stretched unduly; and this produces emphysema.

For a few years Gairdner's view was widely accepted, it being held

that the affection was thus essentially "complementary" or "compensatory" either to collapse, or to retrogressive tubercular disease, or to some other contracting lesion of the lung. But in 1856 Sir William (then Dr) Jenner addressed to the Royal Medical and Chirurgical Society a powerful argument, in proof that the development of emphysema occurred during expiration. And it is now known that this very same doctrine had already been taught in Germany, as far back as 1845, by Mendelssohn, in a work entitled 'Der Mechanismus der Respiration und Circulation.' Gairdner urged that it is impossible for emphysema to be produced by the act of expiration, even with a closed glottis, because the force by which the air becomes compressed within the lung opposes exactly as much resistance without as it creates pressure within. Jenner now pointed out (as Mendelssohn had done before) that certain parts of the thoracic walls are yielding, and consequently incapable of maintaining this resistance. Both these observers indicated the apices of the lungs as being devoid of adequate protection against an expanding force from within; and we have seen that Louis had long before shown that the apices were especially apt to become emphysematous. Jenner remarked that during a fit of coughing the supra-clavicular regions may be seen to bulge, and that by placing one's hand upon them one can feel that they are distended by a considerable force. If the apices are the seat of emphysema, this bulging under violent expiration is extreme, and percussion over the bulging parts may elicit an almost tympanitic sound. He further showed that the upper costal cartilages are to some extent yielding, and that therefore the alveoli of the anterior margin of each lung becomes affected with emphysema as well as those of the apex. Other parts which he also named as often becoming emphysematous were: the margin of the base of the lung, the part of the lung near its root below the entrance of the bronchus, and the little ridge of lung which, on the right side, projects behind the trachea. The base of the left lung generally he showed to be less firmly supported than that of the right, the liver being more unyielding than the stomach; and he cites Louis as having found the left lower lobe emphysematous twice as often as the right one.

The correctness of Jenner's theory has since been supported by observations made in certain cases of congenital malformation of the chest walls. Thus in the case of Groux, who had a fissure of the sternum, the anterior part of the lung protruded through the fissure in the act of coughing. Ziemssen met with an example of absence of the *pectoralis minor*, and of the entire sterno-costal part of the *pectoralis major*, so that the intercostal muscles of the four upper spaces were covered only by fascia and by integument. During forced expiration these spaces bulged from 1 to $1\frac{1}{2}$ mm. above the level of the ribs; when the muscles of one space were faradised that space for the time remained flat, the others bulging as before. Further evidence is afforded by cases in which, after the cicatrisation of penetrating wounds of the chest, the affected parts have ultimately become the seat of hernia of the lung, as the result of weakening of the thoracic parietes. Many such instances may be found collected in a little work by Desfosses, 1875. It may be noted, too, that horses are liable to an affection of the lungs identical with emphysema, as the result of the straining efforts which they are called on to make, during which they keep the glottis closed. As Sir William Jenner says, in vol. iv of 'Reynolds' System,' "No one who watches a horse draw a heavy load up a short steep incline on a damp cold day can doubt this. While making the effort, the horse holds its breath,

having previously inflated the lungs. No sooner, however, does this animal cease its effort than the glottis is opened, and the air suddenly expressed from the lungs. The degree to which the air was compressed may be judged by the distance to which, and the sudden violence with which, the cloud of breath-vapours is seen to be driven forth." In his paper in the 'Med.-Chir. Transactions' Jenner had shown that the parts of the lungs that are emphysematous in a "broken-winded" horse are those which are so placed as to be least able to resist pressure. It is curious that a capital description of this affection of horses was given before the end of the seventeenth century by Sir John Floyer, in a treatise on asthma; the passage is cited in full in 'Watson's Lectures.'

The true theory of emphysema, then, is that it is the result of expiratory pressure with a closed, or partially closed, glottis. The expiratory muscles forcibly compress the air within the chest. If all parts of the thoracic parietes were equally unyielding no harm would result. But as certain parts can and do yield, some of the compressed air is driven into the corresponding alveoli of the lungs, and gradually breaks down their structure in the manner already described. No doubt the resistance of the chest walls in different regions fails progressively, more and more, as the affection advances. Thus the sternum and the upper rib cartilages become arched forwards, a change which probably is due to the frequently repeated application of an expansile force from within the thorax. It is only as the result of long-continued pressure that the diaphragm can become flattened, and that the lung can protrude inwards beneath the heart, as described above (p. 106).

Even when a part of the lung (generally the apex) is shrunken by retrogressive tubercular disease, most pathologists now agree with Jenner in thinking that the development of emphysema in the tissue around is probably still due to the pressure of air driven into the alveoli during violent expiration. Many writers, however, are of opinion that to such cases, at any rate, Gairdner s theory remains applicable, the emphysema being "complementary" in the strictest sense of the term.

It must be understood that, although a frequently repeated cough is one of the chief causes of emphysema, yet in man, as in horses, other actions, besides that of coughing, may induce an amount of expiratory pressure sufficient to give rise to the affection. This is the case, for example, with all violent efforts, in which the glottis is kept closed to fix the chest and thus provide a firm base for the operation of muscles passing to other parts of the body. Dragging or lifting heavy weights, straining at stool, even the act of parturition, may be mentioned as possible causes of emphysema. Thus Waldenburg is cited by Hertz as having seen the affection develop itself in a medical student who, having come from a country place where he had no occasion to ascend stairs, occupied in Berlin an apartment on the fourth floor, up to which he ran without stopping several times daily. Hertz himself met with a similar case in a young shopman, whose lungs became emphysematous in about a year, without any cough or bronchial catarrh, as the result of his having to carry heavy goods up a high staircase in haste a great many times every day. To persons who have suffered from bronchitis, and in whom the distending process has already begun, it is a very important piece of advice that they should avoid all occupations or amusements that involve the repetition of such efforts. Playing a wind instrument may sometimes be exceedingly injurious; for, although the glottis is not closed

the air within the chest is kept under great pressure, while it is being slowly allowed to escape.

Atrophic emphysema.—Of late, German observers have been disposed to attribute emphysema in part to changes in the pulmonary tissue itself, independent of mechanical conditions such as those just described. A *senile atrophy of the lungs*, bearing a close resemblance in its characters to emphysema, is generally admitted by pathologists; it was originally described by Dechambre in 1835, from observations made at the Salpêtrière. Sir William Jenner speaks of it as "*small-lunged emphysema*," in contrast with the ordinary form of the disease, which he calls "large-lunged emphysema." As he says, the small size of such lungs, their lightness, and the very small space into which they may be compressed are often most remarkable. When the thorax is opened after death they "fall in like an inflated bag of wet paper." The subjects of senile atrophy of the lungs are commonly thin, withered-looking, shrivelled old persons. Their chests are very small and narrow, the lower ribs being so obliquely placed that they almost reach the crest of the ilium, and so closely packed as nearly to come in contact with one another. The lungs are so reduced in size that the extent of præcordial dulness may be increased, notwithstanding that the heart itself partakes of the general wasting. Yet there is commonly little distress of breathing, because the volume of the blood is at a minimum and because the deficiency of muscular power forbids active exercise. It has always, however, seemed to the author that the supposed likeness of this senile atrophy of the lungs to emphysema is really due to the fact that a slight degree of emphysema resulting from the bronchial catarrh to which aged persons are so liable, is commonly mixed up with it. Hertz, in his chapter on atrophy of the lungs in 'Ziemssen's Handbuch,' speaks of bronchitis as a frequent "complication;" and he also mentions that the bronchioles are very thin and "generally uniformly dilated, seldom irregularly sacculated." But bronchiectasis can hardly be otherwise than mechanical in its origin; and it seems reasonable to take the same view of the pulmonary rarefaction, which Hertz describes as being most marked at the apices and along the anterior edges.

Predisposition.—That in younger persons some cause for emphysema must exist, beyond expiratory pressure upon the alveoli, is argued by Hertz from the fact that in certain families several members are found to suffer in succession, as the result of comparatively trifling affections of the air-passages. Schnitzler, for example, saw three brothers, whose parents were still alive and well, but who all became the subjects of emphysema at the age of thirty, without definite cause. Walshe cites Dr Jackson, of Boston, U.S., as having upheld the view that the disease is transmitted by inheritance; he found that "of twenty-eight emphysematous persons, eighteen had either a father, or a mother, or both, similarly affected; whereas of fifty non-emphysematous people, three only sprang from emphysematous parents." Dr Greenhow once argued for a relation between emphysema and the "gouty diathesis."

So far as concerns the mere occurrence of this affection in different members of the same family, it is important not to overlook the fact that they may all have been alike exposed to the causes of bronchial catarrh, and perhaps all alike unduly susceptible of taking cold. But Cohnheim and other recent German writers have looked for an explanation of emphysema, apart from mechanical causes, in a varying physical state of the pulmonary tissue, as regards its degree of elasticity. In this connection

certain observations of Perls may be cited, of which there is a record in vol. vi of the 'Deutsches Archiv.' By means of a pressure gauge he determined in a large number of cases the degree of force with which the lungs retracted when the pleural cavities were opened in the dead body; and he found that after death from enteric fever, or (in one case) from phosphorus-poisoning, their elasticity was reduced almost to nothing. Cohnheim regards it as an established fact that in a very large proportion of cases emphysema depends upon a congenital defect of development in the elastic tissue of the lungs. But the reference which he gives to a paper by Eppinger in the 'Prag. Vierteljahreschrift' for 1876 does not seem to bear out this assertion; for although Eppinger found that even in slightly emphysematous lungs there was a great reduction of the network of elastic fibres in the alveoli, the smallest fibres having completely disappeared, he yet appears to have regarded this as a change occurring in the course of the development of the disease, and not as an antecedent malformation.

In this connection a case recorded by Hertz is of great interest. It is that of a regimental cornet-player, aged thirty, who had always been able to use his instrument without any difficulty, even on the march. He was attacked with double pneumonia, which, however, subsided in a week; he felt quite well, had no cough, and began again to practise with his cornet. But in the course of the next seven months he discovered that he was no longer able to take sufficient air into his lungs to maintain a long note for the proper period, that he was short-breathed on exertion, and could no longer play while marching. Hertz found on examination that the lungs were markedly emphysematous, which had not formerly been the case; and his supposition is that the pneumonia had damaged their texture, so that they were not able to resist expiratory pressure as before.

Clinical symptoms.—The recognition of emphysema is based mainly upon physical signs. The chief subjective symptom of the disease is *dyspnœa*. The first thing noticed by the patient is that he is short of breath when he exerts himself, as in running upstairs; but after a time difficulty of respiration becomes a permanent condition from which he is never entirely free. As Dr Walshe says, he feels as if his chest were never emptied of air as it naturally should be; and he is conscious of an annoying sense of inflation or distension. It is true that many emphysematous persons affirm that their dyspnœa is only occasional; but Dr Walshe says that in all such cases which he has seen, the patient has been deceived, a moderate amount of dyspnœa having become to him a second nature—a thing unperceived and giving rise to no discomfort.

It does not necessarily happen that the affection advances; it may continue stationary, and life may be maintained until extreme old age, provided that the risk of intercurrent attacks of bronchitis can be obviated. Hertz supposes that the existence of emphysema necessarily involves an increased liability to bronchial catarrh, by leading to congestion of the mucous membrane of the air-tubes; but it would be difficult to prove this, seeing how very common bronchitis is as an antecedent condition.

On the other hand, in the majority of cases, emphysema becomes more and more marked every year. After a time the noisy hurried breathing may become so short that the patient cannot utter a sentence without stopping in the middle of it. At night he has to be propped up by pillows. His distress becomes aggravated from time to time, sometimes by an exacerbation of bronchial catarrh, sometimes by the supervention of asthma, some-

times by mere pushing upwards of the diaphragm, as the result of distension of the abdomen with flatus, or with undigested food. The face, the hands, and at last the whole body become livid to the most extreme degree, the condition of the patient being exactly that which has been described above (p. 100) as resulting from capillary bronchitis. In fact, chronic bronchitis never goes on for any length of time without being complicated with more or less of emphysema, so that it is impossible to separate the effects of these two lesions. Cough, on the other hand, may be entirely absent in cases of emphysema unless there is bronchial catarrh. But many persons, whose main disease is emphysema, constantly have cough, and expectorate a frothy liquid, or pearly-grey masses of mucus.

Hæmoptysis is not generally said to be among the symptoms of this affection. Dr Duckworth, however, in the 11th volume of the 'St Bartholomew's Hospital Reports,' declares that it is of not infrequent occurrence. He remarks that the wasting of the pulmonary capillaries and arterioles affords a sufficient explanation. In 1869, a woman, aged forty-nine, was brought dead into Guy's Hospital of an attack of severe hæmoptysis, and at the autopsy the only disease that could be discovered was an extreme degree of emphysema of the upper lobes of the lungs, with some excess of fibrous tissue forming the interlobular septa. The air-tubes were full of clots. She was said to have suffered for three months from wheezing and short breath; on the morning of her death she woke up at 4.25 a.m. with "coughing and vomiting of blood through the nose and the mouth, and was suffocated in ten minutes."

Ultimately emphysema gives rise to great wasting, and to extreme enfeeblement of the muscular strength. One noteworthy circumstance, pointed out by Walshe, is that the over-distension of the chest renders the body unnaturally buoyant in water, so that the patient is astonished to find himself able to swim more easily than before, at the very time when he is growing more and more incapable of other kinds of exertion.

Physical signs.—Of these, the most important are derived from *percussion.* Even very slight degrees of the affection may be detected by carefully mapping out the area of the heart and of the liver respectively. Instead of beginning at the upper border of the fourth left costal cartilage, the cardiac dulness is discoverable only at a lower level, over the fifth or even the sixth cartilage. Instead of beginning at the upper border of the sixth right rib the hepatic dulness is discoverable only at the level of the seventh or of the eighth. If in a case of bronchitis one finds that percussion over these two organs yields normal results, one is generally safe in declaring that no appreciable amount of emphysema has yet developed itself.

As the disease advances, the heart becomes so completely covered by the lungs that no cardiac dulness at all can be detected, the pulmonary resonance above meeting the tympanitic sound caused by the stomach below. At the same time the apex-beat ceases to be felt in the normal position, in consequence of the downward displacement of the diaphragm carrying the heart with it; and the axis of the organ becomes altered, so that its pulsations can often be felt in the epigastrium. On the right side pulmonary resonance now extends down to the margins of the ribs. The edge of the liver may sometimes be felt in the hypochondrium. But very often this is not the case and there may be a marked reduction in the area of the hepatic dulness, so that one may be tempted to suspect that the organ is atrophied or cirrhosed when this is not really the case. The chief reason why in such circumstances

the liver fails to be in contact with the parietes of the chest and abdomen to the normal extent is the great increase in the antero-posterior diameter of the trunk. Another noticeable feature of well-marked cases of emphysema is the clear character of the percussion-sound over the sternum as high as its upper border; and over the bases of the lungs behind a clear percussion-sound is elicited to a much lower level than normal.

Again, the quality of the percussion-sound, over parts of the chest where it ought naturally to be resonant, is altered when the lungs are decidedly emphysematous. The alteration is in the direction of over-resonance, and according to Dr Walshe and Dr Gee there is often a fall in its pitch, so that it may accurately be described as tympanitic. The change in the character of the percussion-sound is often very conspicuous over the back of the lungs, a sonorous drum-like note being readily produced where normally there would be great difficulty in eliciting a clear resonant sound.

Next to percussion, *inspection* affords the most valuable indications of emphysema. As already explained, the sternum and the upper ribs arch forwards as the result of their yielding to the frequently repeated expiratory pressure which is the cause of the affection. The sternum not infrequently becomes convex in a vertical plane, with an angle, known as the *angulus Ludovici*, at the junction of the manubrium with the body. According to Freund, the rib cartilages grow in length and in breadth—a change which he actually regarded as the starting-point of the pulmonary disease. The clavicles also are more bent than under normal circumstances. The curve of the dorsal vertebræ becomes greatly increased. Sometimes the back is so rounded that the scapulæ seem to be almost horizontal in position. The effect of all these alterations in the parietes of the chest is to give it a cylindrical form. It is often aptly said to be "barrel-shaped." By the cyrtometer its horizontal circumference is shown to be almost perfectly circular, as is well illustrated in a diagram given by Dr Gee. Sometimes the rounding of the ribs and of their cartilages continues to the very bottom of the thorax, and the hypochondriac regions are permanently expanded to the fullest possible extent. One result of this is, as Hertz has pointed out, a transverse groove, which crosses the abdomen horizontally from one twelfth rib to the other; it is due to the stretching of the upper part of the transversalis abdominis muscle, which is fixed to the rib cartilages, as compared with the relaxed condition of the lower part, which has no such attachment. This groove may form a conspicuous, and at first sight puzzling feature of a case, when there is at the same time a considerable accumulation of fluid in the peritoneal cavity. But in other instances the lower ribs and their cartilages are flattened, or even hollowed inwards; this occurs especially when the pulmonary affection began in bronchitis or whooping-cough at an early period of life, so that the bases of the lungs became collapsed. The "subcostal" angle at the ensiform cartilage is far more open than usual.

The upper intercostal spaces in emphysema are either unaffected, or narrowed, as in full inspiration, while the lower ribs are widely separated. Stokes declared that he had never seen the spaces otherwise than depressed; but Walshe and others say that it is not uncommon for them to be prominent or bulging when the parts of the lungs beneath, being highly emphysematous, have lost their elasticity.

The high shoulders, increased depth of chest, short and full neck, raised ribs, closer together above, and wide apart below, with the obtuse substernal

angle, are the physiological characters of the chest during deep inspiration, and can be imitated by filling the lungs and holding the breath. The same fulness of the chest may be the result of distension of the pleura by fluid on one side or the other, but when bilateral it is characteristic of emphysema alone. It is the exact opposite of the expiratory form of chest seen in advanced phthisis.

Further indications as to the presence of emphysema, and as to the extent to which it is advanced, are yielded by observation of the act of breathing. During inspiration the chest in well-marked cases is seen to be almost motionless. It is, indeed, impossible for the upper ribs to rise and expand, as they normally should do, because they have permanently assumed a position in advance of what could have been reached in health. There is, however, a jerking movement upwards of the thorax as a whole, produced mainly by contractions of the sterno-mastoid and scalene muscles, which start into unnatural prominence, and appear to be considerably hypertrophied. If the lower ribs are thrown outwards as much as the upper ones, the chief agent in inspiration must be the diaphragm. But in many cases the state of affairs is reversed. The lower ribs still retain a certain degree of mobility, but the diaphragm is pushed downwards, so that it can do scarcely anything towards enlarging the thoracic cavity. It may then be observed that the epigastrium becomes hollowed during the act of inspiration.

Violent cough causes bulging of the suprascapular and suprasternal spaces; and in cases of emphysema Sir William Jenner warns against confounding this condition at the root of the neck with prominence of the same part due to distension of the veins. A still more important source of error is the sudden protrusion of an aneurysmal sac during the act of coughing. Another point to which Jenner has drawn attention, is that if one is feeling the pulse of an emphysematous patient while he coughs violently one perceives the artery to become suddenly full and tense, after which it ceases for the moment to beat.

Auscultation gives comparatively little information in cases of emphysema, apart from signs of the bronchitis that is so commonly associated with it. The most marked sign of the emphysema itself that is given by the stethoscope is enfeeblement or nearly complete absence of the vesicular murmur. Moreover, the bronchial breathing which may normally be heard over the roots of the lungs behind, and beneath the sterno-clavicular joints, is often wanting in emphysematous patients. But even in those who are healthy this is not infrequently the case. The expiration is exceedingly prolonged owing to the loss of elasticity in the lungs. Walshe says that, instead of being only one third the length of the inspiration, it may be four times as long; in other words, its relative duration may be increased twelve-fold. The growling, squeaking, or wheezing expiratory sound of emphysema is very commonly spoken of; but it seems doubtful whether this sign does not belong rather to a concomitant bronchitis than to the pulmonary lesion itself. In one case, at Guy's Hospital, as the tubes became free, this sound entirely disappeared, although the extremely feeble state of the inspiratory murmur and the over-resonance of the percussion-sound clearly showed that emphysema still persisted.

In cases of emphysema the vocal resonance is much diminished, and tactile vocal fremitus is often entirely lost.

When air has escaped into the subpleural connective tissue, constituting what is known as "interlobular" or "interstitial emphysema," there is

sometimes heard a friction-sound very like that which occurs in pleurisy. This sign was originally noticed by Laennec. Most writers since then have been disposed to doubt the correctness of the observation; but it has been recently confirmed by Dr. Gairdner; and Dr Hudson, in his edition of Stokes's works, says that he also met with a case in point.

In the latter stages of pulmonary emphysema a tricuspid regurgitant murmur may often be detected; and even at an earlier period increased pressure on the venous side of the circulation may be indicated by an accentuated pulmonary second sound. Epigastric pulsation and pulsation of the veins of the neck with each beat of the heart may also be observed. These symptoms are all the result of obstruction in the pulmonary capillaries having led to dilatation of the right side of the heart and incompetence of the tricuspid valve.

The *treatment* of emphysema will be considered with that of bronchitis at the end of the present chapter.

Hypertrophy of the lungs.—The barrel-chest above described, *i. e.* the thorax when filled by the ordinary or "large-lunged" emphysema, is closely simulated by a condition which is physiological rather than diseased, and may be described as uniform overgrowth of the lungs. It occurs to some extent in all healthy persons who are engaged in hard manual labour or in athletic sports. The chest of the youth who improves his wind by rowing, or of the workman who is accustomed to carry heavy weights, becomes expanded and the lungs increase in volume and capacity. When the efforts made with the glottis closed are too great, rupture of the air-vesicles is apt to occur, and thus true emphysema may be mingled with physiological hypertrophy; but this is far from a necessary complication.

Ascending ladders and climbing hills, especially when a weight is carried on the shoulders, has a similar effect. Greater efforts of inspiration are made, the lungs are expanded more perfectly, and the breathing capacity of the chest is increased.

But another condition produces very similar but exaggerated effects, namely, habitually breathing rarefied air. In persons who live in mountainous regions both causes of hypertrophy of the lungs combine; they are frequently ascending heights, often with burdens on their shoulders, and they are constantly breathing air at a low pressure, and hence each cubic inch of lung gives them less oxygen to aerate their blood. The result is that the thorax becomes enlarged in all its dimensions. Instead of a yard's girth for a man of moderate stature, and a metre's for a tall man, the chest of men of five feet six inches or even less measure more than forty inches in circumference, or often much more.

This condition may be observed in Swiss guides; but it has hitherto been found most constantly and highly developed among the natives of the lofty table lands of the Andes, in Bolivia.

Dilatation of the Bronchi. Bronchiectasis.—This affection was first described by Laennec. It seldom constitutes a primary disease. In some instances it is associated with fibroid induration from chronic interstitial inflammation of the tissue between the enlarged tubes; such cases will best be discussed in the account of chronic pneumonia or cirrhosis of the lung. The cases now to be described are those in which the pulmonary parenchyma is either healthy, or else more or less emphysematous or col-

lapsed, showing, in fact, no changes except the secondary results of bronchial inflammation.

Anatomy.—All writers divide bronchiectases into cylindrical and sacculated, and the definition is of pathological and clinical importance.

In the *cylindrical* form the tubes sometimes run through the substance of the lung, with but little diminution of calibre, until they end abruptly beneath the pleural surface; their appearance is often compared to that of the fingers of a glove, but it very rarely happens that the dilatation is so uniform throughout their whole length. In one instance which occurred at Guy's Hospital in 1873 the bronchial tubes were so enlarged as to be conspicuous at the root of the lung, "pushing the lobes apart from one another," as it is expressed in the report of the autopsy. Much more often the medium-sized and smaller tubes are alone affected. The existence of bronchial dilatation may then be obvious on the cut surface of the lung, far too many large orifices being visible, from which pus wells up in great quantities. Or, in order to detect the enlargement of the tubes, especially if they are empty, it may be necessary to open them up with scissors. One may then find perhaps that only a few of them are dilated in any marked degree. Judging from the author's own observations, such slight forms of bronchiectasis are much more often met with in the extreme bases than in any other parts of the lungs. Not infrequently he has seen enlargement of the principal tube passing into the ear-shaped process of the left lung, when no such change could be made out elsewhere. This was when the ear-shaped process itself was emphysematous; and, indeed, these less-marked examples of bronchiectasis are very often found in association with emphysema. On the other hand, when the bronchial affection reaches an extreme degree, there is seldom a corresponding amount of rarefaction of the pulmonary tissue. If the tubes in any one part of the lung are universally dilated, running to the surface side by side, and perhaps as large as quill-pens or even larger, the parenchyma between them is necessarily reduced to a small space. In these cases it is sometimes difficult, at the first glance, to say whether the affection is or is not secondary to a fibroid change in the lung itself. Such a condition is sometimes found in the middle of the lung or even of the upper lobe, the lower lobe being free. But in the most marked and typical cases of cylindrical bronchiectasis, such as occur in children after whooping-cough or measles, the tissue between the dilated tubes is often quite free from induration or other morbid changes.

It must be understood that cylindrical bronchiectases are by no means always absolutely uniform in diameter at different points. Sometimes they gradually widen as they approach the surface. Sometimes they have fibrous bands or trabeculæ projecting from their walls here and there and rendering their calibre very irregular.

Sacculated dilatation of the bronchial tubes, again, varies widely in character in different cases. The most typical form is one which presents appearances that have scarcely yet been exactly described. When a section is made of the lung, the cut surface appears to be covered with an immense number of shallow smooth-walled depressions, like so many minute saucers. Each of these has in its floor a very small rounded orifice, and it is obvious that they are all sections of small bronchiectases, which probably were spherical before they were cut across, but of which the halves have become flattened by their own elasticity and by that of the adjacent pulmonary parenchyma. At least four instances of this kind have occurred in our

dead-house. In all likelihood, if the lung in such a case could be inflated and dried, and the parenchyma then cleared away so as to expose the tubes in their continuity, each would be found changed into a regular series of globular dilatations, so as fairly to deserve the epithet moniliform. In other instances sacculated bronchiectases are more unequal in size and irregular in form; but it is probable that most of the cavities of this sort are really not dilated tubes but smooth-walled vomicæ which have been formed by ulceration. It is to these latter sacs that in all likelihood the statement of Biermer applies (in vol. v of 'Virchow's Handbuch'), that the majority of the sacculi of bronchiectasis have openings only towards the trachea, and are entirely closed on the distal side. Still less are true bronchial sacs ever converted into shut cysts.

Dilated bronchial tubes often have exceedingly delicate walls, being apparently as thin as, even if not thinner than, they were before they began to increase in size. But sometimes the tissues are thickened, the lining membrane being velvety and showing the changes above described as occurring in bronchitis.

Pathology.—As to the way in which bronchiectasis arises, there can be little doubt that it is the mechanical result of expiratory pressure, being in fact produced by exactly the same cause as pulmonary emphysema. To explain satisfactorily why in one case the alveoli should yield, and in another case the tubes, would perhaps be impossible. But we may fairly suppose that it depends upon the degree to which the walls of the tubes have been softened by inflammation, and also, it may be, upon the amount of elastic resistance originally possessed by the pulmonary tissue in each individual. (See Dr Grainger Stewart's paper, 'Edin. Med. Journ.,' July, 1867.)

The most frequent cause of bronchiectasis is chronic interstitial pneumonia, but with this form we are not at present concerned. The kind which is independent of previous organic changes in the lung is not frequent either in children or adults. It sometimes complicates emphysema but more frequently takes its place.

In children it is most often a sequel of whooping-cough, and sometimes comes on very early, and so severely that the patient grows up in a state of permanent cyanosis, with cold extremities and short breath.

In adults bronchiectasis occurs in middle rather than advanced life, and almost always is preceded by bronchitis, seldom by tubercular disease, and never by acute pneumonia.

Diagnosis.—It is only when bronchiectasis has reached an extreme point that it can be said to be characterised by definite *physical signs.* As a rule, the chief indication of this affection is the presence of râles which appear to be too large and coarse to be formed in the undilated tubes of the part of the lung in which they are heard, as, for instance, at the extreme base, or along the anterior edge. If, however, a number of tubes cylindrically dilated are arranged side by side, while the lung tissue between contains but little air, there may be more or less marked bronchial breathing, bronchophony, and even dulness on percussion. But such a case could not be clinically distinguished from primary chronic pneumonia, with bronchiectasis as a minor feature. Again, the question of the diagnosis of a saccular dilatation of a bronchial tube from a phthisical vomica, to which stethoscopists formerly devoted themselves with much ardour, applies to the dilatation which attends cirrhosis of the lung, not to cases arising out

of bronchitis alone. One circumstance which is strongly indicative of bronchiectasis, as may be understood from what is about to be stated of the symptoms, is for the physical signs over a certain part of the lung to undergo more or less regular variations from time to time, being now well marked, and now again indistinct or absent.

As for its *symptoms*, dilatation of the tubes is of course in part concerned in causing cough, dyspnœa, and lividity in patients affected with it. But one can never clinically separate its share in producing these effects from that due to the bronchitis which is always associated with it, and perhaps also to concomitant emphysema. The only thing that enables one to diagnose bronchiectasis with confidence is a peculiar way of expectorating which may in some cases be observed. For some hours, perhaps, there is no cough at all. During this time liquid is accumulating in the dilated parts of the air-passages, the sensitiveness of which appears to be blunted, so that they do not resent its presence. Then it perhaps happens that some runs over into a tube which is still healthy. The result is a more or less violent fit of coughing, by which all the liquid that has collected is suddenly expelled, pouring out of the patient's mouth, and even through his nose, in enormous quantity, so as to half fill his spittoon. Sometimes this process is set in action by an attempt at physical examination of the chest. Sometimes it occurs, especially in the morning, when the patient rises from the recumbent posture. When this accumulation has been got rid of, he usually feels much more comfortable than before, the breathing is easier, and the chest less oppressed. Such intermittent expectoration (or in children vomiting) of pus is a sure sign of dilated bronchi.

Fœtid or putrid bronchitis.—It is especially in cases in which the bronchial tubes are dilated that bronchitis becomes accompanied with the expectoration of foul-smelling sputa, and sometimes with horrible fœtor of breath. Traube has, indeed, recorded one or two cases in which putrid bronchitis occurred without there having been any bronchiectasis. But, as a rule, it is only when liquids have become for a considerable time stagnant in some part of the air-passages, or in a space communicating with them, that putrefactive chemical changes occur.

The characteristic symptom of putrid bronchitis, as was first pointed out by Traube, is the presence in the matters expectorated of certain soft, friable, smooth masses, of a dirty greyish-yellow colour and very fœtid odour, varying in size from a millet-seed to a bean. Such bodies had been originally noticed by Dittrich in 1850 as plugging the affected tubes in fatal cases; and in Germany they are commonly called "Dittrich's" or "Traube's plugs (*Pfröpfe*). Microscopically they are made up mainly of a finely granular detritus mixed with fat-globules. They also often contain certain very long, narrow, acicular crystals, of which Virchow gave a description long ago in the first volume of his 'Archiv,' as consisting of a fatty acid. These crystals are colourless, often sharply bent or twisted, sometimes collected together in sheaves or in thick bundles. Some of them may appear to be varicose, a condition which Traube has shown to be the result of pressure by the cover-glass. According to Guttmann they contain a combination of palmitic and stearic acids. In 1867 Leyden and Jaffé further pointed out, in vol. ii of the 'Deutsches Archiv,' that under high powers the granular detritus is composed of vegetable organisms, some round, others rod shaped, others forming beaded chains or long filaments. Their presence appears to

be the reason why iodine often gives a purple or a violet, or even a blue tint to the whole mass, as Virchow and Gamgee independently observed. Chemical analysis of the sputum of putrid bronchitis, in different cases, has also shown that it may contain volatile fatty acids (valerianic, butyric, and acetic acids), methylamine, leucin, and tyrosin, ammonia, and sulphuretted hydrogen. The authority for including methylamine and acetic acid in the list is Dr Gregory, of Edinburgh, as reported by Dr Laycock in the 'Med. Times and Gazette' for May 1857.* It is also worthy of mention that Leyden and Jaffé succeeded in inducing in ordinary muco-purulent sputum, outside the human body, a putrefactive process closely analogous in its results to that which must be supposed to give its peculiar character to the expectoration of patients with putrid bronchitis. They suppose that the source of the vegetable organisms described by them is the common leptothrix of the buccal cavity.

In putrid bronchitis the sputum as a whole is generally very abundant. It separates in the spittoon into three layers: of these the uppermost is opaque, greenish yellow, and frothy; the middle is a transparent albuminous liquid like serum; the lowest is opaque, and of a dirty-yellow appearance, consisting mainly of swollen pus-cells and of the *débris* resulting from their destruction.

The odour of the sputum or of the patient's breath in cases of putrid bronchitis is commonly identical with that which belongs to gangrene of the lung, as one observes it in the *post-mortem* room under the most varied conditions. Guttmann compares it with the smell that pervades a soap manufactory. But in other cases it is of an altogether different character. There is, of course, great difficulty in defining the distinction verbally, but Dr Laycock's statement is that in one of his patients the odour was like "that of the mayflower or of apple-blossom, with a kind of *arrière goût* of fæces." The author has often observed this kind of smell, especially in cases in which dilated bronchial tubes were emptied with a gush of enormous quantities of fluid at once after the manner described above. Probably in such cases there is no active process going on in the walls of the affected tubes themselves. On the other hand, in many of those cases in which the odour is like that of gangrene of the lung the development of fœtor in the sputa indicates the abrupt commencement of a destructive change, both in the air-passages and in the pulmonary parenchyma, which rapidly brings about a fatal issue.

The credit of having first pointed out the clinical features of the disease in cases of this kind belongs to Dittrich (1850). His description is that it "commonly arises in persons of the middle period of life, who have suffered for years from bronchial catarrh, with abundant muco-purulent expectoration, and who may either have already begun to waste, or may still remain well nourished. Suddenly, and without apparent cause, the sputum becomes offensive, of a dirty-grey colour; the breath also stinks, poisoning the air around. Thereupon follow severe dyspnœa, fever of typhoid character, rapid collapse, an earthy dirty-yellow complexion, and ultimately cessation of expectoration, coma, and death."

At the autopsy the walls of some of the bronchial tubes are found intensely inflamed and sloughing. There are more or less extensive areas of pneumonic consolidation passing here and there into gangrene. Other

* See, however, a valuable paper on the chemical characters of fœtid expectoration by Dr Gamgee ('Edin. Med. Journ.,' March, 1865), and one by Bamberger in the 'Würzburger med. Zeitschrift,' 1864.

parts of the lung tissue are œdematous, exuding a fœtid liquid. The bronchial glands were greatly swollen, soft, and of a dirty-grey colour. Several cases exactly in point were afterwards recorded by Traube. It must be noted that the occurrence of hepatisation and gangrene of the substance of the lungs is by no means limited to those regions which were before the seats of bronchiectasis. Pneumonic patches may be scattered throughout every part of the organ on each side; and it seems obvious that many of them owe their origin to the inhalation into healthy tubes of particles of putrid *débris* derived from others which are already diseased.

But the issue of putrid bronchitis is not always thus serious. Slight cases sometimes end in recovery. We must then suppose either that there has been no ulceration of the walls of the tubes, or that the necrotic process has been limited, and that healing has taken place after detachment of sloughs of no great size. Other cases, again, run on for months with but little change in the symptoms, and without marked impairment of the general health.

To complete this account of the effects of dilatation of the bronchial tubes it must be mentioned that in vol. xv of the 'Deutsches Archiv' Gerhardt has recorded two cases in which a painful swelling of some of the joints occurred as a sequela. He was inclined to regard this rheumatoid affection as analogous to that which is met with after gonorrhœa or dysentery.

The occasional supervention of abscess of the brain as a complication of suppurative processes in the air-passages or in the lungs has been referred to elsewhere (*supra*, vol. i, p. 638).

Ætiology of bronchitis generally.—The chief cause of ordinary acute and also of chronic bronchitis is *exposure to cold*. In all probability cold air entering the air-passages through the nose, or the mouth, sometimes acts upon them as a direct irritant. It is true that the nasal mucous membrane warms the air that traverses it, but, on the other hand, the experience of persons who venture into the open air at a low temperature, with a bronchial surface already in a morbidly sensitive condition, is conclusive as to the fact that cough is very apt to be excited, and that this is not the result of mere exposure of the skin to the cold. Still the analogy of so many other inflammatory affections of internal structures, which contain no tubes communicating with the external atmosphere, is entirely in favour of the view that bronchitis may also be set up by the action of cold upon the surface of the body; and this is borne out by the circumstance that in many cases there is no reason to suppose that cold air has been inhaled. A patient may, for example, "take a chill" by getting wet through, by sitting in draught, by lying on damp grass, by merely remaining motionless out of doors when perspiring profusely after exertion. As a rule, it is especially when the body, from having been heated, is cooling that danger of catching cold exists. The reason appears to be that, whenever the body has more heat to dispose of than is required to maintain its due temperature, the cutaneous capillaries become dilated to allow as much loss of heat as possible. This is equally true whether the heat is supplied to the organism from without or generated in its interior, as the result of muscular exertion. Accordingly, after a Turkish bath, one plunges for a few seconds into cold water, which causes contraction of the blood-vessels, before one ventures to sit in a room at an ordinary tem-

perature. Rosenthal, and afterwards Riegel, have shown by direct experiment that if animals after exposure to great heat are removed and placed in air which is not warmed, they go on cooling until their temperature falls below the normal point. They therefore suggest that, when a person catches cold, what occurs is that blood from the surface of his body, chilled by loss of its heat, is carried to deeper structures, until they also become less warm than natural. If now there be anywhere a weak spot, it suffers and becomes inflamed.

Some persons are far more sensitive to the action of cold than others. The risk of catching cold may, however, be often obviated to a great extent by "hardening" the skin, that is, by exposing it regularly to sudden changes of temperature, so as to accustom its vessels to contract promptly and vigorously. The best way of doing this is doubtless to sponge the surface with cold water, or to use a cold douche or a shower-bath every morning after a tepid bath. The cold bath by itself is probably less effectual for the particular end in view, although it is all that is required for robust persons with an active circulation, in whom it is followed by a good reaction. Even in young children a warm bath, especially in the morning, may always with advantage be followed by rapid sponging with chilled water.

Many *secondary* or (as they would formerly have been called) *predisposing* causes of bronchitis are really conditions which favour the injurious action of cold. Thus the disease is very apt to attack young children on account of their feeble powers of resistance. A curious point that came out in some investigations made by Geigel as to the infant death-rate in Würzburg was that bronchitis was relatively less fatal to illegitimate than to legitimate children, the reason being in all probability that the latter are coddled up and kept warm, so as to be rendered more sensitive to cold than they otherwise would be.

The prevalence of the disease in Europe is, as might be expected, least in the hot season of the year, from June to September, and its proportionate frequency in different months is not the same as that of acute pneumonia. This is true also of the geographical distribution of the two affections. Bronchitis increases in frequency from the equator towards the poles, but the increase is not uniform in all longitudes, for it varies with the climatic conditions of each particular country. What favours it most is not a low mean temperature, but the occurrence of sudden and violent changes of temperature, and, above all, the presence of much moisture in the air. In some parts of the tropics bronchitis is by no means uncommon at the end of the hot season. There are certain countries in which it is of very rare occurrence, particularly Egypt, the western prairies of North America, the plains of India, a part of the West Indies, and California.

Next to cold, the entrance of irritant substances into the air-passages during breathing is the most important cause of bronchitis. In discussing the ætiology of phthisis we shall have to consider the influence of various occupations, in which the inhalation of dust is almost inevitable, upon the production of that disease. All those occupations also cause a great liability to bronchitis, which may either in the course of time be followed by the development of phthisis, or may run a chronic course without complication until it ends fatally by the supervention of an acute attack, or by dilatation of the heart and dropsy. Pulverulent substances which happen to be coloured, such as carbon or oxide of iron, often tinge the sputum deeply

when they have been inhaled. But, on the other hand, it may happen that a miner, whose lungs are anthracotic, spits up a yellow muco-purulent fluid, containing no carbon whatever. This accords with the fact that the bronchial mucous membrane never itself becomes the seat of anthracosis ; even the peribronchial tracts of fibrous tissue derive the black deposit which is found in them from the surrounding pulmonary alveoli, and not from the tubes themselves.

The inhalation of gases, especially nitrous or sulphurous, is exceedingly irritating to the air-passages, and not infrequently sets up acute bronchitis in workmen whose occupations expose them to it. But, according to Hirt, the chronic form of the disease is comparatively seldom traceable to this cause. After one or two acute attacks a tolerance seems to be established, and no further ill-effects are observed. On the other hand, he speaks of the emanations from certain oil works, from tar factories, and from the pans in which brine is evaporated to make salt, as having a beneficial influence on the bronchial mucous membrane.

The *treatment* required for bronchitis varies widely in different forms and in different stages of the disease. It is not easy, in text-books, to lay down rules for it.

In the milder forms of acute *bronchial catarrh*—such as are called by some writers tracheo-bronchitis—little is necessary beyond placing the patient in an equable temperature, which should be at about 63°. Small doses of ipecacuanha, with neutral salts like nitre, are probably serviceable by favouring exudation from the inflamed mucous surface. The application of mustard plasters or of hot flannels sprinkled with turpentine, to the throat and to the upper part of the chest often gives great relief to the sense of soreness along the trachea and behind the sternum, but in slight cases a linseed poultice and inhaling steam are efficient and agreeable remedies.

Very different measures are necessary in some cases of *capillary bronchitis*. Sometimes, if suffocation appears to be rapidly impending and the right side of the heart to be overloaded, it is advisable to bleed from the arm freely. Antimony is often the best medicine, and for a day or two it may be given in considerable doses, so as to have a decidedly nauseant effect. Another drug which may be very successful is lobelia, the ethereal tincture of which is prescribed in half drachm or even in drachm doses at frequent intervals. There are some patients, on the other hand, for whom all depressing remedies are obviously unsuitable. In these serious and often more dangerous cases the administration of turpentine sometimes affords the best chance of arresting the fatal issue which is obviously near at hand. In one of the very worst cases which the writer ever had to treat, life appeared to be saved by alternate doses of turpentine and of champagne.

It is very important to maintain a moist state of the air round the patient. A kettle on the fire, with a long tube throwing steam out near the patient's bed, fulfils this indication better than anything else. But when it is used, one must never forget that the ceiling and the curtains necessarily become saturated with damp, and that if the temperature of the room should be allowed to fall a few degrees during the night, or in the early morning, a chill will result, which may probably be fatal to the patient. In some cases the inhalation of steam gives much relief, or a medicated spray may be employed, containing conium juice, or morphia, or salines such as chlorate of potass. Large poultices are commonly placed round the whole chest

from front to back; and mustard or turpentine is applied until the surface is thoroughly reddened.

After a few days it is generally necessary to substitute for nauseant drugs, like ipecacuanha or antimony, such remedies as carbonate of ammonia, squill, and senega.

In *chronic bronchitis* a great variety of medicines are useful; the difficulty is to formulate rules for selecting one rather than another of them. If the cough is dry and hard, ipecacuanha is especially serviceable. If it is distressing by its frequency and apparent aimlessness, bromide of ammonium or of potassium often gives relief. In such circumstances morphia or opium may be prescribed with great advantage. After a few hours' sleep the patient may wake greatly refreshed, and in all respects better. It is, however, always necessary to consider, before prescribing opiates in a case of bronchitis, whether one is likely to do harm by checking cough, and so preventing the tubes being emptied of their contents. If there is lividity, stupor, or even drowsiness, such medicines must be carefully avoided. In many cases, especially if the expectoration is viscid and abundant, sal ammoniac is very useful; it may be given in doses of gr. xv to gr. xx, with a little syrup of lemon, or extract of liquorice, to conceal its disagreeable taste. Iodide of potassium is another salt which often does good service in chronic bronchitis.

In cases in which there is excessive exudation and secretion from the surface of the mucous membrane, balsamic remedies are applicable; tolu, benzoin, Peruvian balsam, benzoic acid. Or, again, one may prescribe copaiba, turpentine, ammoniacum, or one of the fœtid gum resins, such as assafœtida.

Of late years several physicians, in Germany especially, have made large use of compressed air, and also of rarefied air, in the treatment of various bronchial and pulmonary affections. In the earlier attempts recourse was had to pneumatic chambers, made somewhat after the fashion of the diving bell, in which the patients sat for an hour at a time, under a pressure of $1\frac{2}{5}$ to $1\frac{3}{7}$ atmospheres. It is obviously in very exceptional circumstances only that such elaborate constructions can be available in practice; and therefore attention has been more recently devoted chiefly to the invention of portable forms of apparatus, by which the patient is made to inhale air of varying degrees of pressure without being himself immersed in it. Most of these instruments are upon the principle of the ordinary gasometer used to receive coal gas at gasworks; an air-containing cylinder, open below, is suspended in another cylinder, open above, so that the one can move freely up and down within the other. By pouring more or less of water into the outer cylinder, and then either pressing down the inner cylinder with weights or lifting it up to varying heights, the air inside it may be compressed, or it may be rarefied to any desired extent. The object is to make the patient *inspire* compressed air, or *expire* into rarefied air. A tube from the inner cylinder is connected with a mask, which can be fitted air-tight over the nose and the mouth. There is a stopcock, which is turned by the patient each time he breathes, so that the mask communicates with the cylinder either during inspiration or during expiration (as may be intended), whereas on reversing the movement it communicates with the external air. Waldenburg, who invented this machine, usually directs that compressed air should be inspired for five, ten, or fifteen minutes, and then, after a pause, that expiration into rarefied air should be practised for a similar period. The range of pressure

variations employed is but small. In most cases two or three sittings a day are sufficient. It is obvious that, so far as emphysema is concerned, the greatest degree of benefit is to be anticipated from expiration into rarefied air. The inspiration of compressed air, when the whole body is not immersed in a pneumatic chamber, might be expected to tend rather towards increasing the distension of the pulmonary alveoli. But, on the other hand, it is said that expiration into rarefied air may, in its turn, do harm by augmenting the flow of blood to the bronchial mucous membrane, whereas inspiration of compressed air has the effect of increasing the blood-pressure in the systemic vessels, and of unloading the pulmonary vessels and the right side of the heart. It is therefore best to alternate the two methods, as Waldenburg and others advise. There is strong testimony of the beneficial action of this mode of treatment, in augmenting (at least for a time) the activity of emphysematous lungs, and in relieving the symptoms of bronchial catarrh. (See Dr Theodore Williams' three lectures in the 'British Medical Journal' (April 18th, 1885), and Dr Gamgee's paper (*ibid.*, December 18th, 1886.)

In some cases of chronic bronchitis recourse may be had with advantage to certain continental spas. According to Braun those waters are the best which contain a considerable amount of chloride of sodium as well as of carbonate of soda. Ems in Germany and Mont Dore in France may be specially mentioned.

When it is thought desirable for a bronchitic patient to spend the winter and spring away from home, the choice lies usually between climates which are soft and "sedative" (such as Torquay, Falmouth, Penzance, Pau, Madeira), those which are stimulant without too much risk of exposure to cold winds (as Mentone and San Remo), and those which are surrounded by pine woods (as Arcachon and Bournemouth).

Plastic Bronchitis.*—This singular affection is certainly nothing else than a bronchitis anatomically; but it has characters so peculiar that from a clinical point of view it would be absurd to group it with the ordinary catarrhal forms. It consists in the exudation of a fibrinous material from the walls of the air-passages which forms "casts" of their channels. A like exudation may occur by extension of a morbid process downwards from the larynx in diphtheria or in membranous croup, and much less frequently by extension upwards from the pulmonary alveoli in pneumonia. But these are totally different in pathology and symptoms. The former has been described with membranous laryngitis in the chapters on Diphtheria and on Croup. The latter will be again mentioned in that on Pneumonia.

It would almost have been better for the disease to have a name altogether unmeaning, since it is not likely that any possible designation could precisely convey the right conception. But for practical purposes the terms "plastic bronchitis" or "fibrinous bronchitis" do well enough, and are preferable to "croupous bronchitis," which suggests an association with laryngeal croup. Some of the earliest recorded specimens were called "bronchial polypi," as were the similar branched masses from the heart of fibrin often tinged with blood, which we now know as coagula.

* *Synonyms.*—Fibrinous Bronchitis—Croupous Bronchitis or Bronchial croup of the German writers—Bronchial Polypi of older authors—Angina polyposa.

The disease is one of the rarest that are known to physicians ("an affection of great rarity," Walshe: "höchstselten," Riegel: "aeusserst-selten," Biermer). The experience of Sir Thomas Watson was remarkable, in having had under his own observation five well-marked examples.

These "bronchial polypi" were known very early. Dr Nicholas Tulp (the lecturer in Rembrandt's famous painting, "The Lesson of Anatomy") records and figures two specimens brought up by a Dutch sea-captain suffering from hæmoptysis: "*Effudit duos insignes venarum ramos, adæquantes singulos expansæ manûs magnitudinem*" ('Obs. Med.,' Amst. Elz., 1652, cap. xiii, p. 122, tab. iii, iv). Afterwards cases were recorded by the younger Bartholin, Cheselden, de Haen, Morgagni, Hunter,* Cheyne of Dublin, and Stokes. Most of the early cases (1690—1730) were published in the 'Philosophical Transactions.'

Anatomy.—In almost all cases of plastic bronchitis the patient soon begins to expectorate masses of the peculiar exudation. It usually appears rolled up into a sort of ball, with a good deal of mucus and blood covering it. All this is easily removed by floating it out in water, and one then sees that there is a complete cast of some part of the bronchial tree, extending perhaps down to its finest subdivisions, so that, according to Biermer, the minute terminal filaments may actually show bulbous ends moulded in the infundibula themselves. The colour of the cast is whitish yellow or grey; its consistence is tough and elastic; it is almost always made up of a number of concentric laminæ, separated here and there by narrow spaces and with a more or less definite central cavity, containing mucus or bubbles of air. Only the more delicate filaments are said to be generally solid.

The laminated structure affords a distinction from the branching clots which are sometimes formed in the air-passages as the result of hæmorrhage, and which are quite homogeneous. Biermer, indeed, is disposed to deny that blood ever coagulates so as to form casts of the bronchial tree. But Dr Walshe speaks of it in the most positive terms, and there could hardly be a better authority.

The casts in cases of fibrinous bronchitis, when examined microscopically, are seen to consist of a hyaline or slightly fibrillated base, in which are embedded large numbers of leucocytes. They seldom contain red blood-discs in any quantity. Several observers have noticed Charcot's crystals in them. In one case Waldenburg found that the thicker parts contained only a few formed elements, but very abundant fat-globules.

The length of a bronchial cast is commonly from one and a half to two and a half inches, but sometimes it may be four or five inches, or even (as in a case of Riegel's) six or seven inches. The diameter of the thickest part of it is seldom greater than that of a goose-quill, being in fact considerably less than that of the space in which it was formed. Biermer has pointed out that from its appearance one can sometimes draw an inference as to the part of the lung from which it came, whether from the short, rapidly-branching tubes of the upper lobe, or from the comparatively longer tubes of the lower lobe. The masses expectorated at different times by the same patient often resemble one another so exactly in size and in the arrangement of their subdivisions as clearly to show that they have all in succession been

* Hunter's case occurs as a short Appendix to his 'Treatise on the Blood, Inflammation, and Gunshot Wounds.' The patient was a man of twenty-two, who spat mucus often mixed with blood. He recovered. The figure agrees with those illustrating the cases of Fuller, Salter, Tuckwell, and the author of the present work, in the 'Pathological Transactions' (vols. v, xvi, and xxi).

derived from the same tract of mucous membrane. For example, in a case recorded by Kretschy seven casts appeared one after the other, all of which came from the middle and lower lobes of the right lung. In fatal cases it is not usually found that the tubes which have poured out the fibrinous exudation show any marked morbid changes. The mucous membrane is sometimes reddened, sometimes pale, and healthy looking. In two instances Biermer found the epithelium still remaining beneath loose casts; but he was himself disposed to think that these cases might be exceptional; and Kretschy has since stated that in his case there was no trace of epithelium in that part of the air-passages which contained the plastic material. The submucous tissue may be swollen and infiltrated with serum. The pulmonary alveoli are healthy, except that some of them are sometimes in a state either of collapse, or of over-distension, or "acute emphysema."

Symptoms.—The expectoration of casts of the lower air-passages is generally attended with severe cough and dyspnœa, the occurrence of which may be the first indication that the patient is otherwise than well. But in many cases there is an antecedent stage during which he appears to be suffering from ordinary bronchial catarrh; and this may last for a long time. Sometimes the disease sets in with rigors, loss of appetite, thirst, oppression of the chest, and pyrexia, so that it may be supposed that an attack of pneumonia is impending. Presently a dry, hard cough appears, which may cause extreme suffering; the breathing becomes rapid, up to 40 or more in the minute; it may be attended with the greatest anguish, as of impending suffocation, with lividity, and with a small tense pulse. There may be some pain in the side, and a feeling of soreness within the chest, but on the whole the attack is more distressing than acutely painful. At first nothing is expectorated, or only a little mucus. The cough may even, it is said, go on for days before any fibrinous masses appear. More often a cast is detached and got rid of after a few hours, and by this the cough and dyspnœa are generally at once relieved, at least for a time. Hæmoptysis often occurs at intervals during the paroxysm, not only at the time when the cast is being expectorated, but also previously. It must therefore be borne in mind that the mere fact of there having been spitting of blood in any particular case in which casts of the tubes are afterwards ejected affords no ground for maintaining that they must necessarily be coagula, and cannot have been formed as the result of a process of inflammatory exudation from the walls of the tubes. But the quantity of blood is not large; perhaps it amounts to a tablespoonful at a time. The case of the late Prof. Daniell, recorded by Watson, is exceptional in the fact that from two to eight ounces were spat on each occasion.

Physical signs.—Examination of the chest throws but little light upon cases of plastic bronchitis. If a large tube is blocked, absence of vesicular murmur may be made out over some part of one of the lungs. The fact that the violent cough fails to clear away the obstacle might perhaps suggest to a keen observer the presence of something more than a plug of mucus such as may prevent the entrance of air in ordinary cases of catarrhal bronchitis, and the diagnosis as to the cause of the obstruction would then lie between fibrinous casts, a foreign body, and stenosis of the walls of the tube. In practice, however, it scarcely ever happens that any suspicion of the real nature of the case arises until a cast has actually been expectorated. There is not usually any change in the percussion-sound, but Dr Walshe says that

he has had repeated occasion to observe dulness, as complete as that of pneumonic consolidation, dependent upon collapse of the lung-substance. He also says that local pneumonia now and then occurs, attended with crepitation and with bronchial breathing, as well as with rusty sputa. When there is extensive blocking of tubes the movements of the corresponding side of the chest may be distinctly impeded, and the lower ribs may even be drawn in during inspiration. Râles are sometimes audible over the affected part of the lung, especially when the cast is becoming loose; some writers have described special sounds as arising in such circumstances, but it does not appear that they are really characteristic.

Course.—It very rarely happens that the expectoration of a single cast brings to an end an attack of plastic bronchitis. As a rule the relief is only temporary, After some hours the cough and the dyspnœa return, and are followed by the appearance of another cast. This process is usually repeated about once in twenty-four or in forty-eight hours for several days, and then the affection slowly subsides, and the patient gets well. Smaller pieces may be spat up at very frequent intervals; being embedded in mucus they sometimes remain unnoticed, unless specially looked for.

Prognosis.—It may well be supposed that the expulsion of such large masses as sometimes come from the air-passages in this disease is not altogether unattended with danger. In 1865 the author showed to the Pathological Society a cast which was taken from the body of a girl, aged seven, having been found lying across the bifurcation of the trachea, with its branches extending into the ramification of the right bronchus, but with its broad end occluding the left bronchus. She had been expectorating similar masses for three days; and on the very day on which she died she had already, at 3 a.m., brought up a cast of about the same size as that which killed her at 3 p.m. in a violent fit of cough and dyspnœa. It is therefore clear that Dr Walshe and others go too far in giving an absolutely favourable prognosis in cases of plastic bronchitis without any reservation. Lebert, in a paper in the 'Deutches Archiv' for 1869, divides his cases (collected from various sources) into acute and chronic; of seventeen of the former, four ended fatally; of twenty-seven of the latter, only three. The distinction, however, appears to be rather artificial; and one of the four fatal acute cases, that recorded by Nonat, was, there is reason to suspect, a case of diphtheria. As a rule, when death occurs, it appears to be the result of the extension of the morbid process into so large a part of the bronchial tree that due aeration of the blood can no longer be effected. In such instances it is preceded by stupor and somnolence. Riegel, however, relates a case in which, although the patient died in an attack of asphyxia, after having been spitting up large casts nearly every day for three weeks, the air-passages were all found empty after death. Lebert has placed in a separate category cases in which plastic bronchitis has run on to a fatal termination without any fibrinous masses having been expectorated. As they occurred chiefly in children, and generally in association with broncho-pneumonia after measles, they should probably be regarded as different from plastic bronchitis.

Recurrence.—When an attack of fibrinous bronchitis has passed off, leaving the patient apparently well, it by no means follows that the disease is really at an end. One of the most curious points about it is its liability to return again and again at irregular intervals, sometimes during a very long period. Dr Walshe met with an instance in which the expectoration

of casts continued, with occasional brief intermissions, from the spring of 1843 to June, 1857, when he lost sight of the patient. In the course of this time she married, and she once resided at Buenos Ayres for four months. Other observers have recorded cases which were scarcely less protracted; in many of them the general health seemed to remain entirely unaffected.

Ætiology.—With regard to the causes of plastic bronchitis, scarcely anything can as yet be said. It is more common in males than in females, the proportion being as three to two, if not as two to one.

The period of life at which it is most frequent is between ten and thirty. One case has been recorded at the advanced age of seventy-two; it had lasted seven or eight years.

Adding to the 31 cases tabulated by the late Dr Peacock in the fifth volume of the 'Pathological Transactions' 24 additional ones, we find that of these 55 cases, 42 occurred in men and 13 in women. (Biermer's figures are 39 male to 19 female patients.) Of 37 patients whose ages are given, five were between 5 and 10, twelve between 11 and 20, ten between 21 and 30, eight between 31 and 50, and two between 50 and 60. The editor has himself met with only two cases, one in a woman of 32 fatal after tracheotomy, the other in a boy of about 10 with repeated hæmoptysis but no evidence of phthisis.

A remarkable circumstance, all the more striking because of the extreme rarity of the disease, is its occurrence in different members of the same family. Fuller met with it in two sisters; Watson relates the cases of two brothers, both of whom were affected within a twelvemonth.

Plastic bronchitis is supposed to be rarer in southern countries than in the north of Europe. Riegel says that, like acute pneumonia, it is most apt to occur towards the end of spring, when there are great daily variations of temperature. In one instance the recurrence of the attacks appeared to be connected with the catamenial periods. Eisenlohr met with a case in which fibrinous casts were expectorated during the second week of enteric fever. There seems no reason to suppose that this remarkable affection is at all related to phthisis.

Treatment seems generally to be altogether ineffectual. Waldenburg, however, saw a case in a girl, aged eight and a half, who for more than four years had been coughing up fibrinous masses at intervals of a few days, and in whom a whey-cure and the daily inhalation of lime-water succeeded in arresting the disease in six or seven weeks. Indeed, a spray of lime-water, or of a solution of an alkaline carbonate, should always be employed in plastic bronchitis; the only doubt is whether they reach the lower air-passages in sufficient quantity. Emetics appear to be sometimes useful; probably it is best to use apomorphia hypodermically. Biermer recommends an active mercurial treatment; others have prescribed iodide of potassium, with apparent advantage.

Dr Walshe believes that neither inhalation of iodine, nor exhibition of alkaline medicines, nor the best of general health, nor the most favoured climates, have the least beneficial effect in preventing or curing the attacks of this paradoxical disorder.

PNEUMONIA

AND INFLAMMATORY DISEASES OF THE LUNGS

PNEUMONIA.*—*History and definition—Anatomy and histology: stages, locality, events—Complications—Physical signs—Clinical symptoms—Prognosis—Mode of death—Convalescence—Origin and nature—Treatment.*
Secondary pneumonia—Chronic lobar pneumonia.
BRONCHO-PNEUMONIA, *or Pulmonary Catarrh.—Causes in children and in adults—Anatomy—Symptoms and course—Prognosis and treatment—Vesicular, caseous, and pyæmic pneumonia—Inflammatory œdema of the lungs.*
CHRONIC INTERSTITIAL PNEUMONIA, *or Cirrhosis of the Lung.—Anatomy—Origin—Symptoms.*
SYPHILITIC INFLAMMATION OF THE LUNGS.—*Cases—Diagnosis—Anatomy—Acquired and hereditary forms.*
GANGRENOUS INFLAMMATION OF THE LUNGS.—*Anatomy—Origin—Fœtor and other symptoms—Treatment.*

Definition.—Although the term "peripneumonia"† dates back to the days of Hippocrates, it is only since the discovery of auscultation, and as a result of the systematic study of pathological anatomy, that a definite meaning has been attached to it in distinction from other diseases of the chest. By *pneumonia* (the peripneumony of older writers) is now understood an inflammation of the texture of the lungs.

But the anatomical condition known as hepatisation which is the result of exudation into the alveoli (for they and not the interalveolar tissue, as was formerly believed, are the seat of the morbid process), although its presence is enough to justify the designation of pneumonia in the dead-house, is far from identical with the disease which is now generally recognised by the term. We need a clinical as well as an anatomical definition of pneumonia, and from the former point of view we must considerably limit the application of the term.

First must be excluded that form of lobular inflammation of the lungs which arises by extension from the bronchial tubes, and has been called catarrhal or lobular or broncho-pneumonia; next the suppurative or pyæmic lobular pneumonia which occurs as the result of infective embolism; then traumatic pneumonia from wounds of the lung, injuries of the chest, or the

* *Synonyms.*—Peripneumony—Fibrinous, Plastic, or Croupous Pneumonia—Acute or Sthenic Pneumonia—Lobar Pneumonia—Acute Inflammation of the Lungs.

† Περιπνευμονία (or in the Attic form περιπλευμονία), from πνεύμων or πλεύμων (whence *pulmo*) the lung, occurs in Plato. It did not originally carry with it the notion of an inflammatory or febrile disorder. The prefix peri- was only dropped in the present century. The Germans have adopted the designation of "croupous," because the exudation mainly consists of a fibrinous material like that which characterises croup. To English ears, however, this has an awkward sound, and the phrase is on other grounds objectionable. It suggests some connection with the malady known as croup, whereas there is none; and it confounds a clinical term with an anatomical condition.

penetration of foreign bodies into the air-passages. It is a question whether the pneumonia which complicates affections of the heart, and that which so often forms the immediate cause of death in persons suffering from almost any chronic or acute disease, particularly enteric fever, ought not each to be placed under separate heads.

There is no question that the caseous pneumonia which accompanies and destroys the lung in phthisis must be separated from other kinds of pulmonary inflammation. Lastly, chronic fibrous interstitial pneumonia is a distinct process both clinically and pathologically, whether it occurs as the conservative element in phthisis, or by extension from the peribronchial connective tissue and the pleura, and whether tubercular, traumatic, or syphilitic in origin.

Each of these so-called forms or varieties of pneumonia requires carefully chosen adjectives to discriminate it, and it would be well if each had a distinctive and substantive name. But when the term pneumonia is used without a qualifying epithet, it is generally understood (except perhaps in the case of children) to refer to the classical peripneumony.

Nevertheless, for the sake of distinction, we may define the pneumonia now to be discussed as *idiopathic* in its origin, *acute* in its course, *lobar* in its extent, *basal* in its usual distribution, and *fibrinous* in the character of its exudation. No doubt lobular and pyæmic inflammations of the lung are often acute, no doubt idiopathic is at best a negative phrase, no doubt the disease often exceeds and sometimes falls short of occupying a complete lobe, no doubt it may affect the apex of a lung : hence no adjective is quite satisfactory. Nevertheless we shall see that in its natural history and its clinical features as well as in its anatomy, pneumonia in the restricted sense of the term is one of the most peculiar and distinctive of diseases.

Morbid anatomy.—The pneumonic process consists of a series of changes by which the spongy pulmonary tissue is rapidly converted into a solid mass, returning afterwards, in cases that recover, to its normal condition. Systematic writers describe several distinct stages.

The first is the stage of "active congestion" or *engorgement.* The affected part of the lung is massive, heavy, and dark red in colour. It pits under the pressure of the finger, and a reddish frothy serum oozes from its cut surface ; when the pressure is increased, its substance breaks down much more readily than that of healthy lung. Microscopically, the most obvious appearance is the dilated and tortuous state of the capillaries of the alveolar walls ; minute punctiform hæmorrhages are also to be seen in the connective tissue between the lobules and beneath the pleura.

Next come the stages of *red hepatisation,* so called because the texture of the lung is solid, like liver. It now sinks in water, it does not crepitate when pressed between the finger and the thumb, it is easily broken, and little or no fluid can be squeezed from it. Its cut surface has a dull, lustreless appearance, and is distinctly granular. The granules are composed of a solid inflammatory exudation, which completely fills up both alveoli and infundibula. Rindfleisch gives drawings of masses of it obtained by scraping the cut surface ; they form complete casts of the interior of the spaces in which they were moulded. The red colour of the lung at this period is probably due partly to the large quantity of blood in its capillaries, partly to the fact that great numbers of red discs are extravasated and mixed up with the exudation. The exudation itself consists mainly of coagulated fibrin, together with more or less numerous leucocytes.

The third stage is that of *grey hepatisation*. This is characterised not only by a change in the colour of the affected parts of the lung-substance, which now passes through reddish grey to grey or whitish yellow, but also by the diseased tissue becoming even softer than before, by its being less markedly granular on section, and by its beginning to emit on pressure a turbid fluid, more or less opaque, white and puriform. The extreme forms of grey hepatisation are in fact described by some pathologists as constituting a fourth stage, which they term *purulent infiltration*. Histologically there is a wide difference between the characters of "red" and those of "grey hepatisation." In the grey stage no fibrinous coagula are visible; the substance which fills the alveoli now appears to consist merely of a mass of crowded leucocytes. The extravasated red discs are no longer to be seen. Rindfleisch speaks of them as becoming decolourised; perhaps it is fair to suppose that they are absorbed by the rapidly multiplying leucocytes (cf. vol. i, p. 55). To account for the change of colour it must also be assumed that the increased amount of exudation compresses the pulmonary capillaries and drives the blood out of them. But Rindfleisch remarks that it would be a great mistake to imagine that this occurs during life to the same extent as after death. He finds that in the dead body it is always easy to inject the vessels, and he therefore concludes that the heart, so long as it is beating, must be able to keep up a more or less active circulation through them. In other words, it is probable that the grey colour is, strictly speaking, a cadaveric change dependent on failure of the circulation after death. A very important histological distinction between the two kinds of hepatisation, however, is afforded by the state of the alveolar walls. In the "red" stage they are unaltered, except that their capillaries are distended; in the "grey" stage they are infiltrated with leucocytes, which fill up every interstice.

Distribution.—Pneumonia attacks parts of the lungs only, never the whole at once. It almost always begins at some one spot, from which it rapidly spreads.

All observers are agreed that the *right* lung is more often the seat of pneumonia than the left, the proportion being about as five to three. Sometimes both lungs are attacked together or, more often, in succession. On either side the *lower lobe* is affected far more frequently than any other part; Jürgensen says that it escapes altogether only in one case out of four, and probably this is above the mark. As a rule hepatisation begins at the extreme base, and extends gradually upwards from day to day. But sometimes it spreads downwards from the summit of the lower lobe, or upwards and downwards from its middle, or backwards from the anterior border. In the upper lobe it may either pass from the apex downwards or from below upwards and forwards. Sometimes its distribution remains strictly limited by the lobar septa; sometimes its spreading edge forms a horizontal line, ignoring them altogether. The most common seat of pneumonia is the right base, next the left base, next both bases. Even at the apex, left pneumonia is more rare than right. Henoch found right apex pneumonia in 21, and left apex pneumonia in only 4 cases out of 74 of lobar pneumonia in children. Dr Goodhart's numbers are 18 right and 17 left apex pneumonia out of 120.

Events.—The results of pneumonia are unlike those of ordinary inflammation. Either death occurs, or the whole affected tissue clears up and returns to its former condition. The formation of an *abscess* in the lung, as the result of true pneumonia, is admitted by all writers to be very rare, and many, including the present writer, doubt whether it ever occurs. Cases

have, indeed, been recorded; but the question is whether a more accurate pathology might not have led to a different interpretation. They may have been circumscribed empyema, or suppurating bronchial sacs or hydatids; or true pulmonary abscesses, but not of pneumonic origin—traumatic, pyæmic, or gangrenous. The termination of this disease in *gangrene*, which is also very infrequent, is undoubtedly sometimes seen under special circumstances to be afterwards discussed (*infra*, p. 158).

When pneumonia ends in *resolution* an interesting question presents itself; whether it is possible for the disease to pass through all its three stages above described and yet afterwards to undergo recovery. Now, it is certain that pneumonia not infrequently subsides at quite an early period, before there is reason to suppose that any part of the lung has passed beyond the stage of engorgement. But it is equally certain that many other cases end favourably after there has been clear evidence from physical signs that the pulmonary tissue has become "consolidated," *i. e.* has reached the stage of red hepatisation. The doubt is whether the supervention of the third stage of grey hepatisation or of purulent infiltration is compatible with recovery. In their work on Pathological Anatomy, Wilks and Moxon express a rather decided opinion that in most cases the disease does not advance far beyond the "red" stage before resolution begins. An opposite view seems to be generally entertained, chiefly in consequence of theoretical conceptions as to the nature of the process by which resolution is affected. There are obviously two ways in which the pulmonary alveoli may be emptied of the exudation that fills them; one is by its escaping into the air-passages and being expectorated; the other by its being reabsorbed into the blood. Now, Rindfleisch maintains that most of it takes the former course; but I think that every clinical physician will agree with Jürgensen (in the article on pneumonia in 'Ziemssen's Handbuch') that he is mistaken, inasmuch as in many cases sputum is altogether wanting at this period of the disease. However, before absorption can occur it is generally supposed that the exudation must liquefy and undergo a change more or less analogous to that of fatty degeneration. And the assumption has been that this is equivalent to the conversion of red hepatisation into grey. But that such an assumption is erroneous is apparent from the description given above of the histology of the more advanced stage of the disease, which we have seen to be attended not merely with softening of the inflammatory products, but also with a greatly increased infiltration of leucocytes. It appears, therefore, that those are right who hold that when this stage is reached recovery is no longer possible.

After the subsidence of pneumonia, if the patient should die at no long interval from some other disease, the lung is found to have nearly regained its healthy appearance, but to be slightly redder and tougher than natural. Two such cases at least have been observed at Guy's Hospital, but unfortunately no microscopical investigations were made as to the exact state of the pulmonary tissue. Rindfleisch speaks of a "loss of elasticity" as resulting from pneumonia, and as continuing a long time after recovery.

It is a very important question whether, instead of subsiding, true pneumonia ever leads to permanent changes in the lung, to the development of fibrous tissue in it, constituting what is termed cirrhosis or chronic pneumonia, or to a destructive process, ending in the formation of cavities more or less like those that are seen in phthisis. Now, as regards cirrhosis, though some observers (including Wilks) are opposed to the belief that it ever arises in this way, cases will hereafter be cited which seem to prove that

such a result does occur, although very rarely. But as for the supposed termination of pneumonia in phthisis, there is every reason to believe that the cases that have been so interpreted were phthisical from the beginning.

Complications.—Few diseases are so seldom as pneumonia varied by complications, *i. e.* by occasional additional morbid processes.

Pleurisy of the dry kind is a constant part of the disease. Whenever the inflammation reaches the surface of the lung the corresponding part of the pleura always becomes covered with lymph. Accordingly, some physicians speak of the disease as "pleuro-pneumonia," at least in those cases in which they discover physical evidence of pleurisy during life. But this seems to be an unnecessary refinement, and indeed to be rather misleading, since on *post-mortem* examination lymph is found upon the pleura, even when there had been no signs of its presence. It is better to reserve the term pleuro-pneumonia for cases in which the pleurisy leads to fluid effusion, and so becomes of clinical importance.

Not infrequently there is likewise *pericarditis*, which either has arisen by extension from the pleura, or has developed itself simultaneously in the serous membranes covering the heart and the lung, as well as in the lung itself. Occasionally the peritoneum also, especially the upper part of it, is found coated with lymph. Sometimes the mediastinal tissues are extensively infiltrated with a gelatinous exudation.

The bronchial lymph-glands are constantly found enlarged, pinkish-grey, and soft. The sub-pleural lymphatics corresponding with the seat of the pneumonia may be not infrequently observed to be distended with inflammatory products so as to give a marbled appearance to the surface.

As a very rare complication of pneumonia must be mentioned acute meningitis. Four cases have been already narrated (cf. vol. i, p. 700); two of these were cerebro-spinal meningitis. One was complicated by diphtheria, one by chronic Bright's disease, and one by the possibility of a traumatic or pyæmic origin. Less infrequent is ulceration of the larynx; the ulcers are found over the arytænoid cartilages, just where they occur in so many other circumstances.

Probably it is to the pyrexia which accompanies pneumonia that one should refer some other slight but almost constant morbid changes that are met with after death: a moderate degree of enlargement of the spleen, catarrh of the intestine, and cloudy swelling of the kidneys.

Signs.—It is by physical examination alone that the seat and extent of pneumonia can from day to day be determined with accuracy, although the other symptoms commonly enable one to form a confident opinion both as to the nature of the disease and as to the part of the lung in which it is most likely to be found.

The earliest signs are generally recognised by auscultation. According to Stokes, there is first audible a peculiar harshness of the vesicular murmur. But more often the first sign detected is an entirely new or adventitious sound, which is called *pneumonic crepitation* or sometimes "fine crepitation."* This is exactly like the sound produced by rubbing a lock of hair between the fingers close to one's ear, a comparison suggested by Dr C. J. B. Williams.†

* *Fr.* Râle crépitant.—*Germ.* Das knisternde Rasseln.

† It is much "smaller" than any other râle, and would not be recognised as due to bursting of bubbles, like gurgling or mucous rattles. It is consonating, musical in character, and high pitched in note. Laennec calls it "une espèce de crépitation ou de râle léger, dont le bruit peut être comparé à celui du sel que l'on fait décrépiter en le chauffant dans une bassine."

By Laennec, who discovered it, it was supposed to be pathognomonic of pneumonia. But it is sometimes not heard in undoubted cases of this disease (possibly because it lasts but a short time), and it is now known to occur with œdema of the lungs. A sound very like it, if not absolutely the same, may often be heard for successive inspirations in the bases of the lungs of a person who has been lying on the back with some febrile disease, if he is made to sit up and breathe deeply, so as to fill those parts which have been for some time disused. In the last case there can be no doubt that the cause of the sound is the opening up of portions of the tissue that had become collapsed; and almost all observers are now agreed that in pneumonia it has a similar origin, being due to the inspiratory separation of the walls of alveoli and bronchioles which, being swollen, had come into contact in expiration. Crepitation is, indeed, heard only during inspiration, and sometimes only just at the end of deep inspiration, as after coughing. Its mechanism is therefore not that of other râles, whether crepitant or non-consonating; it does not depend on the bursting of bubbles, and should always be distinguished from true "moist sounds."

One must search carefully for this important sign before concluding that it is not present. But, on the other hand, the fact must be admitted that in many cases of pneumonia this sign is not at any time discovered. Whether it is ever really altogether absent in such cases is perhaps doubtful, because it is in its nature transitory, and so may have passed off before an examination of the chest was made. Sometimes, however, it remains audible during almost the whole length of the disease; not, indeed, at the same spot, for where there is complete consolidation it almost necessarily disappears; but in one spot after another, as they are successively attacked by the inflammatory process.

As the state of engorgement passes on to that of red hepatisation, there are developed other signs, the chief of which may be briefly summed up under the heads of dulness on percussion, bronchial breathing, and bronchophony with increased tactile vibration.

The degree of *dulness* varies much in different cases. There is never the absolute tonelessness that is met with in cases of fluid effusion into the pleura; nor is the sense of resistance to the finger so great. Sometimes, however, the percussion-sound undergoes very curious modifications, the explanation of which is by no means obvious. Thus a cracked-pot sound is in exceptional cases elicited; this is attributed by Dr Gee to the presence of "islets of unsolidified lung embedded in the substance of the hepatised tissue." In other rare cases the sound has a more or less markedly tympanitic quality. Sometimes the spot where this tympanitic sound is heard is not directly over the consolidated part of the lung, but in its neighbourhood, over pulmonary tissue which may be imagined to be relaxed as the result of the increased bulk of the consolidated part. Thus hepatisation of the upper lobe of the lung behind may lead to the occurrence of a tympanitic percussion-note beneath the clavicle on the same side.

The *bronchial breathing* that accompanies the second stage of pneumonia may exhibit every variety of quality up to the amphoric. Usually it is a typical in-and-out, whiffing, tubular sound, equal with expiration and inspiration, and in quality not unlike a to-and-fro bellows murmur of the heart. But occasionally no such sounds are audible, and the explanation doubtless is that the tubes passing to the consolidated part happen to be filled with fibrinous plugs.

Bronchophony generally goes with bronchial breathing, and presents like differences of degree. Dr Gee, however, remarks that in infants a bronchophonic cry is often the only auscultatory sign that can be obtained.

Increase of vocal fremitus, although sometimes well marked, is by no means constantly present. Probably its occurrence depends upon the state of the tubes leading to the consolidated part. If they are full, even of fluid secretion, the transmission downwards of the coarser vibrations contained in the voice is interrupted. It is only when the left side is the one on which the fremitus is greater, that this sign can be regarded as of high clinical importance, because on the right side it is very often greater in healthy persons.

It remains to be mentioned that when pneumonia affects only the central part of the lung, or reaches no part of the surface except that which is in contact with the diaphragm, physical signs are altogether absent. What is more common is for the disease to begin deeply in the interior, but to become superficial a few days later, so that all doubt as to the nature of the case is soon at an end.

During the stage of *resolution*, the peculiar auscultatory signs of hepatisation disappear, and they are commonly replaced by râles, which may vary widely in character. Sometimes a crepitation is now heard, which may be scarcely less fine than that of the early period of the disease; this is spoken of as *crepitatio redux*. In other cases the sounds are so large, and at the same time so highly consonating in quality, that one might imagine the patient to be at an advanced period of phthisis, with the lung breaking up in all directions. Yet after a few days these sounds in their turn cease to be audible. A considerable time generally passes before the vesicular murmur becomes as loud and as distinct as it normally should be. The percussion-resonance also may long remain deficient, but chiefly in cases of which a marked feature has been concomitant pleurisy.

Clinical course.—An attack of pneumonia commonly sets in suddenly with a well-marked rigor. This occurred in 241 out of 280 cases, and again in 782 out of 975 cases ('Collective Investigation Record'). Sometimes, however, there is not more than a sensation of chilliness, which may almost escape notice. Sometimes, particularly in children, the disease is ushered in by a convulsive seizure, or by vomiting. As a rare exception when idiopathic, but most frequently when supervening as a secondary complication of other diseases, the onset of pneumonia is insidious, there being no symptoms to mark the exact time at which it begins.

Pyrexia develops very rapidly. Jürgensen cites a number of observations, which show that the temperature may reach 104°, or even a higher point within three or four hours from the shivering. At the same time the pulse is quickened, and it becomes full and bounding in character. There are the usual complaints of malaise, headache, pains in the limbs, and anorexia, as in every other febrile disease. As the rigor passes off the cheeks acquire a crimson flush. A point on which Addison used to lay great stress is that, as tested by the hand, there is in pneumonia a pungent heat of skin hardly observed in any other affection. We saw how this has since been confirmed by the observations of Schülein (vol. i, p. 46).

So far, there is nothing in the symptoms to show that the thoracic organs are the seat of the patient's illness. Before auscultation was practised it was not uncommon for a case of pneumonia to be called "typhus" or "continued fever," and for the autopsy to show for the first time the real

nature of the case. Or if, as sometimes happens, there was violent delirium from the very commencement, and the patient became comatose and died in twenty-four or thirty-six hours, the disease was set down as "meningitis" or "encephalitis." But even then a careful observer seldom failed to notice indications that led him to form a correct judgment. One point of great importance, upon which Dr Walshe has especially insisted, is a change in the ratio of the pulse to the respiration. Healthy persons breathe once for every four or four and a half pulsations of the heart. In febrile diseases generally, both pulse and respiration are more frequent; but the relation between them is not much, if at all, disturbed. In pneumonia the increased frequency of the respiration is out of all proportion to that of the pulse, the ratio being as one to three, or one to two, or higher still; for the patient may breathe sixty or eighty times a minute. Dr Walshe has observed the number of respirations in the minute actually greater than that of the heart-beats. Jürgensen says that the cases in which this occurs are those of old people with slowly-acting hearts and atheromatous vessels. A curious circumstance, to which Dr Walshe again has drawn attention, is that the amount of subjective distress produced by such rapidity of breathing varies extremely in different cases; some patients breathing thirty, forty, or even sixty times a minute, are wholly unconscious of any dyspnœa. Another indication that should draw attention to the respiratory organs as the probable seat of the disease is, that the nostrils work during inspiration, and that the sterno-mastoidei and trapezii are tense and prominent. It may also be observed that the flushed cheeks and the lips have a slightly purplish tint. At present every educated practitioner, after noticing the aspect, and ascertaining the state of the pulse, respiration, and temperature of a patient, examines in every case, as a matter of routine, at least the apices of the lungs in front and their bases behind. Even with careful scrutiny, however, there is sometimes complete absence of the physical signs above described for the first twenty-four or forty-eight hours.

But in many cases there are present from an early period, or from the very beginning of the illness, symptoms which point clearly to its true nature, and even show in what part of the chest the lesion is to be found.

One such symptom is *pain*, which is commonly situated near the nipple or towards the axilla, and which may be the first thing to suggest to the patient that he is otherwise than well. This pain is identical with the *point de côté* which will be described as occurring in pleurisy; and in all probability, in cases of pneumonia, it is really due to the coincident pleurisy, as Addison long ago taught. It is generally urged, as a reason for adopting this opinion, that in many cases of pneumonia there is no pain whatever. But that obviously proves nothing, unless it can be shown that in such cases there is also no pleurisy; and this is not the case. Indeed, pleurisy itself is sometimes painless. Sometimes the pain does not appear until the patient has been ill some hours, or not until the second day or even later. It is exceedingly severe, and being increased by every movement, it leads the patient to endeavour to fix the ribs by pressure with his hand, and also causes him to curve his spine towards that side, so as to bring the ribs as close to one another as possible. Sometimes the act of drinking produces so much suffering that the patient will endure the great thirst produced by the disease rather than attempt to swallow.

The *cough*, which is another early symptom, is also modified by the pain, which interrupts and as far as possible cuts it short. The distress caused

by the cough is greatly increased by the fact that the *expectoration* in pneumonia, though very scanty, is singularly tenacious and viscid, so that it is got rid of with extreme difficulty; even when it has reached the lips, it often clings to them, and can only be removed by the handkerchief; and it adheres equally firmly to the spittoon, which may be inverted without any of it escaping. Pneumonic sputum has also a peculiar colour, due to the circumstance that the frothy mucus of which it consists is intimately mixed with blood that has perhaps undergone slight chemical changes since it left the vessels. This colour varies in shade in different cases. It is usually bright orange, tawny, or like the rust of iron, so that it is commonly called "rusty" expectoration. But sometimes the tint is a paler apple yellow, and sometimes it is the bright scarlet tint of unaltered blood. Spitting of pure blood in such quantity as to deserve the name of hæmoptysis is not common; but it may occur in cases uncomplicated by tubercle, purpura, or Bright's disease, and the result may be as good and speedy a recovery as usual.

In some cases the sputum is thin, watery, and of a brownish-red colour, so that it is compared with prune-juice or liquorice-water. Such sputum is seen chiefly at advanced stages of the disease, and when it is taking an unfavourable course. But it may be present at an early period, and in cases that ultimately do well. Probably, as Dr Wilson Fox suggested, it really comes from a part of the lung affected with œdema.

Some patients, usually children, have no cough, and therefore no expectoration, throughout the whole course of pneumonia.

Remak showed, many years ago, that in pneumonic sputum there can sometimes be detected with the microscope branching fibrinous casts of the smallest bronchial tubes. Micrococci are sometimes, but not constantly, present (p. 142).

Having thus developed itself, pneumonia commonly runs on for some days with but little variation in its symptoms. As more and more of the pulmonary tissue undergoes hepatisation, the physical signs become more and more marked; but otherwise the case often seems to show no proportionate increase in its severity.

The type of the *pyrexia* is as a rule continuous, with the usual diurnal oscillations. Wunderlich, however, points out that it is not uncommon for an irregular and sudden elevation of temperature to occur, which is followed by a no less sudden fall through as many as 7° or even 9° F. to the normal point or below it, and that again in a few hours by a fresh elevation. We have observed more than one case at Guy's Hospital in which similar strange deviations in the regular course of the fever took place again and again, and, in fact, made up the greater part of the temperature chart, without any explanation being found.

The *pulse* may gain in frequency from day to day, but sometimes it remains for several successive days at 90 or 100, the only change in it being that it becomes gradually smaller and softer, or even dicrotic in character. In adults there is always ground for alarm if it rises to 120, but in young children it may reach 130 to 140 without there being any danger; in old people it commonly stands at a much lower point.

The *tongue* is of course furred in pneumonia; as the disease advances it often becomes dry, brown, and covered with sordes. The bowels are generally constipated, but sometimes there is diarrhœa.

A symptom, which usually makes its appearance between the second and

the fifth day, is an eruption of *herpes* upon the lips, and sometimes at the anus, or even upon the limbs. It is said to occur in from two fifths to one half of all cases of pneumonia; and what is very strange is that it is far more common in cases that do well than in those that end fatally. This has long been a traditional opinion, and it seems to have been established by the observations of Geissler, who found ('Arch. d. Heilkunde,' 1861) that out of 421 cases the mortality among those in which there was no herpes was at the rate of 29·3 per cent., whereas among those with herpes it was only at that of 9·3 per cent.

After the first few days the *skin* in many cases becomes moist, and there is occasionally profuse sweating. An icteric tinge of the conjunctivæ is not uncommonly present, and sometimes there is well-marked *jaundice* of the skin. Formerly it was taught that this occurred only when the base of the right lung was the seat of the disease, and that it was due to the extension of inflammation through the diaphragm to the liver-substance. But the truth is that sometimes in these cases the pneumonia affects the upper lobe of the right lung or some part of the left lung. The explanation would seem to be that the jaundice is due either to venous congestion of the liver transmitted from the lung through the right side of the heart, or to a catarrh of the common bile-duct accidentally present as a complication.

The *urine* in pneumonia is scanty, of high specific gravity, high coloured, and strongly acid, depositing urates in abundance. The amount of *chlorides* excreted by the kidneys undergoes a great diminution; they may even be altogether absent. At one time it was thought that this was of considerable diagnostic importance. But the same thing occurs in almost all other febrile diseases, the explanation being apparently that the salts in question are retained in the body, for, as Cohnheim argues, the diminished supply of them in the food is insufficient to account for so great a difference. During convalescence they reappear, probably in increased quantity. Not infrequently the urine contains *albumen*. This probably happens in from a fourth to a third of the cases. It is not of ill prognosis and never leads to permanent renal disorder.

The *nervous system* is, as a rule, less affected in pneumonia than in other maladies with equally high temperatures. Many patients retain their consciousness throughout the whole of the illness, or merely wander a little at night during their broken sleep. But in other patients *delirium* is a very prominent symptom, and it may be a furious maniacal type. This is said to be more often the case when the upper than when the lower lobe is the seat of the disease. In persons who have been intemperate pneumonia is often attended with symptoms exactly like those of delirium tremens, or, to put it in another way, that affection may be present as a complication. Another thing that seems to determine the occurrence of severe cerebral symptoms in pneumonia is an inherited neurotic tendency; in one of the most terrible cases, that of a man whose last hours were passed in a paroxysm of raging madness, it was stated that there was hardly one of the brothers and sisters of the patient who had not suffered from one form or another of nervous disease. Apex pneumonia is particularly apt to cause delirium, or in children convulsions.

Prognosis.—A fatal termination of pneumonia is, on the whole, far less frequent than might have been anticipated. But to state with accuracy what is the average death-rate in this disease is at present impossible. One difficulty is that it varies enormously, according to the number of primary

cases in proportion to those which are secondary to some other disease; many of the latter are obviously hopeless from the first. Then the danger of pneumonia is widely different at different ages—children almost always recover from acute lobar pneumonia; and in young healthy adults, of temperate habits, recovery is the rule; Jürgensen gives the death-rate in such cases as from 3 to 6 per cent. But to old people, *i. e.* to all above sixty—or to those who, though not old, are worn out by overwork, dissipation, or drunkenness*—the disease is exceedingly fatal. Nevertheless, from time to time one sees a patient recover even when the circumstances have appeared most adverse.

The most important considerations as to the prognosis in a case of pneumonia—apart from the age of the patient (which is almost as important as in typhus or in diabetes) and his habits—are the presence of renal disease and the presence of heart disease; either of these complications makes even a limited consolidation of a single lung in a young and temperate person of the gravest significance.

In an individual case, danger is indicated if the local lesions are extensive, and especially if both lungs are affected. But experience has taught that it is never safe to speak confidently of the prospect of recovery of a patient with pneumonia, however favourable its course may be during the first few days. For what was single may soon become double pneumonia; a change for the worst is apt to occur suddenly; the pulse, of moderate frequency hitherto, runs up quickly, respiration becomes more frequent and shallow, the strength fails with terrible rapidity, and in a few hours the end may come. In all probability the cause of the fatal issue in such cases is the supervention of inflammatory œdema in parts of the lungs previously unaffected. Other cases go steadily downwards from the very commencement.

In children lobar pneumonia is generally of good prognosis. But the less apparently severe cases which appear in rachitic children, or after measles and whooping-cough, and perhaps are really catarrhal pneumonia affecting a number of lobules close together, are more dangerous, particularly in children under three years old. Of 120 cases of lobar pneumonia in children, Dr Goodhart lost 25, but this was chiefly in out-patient practice.

Modes of death.—Whatever the reason of the fatal event, the symptoms which precede death are generally the same. The breathing becomes more and more rapid, and it is shallow and ineffectual, the patient being no longer conscious of the necessity for filling his lungs, as is shown by his sinking down in bed away from the pillows by which he had been propped up; the face is intensely pale and livid in hue, leaden or slate coloured; the skin is covered with a cold sweat; the pulse becomes weak, irregular, and finally imperceptible. But in certain cases death is sudden, from the heart failing during some effort, as when the patient raises himself up in bed. Lastly, it now and then happens that the occurrence of the crisis (which will be described in the next paragraph) is followed by great prostration and collapse, from which the patient never rallies.

Defervescence.—In cases of pneumonia that end in recovery, the subsidence of the pyrexia and of the other symptoms commonly takes place

* Of 37 cases of pneumonia in persons reported as intemperate, 15 died; of 228 reported "temperate," 42 died; and of 80 total abstainers, 9 died ('Collective Investigation Record,' vol. i, p. 95). The third class would include a large proportion of children and young adults, and the first would probably include a large number of elderly people.

abruptly, *by crisis*. And in about sixty-five cases per cent., when the exact duration of the disease can be reckoned from there having been an initial rigor, or convulsive seizure, or attack of vomiting, the crisis is found to occur between the fifth and the eighth days, often on the sixth, and more often on the seventh. In some cases it is earlier; in others it is delayed until some time in the course of the second week. Sometimes fever continues longer than this; but Jürgensen is probably right in thinking that whenever defervescence fails to occur by the fourteenth day there is ground for suspicion that the case is either not one of pneumonia in a strict sense, or else that some complication or other is present. It almost always happens that the crisis begins in the evening or night, scarcely ever in the forenoon. The fall of the temperature is usually rapid, being completed in about sixteen hours, or sometimes in five or six hours; but in some cases from twenty-four to thirty-six hours pass before the thermometer reaches 98·5°. For the next two or three days it often stands below the normal point; the lowest reading observed by Jürgensen during this time was 95·5°, in the rectum.

Less frequently the defervescence in pneumonia takes place gradually, *by lysis*, and occupies two or three days.

In cases which end by crisis, it is surprising how much better the patient feels as soon as the pyrexia has subsided. His appetite quickly returns, he sleeps well, his skin perspires comfortably. Both the pulse and the respiration decline step by step with the temperature. Yet the physical signs may at first fail to indicate any improvement in the state of the affected lung, and several days may pass before the consolidation can be said to have cleared up to any great extent.

There are no *sequelæ* of pneumonia. The patient may be allowed to eat as his appetite returns, and to go abroad when he feels able to. There is no danger of phthisis or other chronic affections following. In this respect it resembles typhus, and differs widely from enterica, and from pleurisy or lobular pneumonia.

Recurrence.—A person who has once suffered from pneumonia is afterwards more likely to be attacked by the disease than if he had not already had it. Instances have been recorded in which it has recurred eight, ten, and even twenty-eight times. Relapses are not frequent, if by this term we understand the repetition of the morbid process before the patient has completely recovered from a former attack. When this happens, it probably ought to be regarded rather as an independent illness, due to the renewed operation of the exciting cause upon a system exhausted by pyrexia. Sometimes the same part of the lung is affected on successive occasions, sometimes not.

Ætiology.—Pneumonia is met with at all ages, though rare under three years of age; and in both sexes, though commoner in men than in women. It is met with in all climates and in every race of the human family. Moreover, it is common among brutes. Pleuro-pneumonia is a frequent cause of death in monkeys, horses, and among cattle. It frequently assumes an epizootic form and decimates both horned cattle and sheep.

Different opinions have been expressed as to whether pneumonia is more apt to occur in persons who are strong and healthy, or in those who are weak and delicate. There is no difficulty in finding instances in support of either view, and one cannot help being profoundly impressed by the cases that one now and then sees of vigorous young men carried suddenly off by this disease. Yet these are, after all, exceptions. "Acute pneumonia

occurring in a previously young and healthy subject" is more often described than seen.

If we take the cases of hepatisation of the lung, as they occur in the dead-house, the majority are found to be secondary to cardiac or renal disease, to enteric or other specific fevers, or to wasting maladies like cancer, diabetes, and paraplegia. On the other hand, it is not frequent for acute lobar pneumonia to be associated with phthisis, with bronchitis, or with primary pleurisy.

Even if these cases be excluded and we examine the records of apparently primary idiopathic pneumonia, we find, as Dr Wilks has long taught, that in many cases the patient is old and feeble, or half-starved, or habitually intemperate.

Occasionally pneumonia assumes an *epidemic* character among men, as among cattle. This is seldom seen now, but there is reason to believe that, like measles, syphilis, influenza, and perhaps the sweating sickness, it was once a more frequent type, and that its present sporadic occurrence has not been, and possibly may not be, always the rule.

In Dr Sturges' interesting monograph there are accounts of epidemic and very fatal pneumonia in Flanders during 1557, in the garrison of Philipsbourg in 1688, and in Iceland in 1863. He also refers to an epidemic in the 22nd Regiment, when stationed in New Brunswick, recorded by Dr Walshe in the 'Army Medical Reports' for 1867, and to one in the Mediterranean Fleet in 1860, described by Dr Bryson in the 'Lancet' (Jan. 9th, 1864). Lastly, the epidemic occurrence of pneumonia is recorded in an account published in 1883 by Jürgensen, of the prevalence of the disease from 1873 to 1881 in the village of Lustnau, near Tübingen.*

Cases have been reported of *infectious* pneumonia. One striking instance of five adult brothers and sisters, living in the same house and successively carried off by the disease, was described by Dr Patchett in the 'Lancet' for 1882. It had occurred in 1876.

The principal exciting cause of pneumonia—undoubtedly a cause of pleurisy and of bronchitis—is commonly supposed to be "catching cold." There is no physician who cannot recall to his memory numerous instances in which patients were attacked immediately after getting wet through, or after lying on damp grass, or after some other very definite exposure to cold. But writers who have tabulated their cases, with the object of determining the frequency of the operation of cold as a cause of the disease, have failed to trace it in any but a comparatively small proportion. Among 205 cases collected by Grisolle, 45 were, indeed, supposed to be due to cold; but among 186 cases of Ziemssen's only 10, and among 212 cases of Griesinger's, only 4 could be clearly attributed to this cause. (These figures are taken from Jürgensen's article in 'Ziemssen's Handbuch.')

Again, in the relations of pneumonia and of bronchitis respectively to different climates and to different seasons of the year, there appear to be certain differences that would hardly have been anticipated on the view that the two diseases have the same ætiology. Pneumonia does not, like bronchitis, increase in frequency with the latitude, from the equator towards the poles. And with respect to the various seasons of the year, observations made at Vienna have shown that whereas in that city the prevalence

* See a full bibliography by Dr Coupland in the Report on Acute Pneumonia, 'Collective Investigation Record,' vol. ii (p. 10); also four instances in vol. i (p. 104), and a very full and important account of an epidemic of pneumonia in the Punjab by Surgeon-Major Maunsell, vol. ii, p. 77.

of bronchitis reaches its maximum in March and then gradually declines through the rest of the spring and summer, the prevalence of pneumonia increases steadily from February to May, after which it falls rapidly. This last part of the statement must not be taken as applying to other places besides Vienna, still less to Europe generally; for Jürgensen points out that there is a broad difference between continental and insular climates as regards the months in which pneumonia is most apt to occur; in the former it is between March and May, in the latter between December and February. But if the year be divided into two halves, the one from December to May, the other from June to November, then it is found that, throughout Europe, two thirds of the cases of the pneumonia fall into the first half, one third into the second. Another point which tends to confirm the opinion that pneumonia is not generally directly due to cold is that sailors and others whose occupations expose them to bad weather do not seem especially prone to be attacked.

Again, true pneumonia is not set up by local *injuries* to the chest, wounds of the lungs, foreign bodies in the bronchi, or any like causes. Traumatic inflammation of the pulmonary tissue is chronic and interstitial, or may be acute and caseous, but it is not hepatisation. It is impossible to induce the affection experimentally in animals. Sommerbrodt stated that he saw it follow the injection of a solution of perchloride of iron into the air passages; but Jürgensen repeated the experiment, and the result was not the same.

In the pneumonic lung as in so many other morbid structures, the discovery of microphytes has rewarded the diligence and skill of modern histologists. Friedländer first described in 1882 micrococci, usually occurring in pairs or chains, and surrounded by an envelope (*Diplococcus pneumoniæ*). They are frequently if not constantly present in the hepatised lung, and have been found in the rusty sputum. Dr Giles found them in India ('Brit. Med. Jour.,' July 7th, 1883). But they may also occur in lobular and other kinds of inflammation of the lung. Moreover, other microphytes, spherical and rod shaped, have been found in cases of pneumonia.

Pathology.—The traditional view is that pneumonia is an acute inflammation of the lung, and that the pyrexia and other symptoms are seondary to the local lesion.

For many years, however, doubts have been expressed as to whether this is the true pathology of this remarkable disease; and it is possible to regard pneumonia as a general and specific disease to which the hepatised lung bears the same relation as the intestinal ulcers to enteric fever, or the angina to scarlet fever. The following considerations bear upon this question, which is far from having only a speculative interest.

(1) Are we right in assuming hepatisation to be an inflammatory process at all? We have seen that it cannot be caused by injury or irritants. It does not lead to suppuration on the one hand or to fibroid induration on the other. The exudation is peculiar and unlike that of undoubted inflammation which has extended from the bronchial tubes to the air-vesicles. The distribution is remarkable. The disease never or scarcely ever assumes a chronic course.

On the other hand, the inflammatory nature of the process seems witnessed to by the constantly concomitant pleurisy and occasional extension to the pericardium; and by the exudation of fibrin and leucocytes along with blood-discs. Moreover, we are familiar with other cases of non-traumatic inflammations which are more or less peculiar to the organs they affect,

and which as they do not originate from ordinary irritants, so fail to produce the ordinary results of inflammation. Such are tubal nephritis in Bright's disease, membranous laryngitis in diphtheria, and acute yellow atrophy of the liver.

(2) If the characteristic lesion of pneumonia may be accepted as a special form of inflammation, it is not to the catarrhal inflammation of mucous but to the plastic inflammation of serous membranes that it should be compared. The pulmonary alveoli are lined by endothelium with lymphatic stomata, not by columnar epithelium with a vascular mucosa. This is in all likelihood developed from the mesoderm like the pleuro-peritoneal lining, and not, like that of the bronchial tubes, from an outgrowth of the hypoblast. And the alveoli, not the "parenchyma of the lung," is the primary seat of the exudation of acute pneumonia, as Addison proved nearly fifty years ago.

(3) If the other symptoms of pneumonia are secondary to the pulmonary lesion, why is the temperature so much higher than in other inflammations, whether serous, mucous, or visceral, higher than in any other disease except specific fevers, tuberculosis, or pyæmia? Why is there albuminuria with cloudy swelling of the renal epithelium, which disappears with the pyrexia, and never leads to permanent local changes? Why are the chlorides so remarkably deficient in the urine? Why do the general symptoms sometimes precede evidence of the local lesion, when we cannot refer them to patches of pneumonia too far from the surface to give physical signs of their presence?

Again, it has been asked why the general symptoms are so independent of the severity and extent of the local lesion. But its "severity" is probably the same, or nearly so in every case, and its extent is certainly not without influence; double pneumonia is a more serious disease than single, and consolidation of three fourths of a lung than of half its lower lobe.

Again, the course is said to be too typical for that of a local inflammation. Why does crisis often occur at the end of a week, and why is convalescence so rapid and complete?

Pneumonia, though not infectious except in the rarest cases, sometimes occurs, as we have seen, in epidemics.

Lastly the presence of a microphyte, even if not yet ascertained to be constant and characteristic, suggests a specific origin and character.

In the first edition of the present work, the author regarded as not disproved the common opinion that pneumonia is essentially a local inflammation, accompanied by a symptomatic pyrexia. "The truth," he remarked, "seems to be that the question of the real pathology of pneumonia is involved in a much wider one, to which at present no positive answer can be given. We have seen, in the chapter on inflammation, that many modern observers believe that no form of spreading inflammation is due merely to the reaction of the organism against a local injury. If this be the case, some specific exciting cause, such as Jürgensen assumes for pneumonia, must be supposed to be present in a vast number of other inflammatory diseases. Probably many years will pass before the true relations of all of them are fully understood. In the meantime it would not be really advancing in the right direction to detach pneumonia from other thoracic affections, with some of which it may after all prove to be closely allied, or to force it into a close relation with the group of acute specific diseases as now understood, from which it certainly differs in many important particulars." It appears to the present writer that at present the evidence tends in the

other direction, but much depends on the view taken of the pathology of inflammation generally, and on the validity of the distinction between acute pneumonia and other forms of inflammation of the lung.

Treatment.—Pneumonia has been the battle field of therapeutics no less than of pathology since it was first clearly described clinically by Laennec and anatomically by Rokitansky.

During the first period—from 1820 to 1840—antiphlogistic treatment was vigorously applied to this, the typically sthenic inflammation in a young and healthy subject. In France especially, "la saignée coup sur coup" was practised in the hope and belief of "jugulating" the disease. In England twenty ounces of blood and more were often abstracted from the arm, time after time, or the patient was bled in the recumbent posture so as to obtain a larger flow before syncope occurred. Any failure was ascribed to want of early and bold venesection.*

Mercury, usually in the form of calomel and opium, was also given as a necessary adjunct to venesection, particularly in England. Antimony was often conjoined with mercurials, and in Italy it was given in large and nauseating doses as a specific remedy for pneumonia.

A great revulsion of practice occurred in this country in the fifth and sixth decades of the century against "antiphlogistic" and in favour of "corroborant" treatment of inflammations and of acute diseases generally. Wine and brandy, often in "heroic" doses, were prescribed under the influence of the late Dr Todd.

Meantime, some physicians, among whom the late Dr Hughes Bennett, of Edinburgh, deserves the foremost place, had been observing the natural history of pneumonia and had found that, at least in young and temperate patients, its danger had been much over-estimated, that it tended to recovery after a week's duration, and that neither bleeding, nor mercury, nor antimony, nor alcohol, were necessary in favourable cases or could be depended on to save life in unfavourable ones. For many years past the treatment of pneumonia in England has been practically the same as that of typhus, enteric fever, or scarlatina—expectant, not in the sense of doing nothing for the patient, but of putting him in the best circumstances for recovery when the malady, which we cannot cut short, has run its course, and meantime of watching for any unfavourable symptom and meeting it as it appears.

Of late, however, it has been asserted by some German physicians that better results can be obtained by vigorous antipyretic treatment, like that which has been described in the chapter on enteric fever. At Basle cold baths have been used whenever the temperature rose a little above 102° Fahr. Jürgensen advises that 104° should be the point at which the baths should be systematically employed; while for patients whose temperature ranges at from 101° to 103°, he merely orders a tepid bath in the morning so as to increase the normal remission during the early part of the day. He also administers quinine in doses of thirty grains at intervals of forty-eight hours. The theoretical basis of this practice is that the great danger of pneumonia is failure of the heart, as the result of the combined action of pyrexia and of

* In Germany Skoda, then at the head of the Viennese school, had learned to distrust large and systematic depletion, although he still bled in the earlier stages of pneumonia, and believed in the power of drugs to defibrinate the blood and cut short the disease. The contrast between the treatment of pneumonia in his wards, as witnessed by the writer in the year 1865 and in those of Bouillard during the preceding winter, was remarkable. At the same time cases of pneumonia were being "cured" at the Homœopathic Hospital in Vienna by infinitesimal doses.

disturbance of the pulmonary circulation. In proof of its efficacy Jürgensen adduces a tabulated statement of the fatal cases that have occurred in his practice, showing that scarcely any of his patients died except such as had some dangerous complication. He makes a point of giving wine as a stimulant to the heart before each cold bath, and when signs of cardiac failure appear in the course of the disease he gives alcohol freely, as well as camphor and musk.

Few English physicians, however, believe that pneumonia can be jugulated by cold baths any more than by bleeding, or that moderate pyrexia is so injurious that it must be persistently attacked. Even if cold baths are harmless, there is reason to believe that antipyretic drugs are as liable to do mischief as the antiphlogistic treatment which they now supersede. A careful trial of antipyrrhin in a series of cases of acute pneumonia led Dr Botkin, of St Petersburg, to the candid avowal that it was doubtful if it ever did good and certain that it often did harm.

The present writer has more than once employed venesection in what seemed a suitable case without preventing death, has used cold baths to check pyrexia without doing any harm but with only doubtful effect on the course of the disease, has given aconite from the beginning without in the least altering the rise of temperature or averting serious complications, and has seen quinine, antimony, wine and brandy, all in turn prove useless to check the progress of the disease.

The non-specific and rationally expectant treatment of pneumonia, as generally carried on in hospitals and in private practice in London, is somewhat as follows.

On the first rigor and rise of temperature the patient is put to bed, kept undisturbed, and his strength husbanded. The worst cases in otherwise healthy subjects are those in which the patient has kept up during the first day of illness, or when some unhappy street outcast has walked about with the disease upon him until brought exhausted to a hospital.

The room must be kept comfortably warm. The patient must not talk and must not sit up in bed. A single calomel purge does no harm, and has the advantage of preventing the exhibition of physic afterwards, when the patient is less able to bear its effects. The diet should consist of beef-tea, milk, and arrowroot or gruel, with tea, fruit, jelly or ice if desired, and a free supply of water, effervescing drinks, or any harmless form of diluent. Nitre, citrate of potash, or acetate of ammonia are usually prescribed and probably are of real service as diuretics, and by promoting the solution and excretion of inflammatory products towards the close of the disease; but they need not be insisted on if (even when suitably disguised) they are refused by a child. If pleuritic pain is present it may often be relieved by poultices, a mustard plaster, or, according to some authorities, by a blister; but in really severe pain nothing gives so rapid and complete relief as a few leeches. In aged or feeble patients we must trust to hot applications and morphia injections. When cough and expectoration are difficult, ipecacuanha with paregoric and squills is indicated. If there is continued want of sleep, ten grains of Dover's powder is probably the most efficient remedy, and mere symptomatic albuminuria does not counter-indicate it. If, however, there is any doubt as to the efficiency of the kidneys, it is safer to give chloral hydrate or hyoscyamine.

If the temperature rises over 104° in the case of an adult, cold

sponging should be employed, and if this is ineffectual and delirium is present, Deiters' coils, or the wet pack are probably safer and better than the cold bath. Nevertheless, if the heart is sound and the patient young, a bath may wisely be used.

If cyanosis appears, with signs of dilatation of the right side of the heart—a small, frequent, and feeble pulse, epigastric pulsation, distended and pulsating jugular veins—the abstraction of six, eight, or even ten ounces of blood from the arm will often relieve the pressing symptoms and probably do permanent good. Wet or dry cupping between the shoulders is generally less effectual.

In the case of children, writes Dr Eustace Smith, where there is great dyspnœa and threatened cardiac failure from over-distention of the right side of the heart, life may often be saved by taking one, two, or more ounces of blood from the arm, according to the age of the patient. "I can look back," he continues, "to some fatal cases which I now believe might have been saved had I had the courage to relieve the labouring heart by judicious removal of blood.'

But the greatest danger in pneumonia is not from the fever nor from the pulmonary obstruction—it is from failure of the heart. To meet this, digitalis may be given with advantage, but it is less effectual than in cases of valvular disease. Ammonia, with or without senega, ether, and Hoffmann's anodyne (Sp. Ætheris Comp.), are all valuable drugs. But the most important means of meeting either an obstructed pulmonary circulation or a directly failing left ventricle is to use brandy frequently and in measured doses, but increased if necessary up to twenty ounces in the day. Children and young adults seldom need it, but there are few patients above forty, and probably none above fifty, who do not need stimulants in larger or smaller quantities from very early in the disease. Sometimes port wine or champagne suits better than brandy; the kind of stimulant may be decided by the patient's feelings, but the quantity and frequency must depend on the state of the pulse and the first sound of the heart.

Secondary forms of pneumonia.—We have seen how the course of pneumonia differs according to the age and habits of the patient, and according as the heart and the kidneys are healthy or the reverse. Another important distinction is between primary pneumonia, with or without complications, and pneumonia secondary to other diseases.

In *rheumatic fever* pneumonia is not very common, and does not appear to differ in anatomy from primary hepatisation; when it occurs it is a very grave complication.

In the course of *Bright's disease*, particularly in the parenchymatous and lardaceous forms, pneumonia is also a very fatal event.

In *disease of the heart* it commonly attends pulmonary hæmorrhage (or "apoplexy," as it has been called), a condition in which the air-vesicles are filled with blood, and a patch of lung becomes solid, dark red on section, and like damson cheese in appearance. Hepatisation often takes place around the wedge-shaped mass of solid lung, but it does not spread far, and the symptoms are not so severe as those of primary pneumonia.

In typhus, enterica, and other *fevers*, ordinary lobar pneumonia may occur (vol. i, pp. 140, 158, 181); but far more frequently a condition of the lungs is recognised during life, and verified after death, which has been called "typhoid pneumonia," or, more properly, "*hypostatic pneumonia with*

congestion." It affects, not the bases, but the backs of the lungs—the lowest part as the patient lies in bed; it almost always affects both lungs, more or less; it is not strictly bounded, nor continuous, but consists of patches of airless tissue, including several lobules, and surrounded by congested and œdematous but crepitant lung. It is not accompanied by the high temperature of primary pneumonia nor by its other striking characters, and yields imperfect resonance rather than dulness, toneless or subcrepitant râles rather than true pneumonic crepitation. When recognised, it shows prostration in general, and weakness of the cardiac muscle in particular, and calls for ammonia and for brandy.

Alcoholic pneumonia, or that which is found associated with delirium tremens, has been already mentioned.

Inflammatory œdema and acute pulmonary congestion.—Dropsy of the lung is not infrequent in the course of Bright's disease; as with œdema of the glottis, it is probably always in some degree inflammatory. But apart from these cases there is an œdematous inflammation of the lungs which comes on acutely and without previous disease. It has been described as acute pulmonary congestion. A case is narrated by Hertz in 'Ziemssen's Handbuch,' and also by Dr Leuf in the 'American Journal of Medical Science,' January, 1885.

Chronic lobar pneumonia.—We have seen that one peculiarity of acute pneumonia is that, when the patient recovers, the hepatised lung clears up completely, and the tissue retains no trace of injury from the severe perversion of structure it has passed through. But to this, as to every pathological rule, there are occasional exceptions. Abscess following pneumonia is so rare that its very existence is doubted (p. 131). Gangrene only ensues under special conditions, which will be discussed further on. Phthisis scarcely ever originates with acute lobar pneumonia.

But Addison described certain rare and exceptional cases in which, after an illness of a few weeks, with all the evidences of consolidation present, the patient dies, and the lung is found uniformly "albuminised." The section is not soft, lacerable, and granular, as in acute grey hepatisation, but smooth, solid, and tough. The alveoli are full of fibrillated lymph, and the exudation cells may have begun to be granular and fatty. It is a condition of the lung very liable to break down, and thus constitute a case of rapid phthisis; and, on the other hand, it is a condition from which recovery may take place, and the lung be completely restored.

Wilks and Moxon, after quoting this account, continue: "That there is a chronic pneumonia of such a kind can scarcely be denied when it is remembered for how long a time all the signs of consolidation may endure, and then a complete restoration take place. We must therefore believe that there is a true chronic pneumonia, whose origin is an ordinary inflammation and exudation into the alveoli, and whose appearance is best denoted by the term 'uniform albuminous induration.'"*

They add, however, that such chronic pneumonia may be of the lobular kind, and Addison himself said that this condition may be limited to one or a few lobules only; so that it may perhaps be doubted whether this "least frequent of the permanent pneumonic indurations of the lung" is not, after all, an unusually extensive catarrhal pneumonia.

* 'Pathological Anatomy,' p. 338. Addison's original description appeared in the 'Guy's Hospital Reports' for 1843, and will be found in the volume of his collected papers, published by the New Sydenham Society, p. 27.

Broncho-Pneumonia.*—In children acute bronchitis affecting the smaller bronchial tubes is often accompanied by inflammation of the pulmonary alveoli, usually of both lungs. And even in adults the same thing is sometimes observed, although very rarely. Among children it is especially at an early age that this is apt to occur. Ziemssen, out of ninety-eight cases, found that sixty-seven, and Steffen, out of seventy-two cases, found that fifty-two occurred in patients under three years old.

Ætiology.—In many instances the affection is secondary to some infective disease. Measles and whooping-cough furnish by far the largest number of cases of broncho-pneumonia. It is also sometimes seen after diphtheria, rubeola, smallpox, or scarlet fever. Jürgensen insists, as other writers had before, on the greatly increased liability to broncho-pneumonia, as a complication of bronchitis (whether after measles or independently of any infective disease) in those who are obliged to breathe impure air, in the close narrow rooms of the poor. He is even disposed to attribute the greater frequency of the affection in winter to defect of ventilation rather than directly to inclemency of season. It is probable that rickets renders a child more apt to become affected with broncho-pneumonia if it is attacked with bronchitis. Dr Wilks, in the 'Guy's Hospital Reports' for 1860, pointed out that broncho-pneumonia is a common cause of death in children (even up to puberty) suffering from severe burns.

As to the special causes of the exceptional cases when broncho-pneumonia like that of infants occurs in adults, it is at present difficult to say anything definite. The following are a few cases which occurred at Guy's Hospital. A woman, aged thirty, having miscarried eight days before, was attacked with shivering and headache, and died after an illness that lasted ten days. A man, aged twenty-five, of dissipated habits, who was said to have been under a course of mercurial treatment for syphilis, received a blow on the nose while he was drunken; this was followed by epistaxis, which continued until the nares were plugged; the mucous membrane then suppurated profusely, the discharge being very fœtid; and he sank and died at the end of a week. A third case was that of a man, aged twenty-eight, a singer at a music-hall. He had a fall from a cart, and this led to an illness which proved fatal in three weeks. At first he tried to go on with his singing, but he was soon obliged to give it up. When admitted, shortly before his death, he was already comatose. In this instance some of the patches showed central sloughs, but in the reports of the two other cases it is expressly stated that the appearances were identical with those that are commonly seen in children. It seems probable that in the second case the inflammation was set up by the inhalation of fœtid pus from the nasal cavities into the air-passages. Indeed, the report of another case, in which a similar affection of the lungs was met with as a complication of tubercular meningitis in a woman aged thirty-four, attributes broncho-pneumonia to the entrance of food into the bronchial tubes while she was struggling in her delirium.

Breathing irritant gases, especially chlorine in large quantity, sometimes sets up inflammation of the pulmonary tissue, simultaneously with an intense bronchitis. It is not improbable that the immediate cause of the ordinary broncho-pneumonia of children is often, if not always, the entrance into the

* *Syn.*—Catarrhal Pneumonia—Lobular Pneumonia—Pulmonary or Alveolar Catarrh.

alveoli of inflammatory products formed in the smaller tubes, as the result of violent inspiratory efforts.

It is also said that broncho-pneumonia is not infrequent in extreme old age, and that it often ends the life of those who have long laboured under senile bronchial catarrh.

Anatomy.—The most obvious indication that pneumonia in a given case has arisen by extension from the bronchial tubes is its occurring in scattered patches throughout the lungs. These patches are more or less rounded in form; they are usually of about the size of peas, but sometimes it is said they may be as large as hazel-nuts. They are reddish brown in colour, or more or less grey, or even greyish yellow, according to the stage to which the inflammation has advanced in them. As seen upon the cut surface of the lung, they appear slightly raised above the rest of the parenchyma. They have a dull, lustreless appearance, and are often somewhat granular on section, although no doubt the granulations are less obvious than in the red hepatisation of genuine acute pneumonia. The substance of the patches is soft and friable, and when squeezed they emit a more or less opaque fluid. It sometimes happens that they are so closely collected together in part of a lung that a whole lobe may seem to be consolidated. Even then, however, the lobular markings are usually very distinct and the cut surface is devoid of that homogeneous, even appearance which characterises common lobar hepatisation. Many lobules are found collapsed and others dilated.

More or less pleurisy is very commonly associated with broncho-pneumonia, there being a thin layer of lymph upon the pleural surface, especially over any patches that happen to lie just beneath the serous membrane. Indeed, both at the bedside and in the deadhouse it is a remarkable fact that, altogether apart from the presence of any obvious pneumonia, pleurisy is a far more frequent complication of bronchitis than one would have anticipated: in adults affected with bronchial inflammation a pleuritic friction sound may very often be detected, if it is listened for.

Histologically, the morbid process in broncho-pneumonia is a *catarrhal* inflammation; that is to say, the cells which fill the affected alveoli are many of them epithelial in character, large, of irregular shapes, with bold nuclei. Collapse of the alveoli does not prevent this condition.

In some cases the inflammation appears to spread to the connective tissue by which the pulmonary lobules are united together. Jürgensen speaks of "thick whitish-grey bands, which are seen crossing one another upon the cut surface of the organ." In 1878 a well-marked instance of this occurred in a child, aged three, who died after an illness of five weeks' duration, which perhaps began in whooping-cough. The left lung contained many patches of broncho-pneumonia. The right lung was almost entirely consolidated. Its tissue, however, felt hard; and the fibrous tissue in it had obviously undergone a great increase. Such cases seem to suggest an explanation of an appearance which one not infrequently sees in making autopsies of persons at all periods of life, namely, marbling of a part or the whole of a lung by fibrous bands which intersect one another and divide it up into irregular areas. Such an affection is commonly regarded as indicating early cirrhosis of the organ. But it has not at all the look of a progressive or active lesion; and it is far more likely to be a residue of a former attack of inflammation. Ordinary acute pneumonia appears not to cause this appearance.

Clinical course.—The recognition of broncho-pneumonia, in addition to

the acute bronchitis which precedes and gives origin to it, is usually more or less uncertain. Physical examination of the chest often helps but little. If several lobules side by side beneath the pleura are consolidated, there may be impairment of resonance (or even dulness) on percussion, bronchial breathing and bronchophony, the latter being especially obvious when the patient, if a child, is crying. Moreover, the bronchitic râles often become clear and "consonating" in quality. Jürgensen lays stress on the occurrence of a crepitating râle like that which is heard in ordinary acute pneumonia, but rather less fine, and audible during expiration as well as inspiration.

Nor, again, is broncho-pneumonia attended by very marked symptoms. Ziemssen has rightly laid stress on the importance of a sudden rise of temperature, as an indication of its supervention in the course of acute bronchitis, if the pyrexia should previously have been moderate. But, as already stated, the thermometer may indicate 104° or even higher in a child affected with bronchitis, independently of any complication (p. 99). Jürgensen says that broncho-pneumonia—as for example, after measles—may be accompanied with a temperature of 105·8° for days together, with scarcely any remissions. In fatal cases, the temperature sometimes rises before death, reaching perhaps 107° or 108°; sometimes it falls below normal. The pulse is often extremely rapid, so that it cannot be counted at the wrist. In children a pulse-rate of 150 to 200 is by no means incompatible with subsequent recovery; and one must be careful not to allow it to lead one to give an absolutely fatal prognosis. It is surprising, too, how hurried the breathing may be in cases which yet do perfectly well. What is really alarming is that the pulse should be feeble and "running" in character, from emptiness of the arteries. When the disease ends in recovery the pyrexia and the other symptoms subside gradually; there is never a critical fall of temperature.

Herpes on the lips seldom accompanies this affection. Albuminuria and diarrhœa are not infrequent. As regards what may be termed chest-symptoms, it often happens that with the supervention of broncho-pneumonia the cough from which the patient had been suffering ceases, or becomes less loud and shorter in character. Usually no expectoration makes its appearance. There is generally extreme restlessness, the child tossing about in bed, or requiring to be taken up by its nurse and shifted in position every few minutes. The breathing is often shallow. The face and the lips are apt to become pale, with perhaps a livid blush upon the cheeks. There is more dyspnœa and sense of distress than in cases of lobar pneumonia.

Diagnosis.—One point, of great clinical importance, is that broncho-pneumonia sometimes gives rise to cerebral symptoms of so prominent a character that one may easily suppose the case to be one of tubercular *meningitis.* Delirium, coma, retraction of the occiput, vomiting, strabismus, convulsions, may all appear in turn. Jürgensen makes the formal statement that, in a child affected with broncho-pneumonia it may be absolutely impossible to say with certainty whether or not tubercles are also present in the brain. Of course the question is then whether the pulmonary affection also is not really *acute tuberculosis.* Indeed, apart from nervous symptoms this is often difficult to decide.

Broncho-pneumonia in children may also be mistaken for *enteric fever.* Diarrhœa is frequent in broncho-pneumonia, and it as well as rose-spots are often absent in enterica; but a full abdomen and a swollen spleen point to the

latter, and the curve of temperature with its relation to the respiration will generally decide the question.

Prognosis.—The ordinary duration of broncho-pneumonia is about a fortnight; but sometimes it runs on for three or four weeks or even longer. It may destroy life very rapidly—within a few days, or even in twenty-four hours. Sometimes the cause of death seems to be marasmus, all acute symptoms having passed off. Jürgensen gives some figures from which it appears that the disease is fatal in from half to two thirds of the cases. When consecutive to measles, the risk is said to be smaller than this. Convulsions are of bad omen. Very young infants are far more likely to die than older children; and the prognosis is also more serious in those who are weakly, rachitic, or very fat. In children it is far more dangerous a disease than lobar pneumonia. Of forty-five cases of acute lobular broncho-pneumonia in children, recorded by Dr Goodhart, twenty were fatal. He notes the frequency with which, when one lung is inflamed in a child, the other shows signs of the same process here and there in a partial and often transient form.

Treatment.—Emetics do good service in the first few days; afterwards they are too depressing. Expectorants are useful, but opium must be avoided. The ammonia and senega mixture is almost always valuable; and, sweetened with glycerine or treacle or syrup of tolu, it will be taken by children as well as other medicines. Counter-irritation is often useful, and mustard poultices are well borne by the skin of even young children. Dr Eustace Smith recommends dry cupping of the back in bad cases. Diarrhœa should be checked by chalk powder. Stimulants are almost always necessary, and sweetened brandy and water or the egg and brandy mixture are the best ways to give them.

During convalescence, which is often long and tedious—contrasting with that of lobar pneumonia—quinine and iron are indicated, with cod-liver oil; and it is desirable to remove the child to the seaside as soon as practicable.

Vesicular pneumonia is a name used by Stokes, and applies to the first stage of broncho-pneumonia. Usually, in all probability, it soon becomes lobular, but occasionally it may be seen after death as a series of minute dots over the cut surface of a lung, too small for lobules, though too large for vesicles, and these are found to contain catarrhal products. But the same appearance to the naked eye may be produced by cross-section of small bronchial tubes, each surrounded with peribronchial inflammation.

When catarrhal or lobular broncho-pneumonia becomes chronic, the products are apt to undergo *caseous degeneration.* Instead of being removed by expectoration or by disintegration and lymphatic absorption, they remain and set up a spreading alveolar catarrh, which goes on to, but unfortunately rarely ends in, a local tubercular process. This is the ordinary beginning of phthisis, and will be discussed in a future chapter.

The term lobular pneumonia is also applied to the suppurative process which attacks the lungs in *pyæmia.* It is lobular in distribution, but its exudation is neither catarrhal, caseous, nor fibrinous, but purulent; and it never occurs except as part of a general process of embolism and septicæmia. The modern doctrine of pyæmia expounded in the chapter on that subject in our first volume was to a large extent worked out by Virchow and Cohnheim in the case of the pulmonary circulation. When the embolus is non-infective, it produces hæmorrhage by the same mechanism as in the

case of the brain; but there is no reason to suppose that this is the only cause of pulmonary apoplexy. Under extreme congestion from obstruction on the left side of the heart, the capillaries of the alveoli may give way; or extravasation may occur from changes in the vessels, or in the blood itself, as in cases of purpura hæmorrhagica. When the embolus is infective it produces intense congestion, and soon after suppurative inflammation. The abscesses (or earlier, only congested patches) are multiple, and are most numerous in the back part of the lung, and generally near the surface.

The symptoms of this form of lobular pneumonia are merged in those of the fatal disease of which it is part, but its presence may be suspected when a case of pyæmia is complicated by pain and other signs of pleurisy, which is caused by an abscess reaching the surface of the lung.

Cirrhosis of the Lung.*—This term was first used in 1838 by Sir Dominic Corrigan, for an affection of the pulmonary tissue, consisting in a replacement of its alveolar structure by a fibroid material, histologically analogous to that which in the liver causes the disease known as cirrhosis of that organ. Unfortunately, however, the mere presence of such a fibroid material in the lung is far from being characteristic of any one morbid process. The condition which has been termed "fibroid phthisis," is probably, in all but an insignificant minority of cases, nothing but an advanced stage, or a very chronic form, of a really tubercular process. There is reason to believe that the same is also true of those destructive affections of the lung which are set up by the inhalation of dust. This question will again come before us when we deal with tubercular phthisis and with coniosis of the lungs. Still there can be no doubt that exceptional instances are now and then met with which are not tubercular, and never have been. One peculiarity that characterises them is that whereas the whole of one lung may be shrunken into a hard grey mass, showing no trace of its proper structure, the other one is perfectly free from any similar morbid process; whereas in phthisis, long before one lung is completely destroyed, the other always becomes involved in the disease. Again, in these cases, when one part of a lung is affected before the rest, it is usually the lower lobe. In each of five cases observed in the *post-mortem* room of Guy's Hospital the bases of both lungs were found to have undergone cirrhosis; in not a single instance was there any indication of the presence of tubercle, nor were any caseous nodules or masses present.

Anatomy.—The process begins, according to some pathologists, in peribronchitis, which spreads from the smaller tubes to the elastic tissue surrounding the alveoli; or, according to others, in a chronic inflammation of the subpleural and interlobular connective tissue. Both origins, however, are denied by others, and Wilks and Moxon state decidedly that the process commences in the alveoli and their walls. When, although fibro-nucleated material occupies the pulmonary structures, there are also found cells and nuclei within the alveoli, they refer these cases to double chronic pneumonia. Of cirrhosis affecting one lung only, with thickened pleura and dilated tubes, the same authors say that sections of the lung show not only fibrous tissue surrounding the bronchial tubes and blood-vessels, but also thickening of the alveolar walls. This is well shown in a drawing

* *Synonyms.*—Chronic interstitial Pneumonia—Iron-grey induration of Addison—Induration ardoisée of Cruveilhier—Induration grise et mélanique of Andral.

given by Dr Coats (fig. 231), in which the alveoli are represented as lined with large nucleated epithelioid cells, quite unlike those of the normal lung; a condition also represented by a drawing by Heschl, reproduced in the late Dr Wilson Fox's article in 'Reynolds' System,' p. 765. When the fibroid degeneration has reached its full development, the lung on section exhibits large tracts of white, slate-coloured, black, or marbled iron-grey tissue, which is completely airless, firm, and sometimes of cartilaginous hardness. The microscope shows only dense fibrous tissue, with no trace remaining of the pulmonary structures which it has displaced.

Origin.—This chronic fibroid induration is an important conservative process in phthisis, few cases of which are entirely without some cicatricial induration; but as a morbid process independent of tubercle its pathology is still a matter of uncertainty; possibly it has different modes of origin in different cases.

(1) Now and again it seems to have begun in an attack of *pneumonia*. This question was already touched upon (p. 132, *ad calc.*), when the opinion of Dr Wilks was cited, that acute lobar inflammation of the lung, if it does not prove fatal, always undergoes resolution, and never leads to a chronic form of consolidation and induration of the pulmonary tissue. But, after all, the evidence on which this opinion was based must have been of a negative kind, consisting in the observation of a number of cases, in none of which such a result was noted. In hospital practice it can very rarely happen that a patient comes under the eye of the same physician during an acute illness, and again, months or years afterwards, when a chronic affection of the lung proves fatal. Moreover, medical literature contains several carefully recorded cases in which the origin of cirrhosis in pneumonia appears to have been clearly established. The two following are taken from Dr Bastian's collection of thirty cases, tabulated in the second volume of 'Reynolds' System.' The first occurs in Charcot's 'Thèse de Paris.' The patient, a man aged sixty-one, was admitted on March 30th, 1850. He had been attacked five days before with rigors and pain in the side, and had rusty sputa. There were all the signs of pneumonia affecting the whole of the right lung. These continued with but little alteration until his death on July 19th. At the autopsy the right lung was of a greyish-blue colour on section, as hard as cartilage, shrunken to two thirds its natural size, and enveloped in an immensely thickened fibrous mass. The other case was recorded by Dr Mayne ('Dublin Hosp. Gaz.,' May, 1857). It is that of a man, aged fifty-four, who in July, 1855, after a hard day's work, was seized with rigors and all the symptoms of pleuro-pneumonia. The acute disease subsided, but he never afterwards regained his health and strength. In October, 1856, he was attacked with fresh febrile symptoms, and he died at the end of the year. The lung on *post-mortem* examination was found affected with well-marked cirrhosis. Recently Dürr has recorded in a volume published by Jürgensen, at Tübingen, two cases, which occurred in very young children, but in each of which the primary attack appeared to be one of lobar, not catarrhal, pneumonia. Probably, however, the "pneumonia" which leads to cirrhosis, instead of being lobar, is as a rule lobular and catarrhal, secondary to measles or whooping-cough.

To this origin in alveolar catarrh the author was disposed to refer one variety of the affection, in which, instead of the tissue of any part of the lung being all converted into a fibrous substance, it is marbled or intersected by bands crossing one another, so as to split it up into areas

of various sizes and shapes. Such an appearance, which is not uncommonly seen in the *post-mortem* room, is not known to possess any clinical significance. It is generally supposed to represent an early stage of cirrhosis, such as might have advanced, if the patient had lived, to total destruction of the pulmonary texture. But may it not be rather a residue, or relic, complete in itself, of the former acute process?

Many pathologists who find difficulty in admitting the origin of pulmonary cirrhosis in true lobar pneumonia would readily allow the probability of so much more chronic, more irregular, and so to speak, more irritative a process as lobular or alveolar catarrh, being the antecedent of the iron-grey cirrhotic induration.

(2) A more frequent origin of this remarkable condition is possibly to be sought in *pleurisy*.

In most cases of cirrhosis, the pleura over the affected part of the lung is adherent to the parietal layer, and the two together form a dense white mass, of cartilaginous hardness, and from a quarter of an inch to an inch in thickness. The only way to remove the lung from the chest at the autopsy is to cut it out with the knife. There is also a similar thickening of the pleural layers separating the different lobes. This state of the serous membrane has led to the supposition that the affection may have begun as in an attack of pleurisy, and that the lung-substance may have been invaded from the surface. But there is reason to agree with Dr Wilson Fox in doubting whether it is possible for the whole of the interior of a lung to become cirrhosed in such a manner by extension along the interlobular septa. A case showing how easily erroneous conclusions may be drawn, even from *post-mortem* observations, occurred at Guy's Hospital in 1877. A man, aged twenty-seven, died of disease of the left side of the chest, with a history of having had inflammation of the lungs at the age of fifteen, and some thoracic affection even before that, in early childhood. There was found to be a localised empyema in front, and the pleura elsewhere was thickened in places to the extent of an inch. The lung was very small, but its tissue was generally healthy, except that it was intersected by fibrous bands. It would have been natural to infer that the organ had been invaded from without. But the apex of the other lung was affected in a precisely similar way, although the serous membrane covering it was in a normal state.

(3) *Dilatation of the bronchial tubes* in the affected lung or part of a lung, is present in most cases of cirrhosis.

So intimate, indeed, has been supposed to be the connection between these two morbid conditions, that Grainger Stewart and Jürgensen discussed them together. But various forms of bronchiectasis occur without there being any change in the pulmonary tissue, unless it be emphysema. And in no fewer than six of Dr Bastian's thirty cases of cirrhosis, it is expressly stated that the tubes were of their natural size. As to the relation between the bronchial dilatation and the affection of the lung, when they coexist, there are differences of opinion. Some think that a chronic inflammatory process may start from the tubes and lead to a gradual fibrous thickening of the alveolar walls with obliteration of their cavities, just as acute gangrenous pneumonia is well known to spread. But the very definite localisation of the morbid process, the completeness of the destruction of the pulmonary texture, and the fact that the pleura becomes so greatly thickened, form strong arguments against this view. By

Corrigan it was suggested that the occurrence of bronchiectasis was secondary to the cirrhosis; he imagined that the contraction of the adventitious fibrous material in the lung dragged upon the walls of the tubes, so as to widen their channels. Dr Wilson Fox, however, seems more likely to be right in thinking that if the dilatation of the tubes really follows the lung affection, it is caused by the expiratory force of a cough, exactly as it is in other circumstances: at an early stage of the disease it is reasonable to suppose that all the affected structures may be soft and yielding.

On the whole, it appears premature to accept either of the two views that would refer the origin of cirrhosis to a starting-point beyond the walls of the pulmonary alveoli themselves. Many of those cases which cannot be traced definitely to an antecedent acute attack of lung inflammation, may, after all, have arisen during an illness that occurred in childhood, so far back as to have been forgotten. And it seems by no means impossible that in others the morbid process may be chronic from its commencement, a true primary chronic inflammation of the lung.

Incidence.—Cirrhosis is most apt to occur in adult life. In Dr Bastian's list there were twenty-four male and six female patients.* At the present time (1887) we have a typical example in Mary Ward, in a boy seven years old, an unusually early age.

Symptoms.—Clinically the symptoms of cirrhosis vary according to its extent.

If it affects only a small portion of one lung, or even of both lungs, the patient becomes a chronic pulmonary invalid, with cough and dyspnœa; the expectoration is purulent, sometimes stained with blood, sometimes dirty-grey in colour and offensive; the fingers become clubbed, and there is more or less marked emaciation. The physical signs are those of consolidation of the pulmonary tissue, with the addition of more or less abundant râles, which may be consonating or even metallic in character. Such cases are not at all uncommon in hospital practice. But after staying a certain number of weeks in the ward, the end is that the patient is discharged unbenefited, or at best with some relief to his symptoms. In each of the five cases of cirrhosis of the bases of both lungs, above referred to as having been observed in the *post-mortem* room at Guy's Hospital, the cause of death was either quite unconnected with the pulmonary affection, or it was an attack of acute pneumonia, or lardaceous disease of the kidneys, the result of the protracted suppuration.

Very different is the course of cirrhosis, when it involves the whole of one lung. In that case the affected side of the chest falls in, so that it measures in each direction much less than the other side. There is often considerable difficulty in distinguishing the disease from a mere chronic pleurisy with retraction, or even from a malignant tumour producing a like condition. According to Dr Walshe, there is not in cirrhosis the same degree of twisting of the ribs on their axes as in pleurisy, nor is the shoulder lowered so much, nor is the inferior angle of the scapula tilted so far outwards. Great assistance in the diagnosis may be afforded by the discovery of râles on auscultation, and by the presence of an abundant purulent, and perhaps offensive, sputum. From malignant tumour cirrhosis is commonly distinguished by the state of the mediastinum. This, in the disease now under consideration, is dragged out to the farthest possible extent. If the right

* One was only 7, eleven were between 16 and 20, twenty-one between 24 and 57, and five between 61 and 71.

lung is cirrhosed, the heart is seen beating at the right nipple, if the left, its visible pulsation may extend upward nearly to the left clavicle. In either case the opposite lung undergoes an extreme degree of enlargement, so that the whole sternal region, and even the space beneath the costal cartilages on the affected side, becomes resonant on percussion, and transmits to the stethoscope a loud vesicular murmur. After a time, however, this over-distended and perhaps hypertrophied lung fails to carry on the respiratory function efficiently. The right side of the heart becomes dilated, a tricuspid regurgitant murmur develops itself at the ensiform cartilage, the patient suffers from permanent orthopnœa, the liver becomes nutmegged, and ascites and œdema of the lower limbs set in. The case in fact assumes all the characters of chronic valvular disease of the heart with dropsy, and terminates fatally in the same way.

About the *treatment* of cirrhosis of the lung, all that need be said is that for the relief of the different symptoms such remedies must be used as are recommended for the like symptoms in other diseases, such as phthisis, bronchitis and bronchiectasis, which are more commonly met with in practice.

Syphilitic Disease of the Lungs.—Only within the last twenty years has it been recognised that other viscera than the testis, and other regions than those accessible to the surgeon's touch, may be seats of syphilitic disease. It is now certain that (quite apart from the question of a form of phthisis of syphilitic origin, the existence of which will be discussed hereafter) there is a form of chronic interstitial pneumonia, or cirrhosis of the lungs, characterised by deep scarring and contraction of the tissue, by peribronchitis and tracheitis, often with ulceration and deformity, and by the presence of typical gummata. The process affects the roots or bases rather than the apices; it often starts from previous ulceration of the air-passages; and it is apt to lead to gangrene.

The disease is a rare one, and would be little but a pathological curiosity were it not for the importance of recognising the true pathology of these cases in order to treat them successfully.

Lancereaux described cases of this kind in France ('Traité de la Syphilis,' 1866); Dittrich and Virchow in Germany ('Kr. Geschw.,' ii); and Wilks in this country ('Path. Trans.,' vol. ix, 1858, with plate, and 'Guy's Hosp. Reports' for 1863, 3rd series, vol. ix, p. 33, two cases).

In a series of twenty-two cases of visceral syphilis brought before the Pathological Society in 1877, Dr Greenfield described three of presumably syphilitic affection of the lungs (vol. xxviii, p. 258). In the same volume is the description of a specimen shown by Dr Sutton of chronic syphilitic pneumonia, from a patient under Dr Gull and Mr Durham in Guy's Hospital (p. 304); three of "fibroid phthisis" in syphilitic patients, by Dr Goodhart, with a histological drawing in which peribronchitis is clearly shown (p. 313, and abstracts of nineteen cases from Guy's Hospital, p. 322); one apparently of gummata coalesced into a large mass in one lung, by Dr Gowers, with a histological drawing (p. 330); one by Dr T. H. Green of a similar large mass in one lung of a man with "undoubtedly syphilitic lesions in his liver;" two by the late Dr Mahomed, both in women with undoubted syphilitic disease, but the pulmonary lesion less certainly of the same origin and perhaps in an early stage; lastly, three of gummata in the lung from the museum at Netley, by Dr Aitken.

The following cases have come under the writer's notice :—(1) A woman of about forty at the Hôpital Beaujon in Paris. She was wasted, with cough, purulent expectoration, and hæmoptysis; and from other proofs of lues was treated with perchloride of mercury to her great benefit. (2) A groom of about thirty who, together with signs of chronic pneumonia and hæmoptysis, had nodes on the tibia and amygdaloid lymph-glands. He improved greatly under iodide of potassium and mercury, with which, however, it is right to add, cod-liver oil was given, and when last seen had gained weight, was free from pain, and able to resume work.* (3) A strong and muscular seafaring man about forty, who had symptoms and physical signs resembling phthisis, but was well nourished and had a good appetite. A gumma was discovered near the hip-joint; he was put on iodide of potassium, and under this treatment not only did the node disappear, but his cough and other pulmonary symptoms ceased, and he was to all appearances cured. (4) A patient seen with Dr Miller, of Norwood, who had certainly suffered from syphilis, and in whom there were physical signs of very local consolidation in one lung, together with tracheo-laryngeal ulceration and hæmoptysis. He was treated with mercury, but died from stenosis of the air-passages before much good could be effected. An autopsy was obtained, and beside deformity and contraction of the trachea and bronchi, there were several gummata in the right lung with scarring and fibrous induration. (5) A patient in Guy's Hospital, aged forty-two, with signs of chronic disease of the lungs, which had been called consumption, and with no history or signs of past syphilitic lesions. The nature of the case was not recognised until after the man's death from eclampsia. Previous to this severe caries of one ulna had led to such extensive suppuration that the arm was amputated. At the autopsy were found caries of the frontal bone with pachymeningitis, fibroid testes, and two small fibroid patches in the left ventricle. The two primary bronchi were contracted aud deformed. The right lung contained a single fibroid nodule in the lower lobe ; the left was solid in patches, firm and grey, with dilated tubes. There was nothing that could be called tubercular in either lung, the larynx and ileum were healthy, and there were no miliary tubercles anywhere to be found ('Path. Trans.,' 1877). Microscopical examination of the indurated tissue in the lung showed it to consist of a fibro-nuclear growth with numerous vessels. (6) A sailor aged forty-four, in Guy's Hospital, February, 1876. Signs of chronic phthisis with hæmoptysis; chancre but no history of secondary lesions; intense dyspnœa evidently from obstruction to entrance of air; larynx perfectly normal; no sign of aneurysm or thoracic tumour pressing on trachea. Tracheotomy was therefore not performed, and the patient died six days after admission. There was found after death ulceration with stenosis of the trachea, "fibroid phthisis" with one old vomica and much puckering and cicatrisation. Scars on the surface of the liver but no actual gumma. Liver and kidneys lardaceous.

Between syphilis pulmonum and chronic interstitial pneumonia (so-called fibroid phthisis, or cirrhosis of the lungs) there is no anatomical distinction in the absence of gummata. But we have two unfailing criteria, one in the associated changes in other viscera, the other in reaction to treatment.

Syphilis is no protection from true phthisis, and what has been called syphilitic phthisis is in most cases nothing more than true tubercular phthisis

* This case resembled one recorded by Dr Walshe (p. 513 of the 4th edition of his work on the Lungs).

in a syphilitic subject, which runs its course uninfluenced by the latter disease. But there is also a form of chronic pneumonia, with fibroid induration and bronchiectasis, with irregular local distribution, with no tubercle and little or no caseation, which starts either from gummata or from thickened patches of pleura, or from a chronic contracting peribronchitis. This peribronchitis is associated with an ulcerative inflammation of the trachea or bronchi, or both, which is closely related to the ordinary syphilitic inflammation of the larynx. The symptoms during life are indistinguishable from those of ordinary phthisis, though the physical signs point to a more chronic and fibroid, less acute and caseous, form of disease. If the physical signs are confined to one lung or absent from the apices, one may suspect the true nature of the case; but it is only by concomitant lesions of other organs and by the effect of treatment that we can establish the diagnosis during life. Hæmoptysis is often a striking feature. Dr W. H. Porter, of New York, mentions tenderness of the tibiæ and sternum on pressure as a symptom of value. The absence of the bacillus tuberculosis, if found to be constant, will be a most important diagnostic sign.*

Hereditary syphilis.—It is quite possible that some of the cases of gummata with cicatrices and chronic induration just described may be due not to acquired but to congenital syphilis.

But there is another form of pulmonary disease which appears to occur only in children who show signs of hereditary lues. It is uniform, without gummata or cicatrices, and with no primary lesion of the trachea and bronchi, or of the pleura. Lungs in this condition have been described by Wagner and Virchow in Germany, by Robin in France,† and by Wilks and Moxon in this country, as a diffused form of hepatisation, firm, dense, or even tough in texture, white in colour, and airless. They are sometimes both affected, more often one, either throughout a lobe or in smaller circumscribed masses. The more universal cases are, as might be supposed, found in stillborn children, or in those who only survive birth a few days. The condition seems to be essentially chronic thickening of the alveolar walls and of the interlobular septa, whereby the alveoli are compressed, and the part affected rendered heavy, bulky, and more or less completely solid. On certain points there is conflicting evidence. Some writers describe the diseased patches or lobes as quite exsanguine with obliterated capillaries; others speak of free production of new vessels, so that the new growth is very vascular. Dr Greenfield described the lung of a presumably syphilitic child, twelve months old, in the 'Pathological Transactions' for 1876 (xxvii, p. 43). It was tough, yellowish white in colour, with a smooth (not granular) section, and exuded very scanty fluid. Histological drawings are given which show bands of fibrous tissue obliterating the alveoli, of which the walls are remarkably thickened, but the endothelium is unaltered. There was unfortunately no positive proof of syphilis in this case, but Dr Goodhart mentions a specimen ('Diseases of Children,' chap. xxii) in which similar *post-mortem* appearances were found in a child three months old, together with undoubted syphilis of the liver. Micro-

* Beside the papers quoted above the following may be mentioned:—Dr Porter ('New York Med. Journ.,' July, 1885), with plate; Dr Moxon ('Guy's Hosp. Rep.,' 3rd series, vol. xiii, p. 374); Dr Goodhart (ibid., vol. xxv, p. 31); the late Dr Wilson Fox's article in 'Reynolds' System' (no cases given of his own); and Dr Bäumler's article in 'Ziemssen's Handbuch.'

† Lorain and Robin call it "epithelioma" of the lungs.

scopically, it showed all the features exactly as described by Dr Greenfield—excessive fibro-nucleated growth, extreme vascularity, and collapse of the air-vesicles.

The symptoms observed during life appear to be remarkably inconspicuous.

GANGRENE OF THE LUNG.—This affection bears out the statement made with regard to gangrene in general (vol. i, p. 63), namely, that the death of any part of the human body is always either the result of an intense inflammation, or else of an injury which, if a little less severe, would have set up inflammation, but which by its violence kills the tissues outright before there is time for inflammation to occur.

Anatomy.—Of pulmonary gangrene without antecedent pneumonia it would be difficult to find unequivocal examples. But in the *post-mortem* room cases are sometimes seen in which there has been rapid and extensive sloughing of a portion of a lung, and in which no zone of hepatised tissue separates the gangrenous part from that which is healthy or merely œdematous. In such cases the fact of there having been inflammation is, at any rate, unproven. Generally, however, the sloughing mass lies within a more or less broad area of consolidated lung-substance, of which it had evidently at one time formed a part. Should the disease have proved fatal at an early stage, one condition may gradually merge into the other; should it have reached a more advanced stage, there may be a well-marked line of demarcation, or the dead tissue may have been cast off with the formation of a cavity. Actual experience in the deadhouse does not appear to support the distinction between two separate forms of pulmonary gangrene, the one "circumscribed," the other "diffuse," which has been taught since the time of Laennec; for he was the first to recognise gangrene of the lungs as a special affection.

Little more need be said of the anatomical characters than that the diseased part is of a dirty greenish brown or black colour, and so soft as to be sometimes almost diffluent. It is often horribly fœtid, but occasionally the odour has been little marked, as Cruveilhier long ago noticed. Microscopically, the pulmonary structure is hardly to be recognised, the alveolar walls having broken down into a mere granular débris.

Origin.—Pneumonia (*i. e.* the acute disease which causes lobar hepatisation) rarely leads to gangrene, except in very old and feeble persons, in drunkards, and in those who are exhausted by some other malady, for example, by diabetes. But one or two instances have occurred in Guy's Hospital, of what had appeared to be an ordinary attack of pneumonia in a healthy subject which when the acute stage subsided, was followed by symptoms which seemed to indicate that at least some of the hepatised tissues had undergone sloughing. Dr Walshe records just such a case—that of a man who was slowly recovering from an attack of pneumonia of the right lower lobe when hæmoptysis set in, and was followed by the copious expectoration of a frothy, intensely fœtid sputum, while at the same time physical signs like those which indicate the formation of a cavity made their appearance. Ultimately this patient left the hospital in fair general health, and free from all physical signs except those commonly denoting consolidation.

Another pulmonary disease, in the course of which gangrene may occur, is phthisis. Dr Walshe speaks of having seen some six cases in which the special fœtor appeared incidentally in connection with *tuberculous vomicæ* already formed.

But in the majority of cases gangrene of the lung arises out of an inflammatory process of *septic* character. There are various ways in which such a process may be set up. Sometimes it is by direct extension from neighbouring parts, as when an ulcerating cancerous growth in the œsophagus eats its way into the lung, or when perforation occurs from a suppurating hydatid or from simple abscess of the liver, or from suppuration spreading from an ulcer of the stomach, or even from a putrescent empyema. Sometimes the infection is brought by the blood-vessels, as when septic emboli become lodged in branches of the pulmonary artery, having been derived from a cerebral sinus, diseased as the result of some affection of the ear, or from a systemic vein in the neighbourhood of a part involved in an unhealthy inflammation, or even from the right side of the heart in a case of ulcerative endocarditis. In yet other cases the disease starts from the bronchial tubes. A foreign body, as a piece of bone impacted in one of the bronchi, is very apt to set up a sloughing pneumonia; or it may even be caused by the entrance into the air-passages of pulpy or liquid food, as in patients who have chronic laryngeal disease, or in those who are comatose from apoplexy, or in lunatics who have to be fed by force. A like result may be produced by matters from the stomach drawn into the lungs during the act of vomiting, especially in persons rendered insensible by anæsthetics; we have had instances of death brought about in this way in cases of hernia or of intestinal obstruction, in which there had been a copious discharge upwards of the contents of the small intestine. Again, putrid materials that pass into the air-passages may be originally derived from the mouth, as in cases of gangrene of the cheek or of the tonsils, of diphtheria of the fauces, or of sloughing cancer of the tongue; Volkmann has suggested that sometimes a severe disease of the ear leads to pulmonary gangrene as the result of the dropping of morbid secretions down through the Eustachian tubes into the pharynx, and not (as is more usual) through the occurrence of thrombosis and embolism. Lastly, there are cases in which a sloughing pneumonia is due, not to the entrance into the bronchi of matters from without, but to the decomposition of retained secretion or inflammatory exudation, which had been formed within their channels.

Sputum.—This leads to a question on which few pathologists seem to have clear views. In fœtid bronchitis, as we have seen (p. 119), the patient's breath and his expectoration may have either the true odour of gangrene, dependent upon the decomposition of dead tissues, or a peculiar nauseous acrid odour, which is sometimes not unlike that of fæcal matter, and which appears to be due to the presence in it of free fatty acids.

In the latter case the sputum presents the further peculiarity of separating into three layers, in the lowest of which are found certain masses or "plugs," consisting of exudation that had accumulated in the dilated tubes, and undergone chemical and other changes there. Now it seems to be assumed that the same description applies also to other forms of sloughing of the lung. But surely this is a complete mistake. Unless there is an antecedent bronchiectasis, the peculiar "plugs" cannot be formed, nor is there any reason why fatty acids should be set free. And the odour in all other cases is simply that of gangrene—an indescribable fœtor, but one which is always of the same character, though it varies greatly in intensity, being sometimes only just perceptible, sometimes so strong as to poison the whole air of a room. As a rule, the patient's breath has the same smell as the expectoration, especially after coughing; and it now and then happens that

the breath is characteristically offensive for some days, while the sputum remains odourless. But there are a good many cases in which during life no fœtor is discoverable, either in the breath or in the expectoration, so that the presence of gangrene may not be suspected until it is seen at the autopsy. Hertz (in 'Ziemssen's Handbuch') accounts for this fact by assuming that the tubes passing from the sloughing parts are obstructed by secretion. But judging from experience in the dead-house, it would seem that the absence of fœtor before death occurs, especially in cases in which the gangrene is acute, which would be commonly described as belonging to the "diffuse" variety of the affection, and in which therefore it is most unlikely that obstruction of tubes would be present.

The fœtid sputum of gangrene of the lung is commonly of a dirty grey or greenish colour; sometimes it is brown or almost black, from the presence of altered blood. The microscope does not often lead to the detection in it of recognisable fragments of pulmonary tissue, although such fragments are so often to be found in ordinary cases of phthisis. Obvious hæmoptysis is said to occur comparatively seldom in adults, but frequently in children. Fatal hæmorrhage from this cause, due to the erosion of a large vessel, is an event of great rarity. When the surface of the lung is affected the pulmonary pleura usually gives way, leading to the formation of pneumothorax, which is quickly followed by septic pleurisy. If there should happen to be local closure of the serous cavity by adhesions, it is said that a subcutaneous emphysema may develop itself, or that an abscess may form, which may open externally after burrowing to a greater or less distance. Another occasional effect of the presence of a patch of gangrene in the lung is said to be the dropping of putrid matters into tubes belonging to other parts of the organ, so as to set up sloughing in them also. In this way, according to Hertz, it often happens that the diffuse form of the affection arises out of the circumscribed.

The only *physical signs* that can be said to belong to gangrene of the lung are such as serve to indicate the formation of a cavity in the organ at a spot where the tissue previously was either healthy or simply consolidated. But it can be only in very rare cases that such signs are to be definitely made out. They would include amphoric breathing, consonating or even metallic râles, and loud bronchophony. It is important to remember that where there is a possibility of the presence of phthisis the mere detection of a cavity proves nothing as to the exact seat of the sloughing process, unless it is known that no vomica existed at the same spot before the fœtid expectoration began.

The general *symptoms* that accompany gangrene of the lung are often very severe, but it does not seem that they point to the presence of this affection so definitely as might be supposed from the statements made by most writers on the subject. It is said, for example, that the pulse is small, feeble, and very frequent, and that the pyrexia quickly passes into an adynamic form, with great prostration of the vital powers. That absorption of putrid matters into the blood from the lung should produce such results is, indeed, to be expected, but when the characteristic fœtor is absent it surely is not possible for anyone, from the intensity of the general symptoms alone, to suspect that sloughing of the lung is taking place. In pneumonia the patient commonly falls into a similar condition before death, even when the affection has not advanced beyond the stage of grey hepatisation. And of many other diseases that may lead to the occurrence of

pulmonary gangrene, the same thing may be said apart altogether from their being ever attended with any pulmonary complication. Nor does it appear that when the sloughing part is very limited in extent the nature of the morbid process is commonly to be inferred from the fact that the constitutional symptoms are disproportionately severe. The truth rather is that in such cases the patient's general condition often remains for several weeks much better than might have been anticipated. Hertz, indeed, speaks of anorexia and gastric disorder, and even diarrhœa, as being caused by the swallowing of some of the offensive material expectorated from the lung, but I suspect that this would be rather difficult of proof.

Prognosis.—It is only when the gangrene is limited to a small part of the lung that recovery is possible. How minute a slough may cause fœtor is well shown by one of the cases of phthisis complicated with gangrene observed by Dr Walshe; in that instance the expectoration of a pea-like mass brought the fœtor to an end. Unfortunately he does not say for how long a time it had been present. When a case of gangrene is about to end favourably, the separation of the dead tissue is doubtless followed by the formation of a lining membrane to the cavity left by it, and perhaps this may ultimately shrink and become converted into a fibrous cicatrix.

Treatment.—It is an important point to diminish the fœtor as far as possible, and this applies also to cases of fœtid bronchitis. The most effectual means of attaining the object aimed at is by inhalations of oil of turpentine, carbolic acid, *oleum cadinum*, or eucalyptol. Turpentine inhalations were used by Skoda about thirty years ago. His plan was to pour a teaspoonful or two of oil of turpentine upon the surface of some boiling water, and to let the patient draw the vapour into the lungs. A much better method is to use a Siegel's spray apparatus, so as to atomise a liquid containing from five to two parts of carbolic acid in 100 parts of water, or of a solution of common salt. The inhalations may be repeated two or three times a day. Care must be taken that there is not enough absorbed to set up headache or giddiness, or to give the urine a brown or black colour. The *oleum cadinum*, or the eucalyptol, may be directly inspired from a sponge placed in an "ori-nasal respirator." The effect of such inhalations is sometimes very striking, especially in cases of fœtid bronchitis.

It is needless to say that the strength of the patient must be maintained by good and abundant food, and that the administration of stimulants in large doses is sometimes necessary. Ammonia, camphor, ether, quinine, and the tincture of perchloride of iron, may each in turn do good service. Oil of turpentine may also be administered by the mouth in doses of twenty or thirty drops, either beaten up with the yolk of an egg, or made into an emulsion with tragacanth or tincture of quillaia.

PLEURISY

Dry pleurisy—Its physical signs—Pleuritic effusion—Compressed and carnified lung—Dulness—Ægophony—Symptoms—Course and events—Empyema—Diagnosis—Complications—Ætiology and relation to tubercle—Prognosis—Treatment of pleurisy and of empyema—Paracentesis and its results.

Pleurisy or pleuritis* was mentioned by Hippocrates, and was described by other ancient Greek writers, who undoubtedly were referring to cases of the same disease to which we now apply that name. Nevertheless, it is only during the present century that its real nature has been known—an inflammation of the two surfaces of the serous membrane which lines the space surrounding the lung, while that organ itself is unaffected, unless pneumonia is also present. For, until percussion and auscultation were discovered, there was no possibility of drawing valid distinctions between the two diseases in clinical practice.

Anatomically, inflammation of the pleura resembles the inflammations of other serous membranes very closely. The earliest morbid appearance is generally said to be an injection of the smaller blood-vessels and perhaps the formation of ecchymoses, and no doubt hyperæmia is really present at the commencement of very severe pleurisy. But, as a matter of fact, one often finds patches of recent lymph upon the surface of a lung without any reddening, when an inflammation of no great intensity has set in shortly before death, in a patient suffering from some other disease; and ecchymoses by themselves indicate, not an early stage of pleurisy, but either that there has been obstruction of some part of the air-passages, causing a great impediment to the entrance of air into the lung, or else that there has been septicæmic or pyæmic infection of the blood. In many instances the effusion of lymph upon the opposed surfaces of the serous membrane occurs only at the very commencement of an attack of pleurisy, of which it may be said to constitute a "plastic stage" or "period." But in others it forms the whole of the disease. The case is then said to be one of *dry pleurisy.* When the inflammation afterwards subsides, the morbid material sometimes undergoes complete absorption, leaving the pleura in its natural condition, or slightly dull and opaque. But very often the two surfaces have in the meantime adhered together, and remain henceforth connected by separate bands or by a uniform layer formed of connective or fibrous tissue, which may have a free supply of blood-vessels.

Signs of dry pleurisy.—There is an auscultatory sign which, when it can be heard, is of itself almost conclusive as to the presence of lymph. This is the "friction-sound" or "pleuritic rub," due to the movement upon one another of the two roughened serous surfaces. One cannot but be interested to know that Hippocrates described the pleura as "creaking like leather,"

* Πλευρῖτις sc. *νοσός*, the side-complaint, the stitch in the ribs. The adjectival termination, *ιτις*, has been taken from this word and from *φρενῖτις* to denote inflammation, and has thus been used to form Peritonitis, Nephritis, Orchitis, and so on.

for such a phrase is often exactly applicable to the sound which is conveyed to the ear by the stethoscope. Yet Laennec, though he must have often heard this sound, failed to understand its meaning, and left to Reynaud the credit of rightly interpreting it. Laennec's idea about it was that it indicated emphysema, especially what he described as interlobular emphysema. And we shall presently see that there are still some observers who think that a precisely similar sound may sometimes be due to that cause, or, again, to miliary tuberculosis of the pulmonary surface. On the other hand, Dr Walshe has expressed the opinion that a friction-sound of the most marked character may be heard when there is no lymph whatever, if the serous membrane is rough from "simple vascularity." But this conclusion appears to be hardly warranted by the case from which he drew it. That case was one in which death occurred sixteen days after a rub had been heard, whereupon fluid was found in the pleural space, but no plastic exudation. But how is one to know that lymph may not have existed previously, and have undergone disintegration, or absorption by leucocytes?

It is difficult to describe in words the characters of a *pleuritic rub*: one must hear it to appreciate them. In its most typical form it consists of an irregular succession of short, harsh sounds, which give one exactly the impression of something catching or dragging against an obstruction and then slipping, but only to catch or drag once more. The patient himself is often conscious of a rough grating sensation each time he breathes; and one may be able to *feel* the rub quite plainly by placing one's hand over the affected part of the chest. Sometimes a rub accompanies both inspiration and expiration. Sometimes it is to be detected only at the very end of a deep inspiration, when the lungs are just becoming expanded to the greatest possible degree. It may be heard within twelve hours of the commencement of the disease, and in cases of dry pleurisy it may persist for days or even weeks with but little alteration. But it much more often disappears after a short time, because fluid effusion is formed which keeps the two surfaces apart. Or, if the inflammation should be spreading, it may, after it has ceased to be audible at one spot, be for the first time discoverable at another. A rub is not often present over a large area at once. The part of the chest at which one is most likely to hear it is in the axilla, or below the nipple, or in the back below the scapula. The reason is not only that pleurisy more frequently affects the surface of the lower lobe than that of the upper, but also that the descent of the diaphragm causes an actual movement of the pulmonary upon the costal pleura, which is wanting elsewhere. Sometimes, however, a rub can be heard over the front of the chest as high as the clavicle. In some cases a sound which is really due to pleural friction possesses characters so indefinite that one cannot distinguish it from moist or dry sounds seated in the bronchial tubes. This has long been taught at Guy's Hospital. In his well-known paper on the "Difficulties and Fallacies attending Physical Diagnosis," Addison cited a case of Dr Barlow's, in which lymph upon the adjacent surfaces of the liver and of the parietal peritoneum caused a "crepitus, which closely resembled a mucous rattle" ('Collected Works, p. 87). It is hardly possible to obtain positive proof of the fact, so far as regards the pleura itself, because, even if an autopsy shows lymph upon the serous membrane, one cannot be quite certain that an adjacent tube may not during life have contained fluid secretion.

The only other physical signs of the plastic stage of pleurisy are a certain degree of impairment of movement of the affected side of the chest and a corresponding enfeeblement of the respiratory murmur.

Pleuritic effusion.—In most cases of pleurisy liquid is effused into the serous cavity, often in large quantity and with great rapidity. Two or three quarts are not uncommonly found, and Watson cites a case in which Crampton, of Dublin, drew off from the left pleura as much as fourteen imperial pints. The liquid is sometimes translucent and of a yellowish colour, perhaps containing shreds and floating masses of fibrin. It has an alkaline reaction; according to Fräntzel, who writes on pleurisy in 'Ziemssen's Handbuch,' its specific gravity may vary within wide limits, from 1005 to 1030. But in other instances it is more or less opaque, and on standing throws down a layer of greenish-yellow pus. Or it may be altogether purulent; in which case its reaction to test-paper is acid. This constitutes what is termed an *empyema.** As a rule, no doubt, the formation of pus in the pleural cavity is a gradual process, the liquid being at first serous or sero-purulent, and becoming more and more opaque as the leucocytes in it increase in numbers. In such cases both the parietal and the pulmonary surfaces may still remain coated with more or less thick layers of fibrin. But when the inflammation is from the first of an exceptionally violent character —as, for instance, when it is set up by the entrance of putrid matter from without—a primary suppuration may occur, and the serous membrane may remain as smooth and shining as the peritoneum under similar circumstances.

Sometimes a pleuritic effusion is of a deep brown or purple colour, from admixture of blood. This may be due to the fact that the patient has scurvy; or it may depend upon the presence of tubercles, or of cancer. According to Fräntzel, "a hæmorrhagic tubercular pleurisy" is less rare in persons advanced in years than in those who are younger; but the only example of it that has been lately met at Guy's Hospital was in a man aged thirty-five. The writer just quoted also hints at cases, comparable with hæmorrhagic pachymeningitis, in which after connective tissue has already been formed as the result of a pleurisy, fresh inflammation is lighted up attended with extravasation of blood; but he does not say that he has actually met with such an instance. The author once removed a quarter of a pint of liquid of a dark brown colour from a patient who had pleurisy as a sequela of scarlet fever. He rapidly recovered.

Liquid pleuritic effusion usually gravitates into the most dependent part of the serous cavity, whatever may have been its starting-point. Thus, at the commencement of the disease, lymph may have covered the front and side of the lung, but when serum or sero-purulent fluid is poured out, it falls into the back and lower part of the chest if the patient is in bed, or it accumulates above the diaphragm if he is not recumbent. But this rule is liable to exceptions, when portions of the lung have previously become fixed to the chest wall as the result of a former attack of pleurisy, or even by the rapid development of adhesions at the commencement of the illness which is still in progress. Thus a considerable quantity of liquid may

* In England the meaning attached to the word *empyema* is, that of a collection of pus in the pleural cavity. But on the Continent, in spite of etymology, collections of serous fluid, and even of blood, are included under the same name (see Littré and Robin's 'Dictionary'). It has also been used as a synonym for thoracocentesis, so that when the pus escapes through an intercostal space, and has to be let out with the knife, an *empyema necessitatis* is said to arise.

accumulate somewhere towards the upper part of the pleural sac, or between the lung and the pericardium, or between two lobes of the lung itself, without there being any in the usual position at the base. And even when the whole of the serous membrane is affected the seat of a serous or purulent effusion may be more or less irregularly circumscribed. Between 1873 and 1876 four instances of this occurred in our *post-mortem* room. In one case there was a broad adhesion to the lateral region of the chest and another to the diaphragm, so that the liquid filled the upper part of the pleural cavity while crepitant lung tissue still existed in the lower part. In another case there was liquid at the base behind, and also above the root of the lung, with an intervening zone where the lung was firmly adherent. But the limitation of pleuritic effusion by adhesions is seldom so complete as to lead to the formation of two or more collections of fluid entirely cut off from one another; generally speaking, they communicate freely, as can be shown at an autopsy by passing a bent probe behind the bands of adhesion from space to space.

A necessary consequence of the presence of liquid in the pleural sac is that the lung becomes reduced in size. It is common to speak of the organ as being *compressed* by effusion, but this is a very inadequate way of stating the case, as must now be shown. Every physiologist will admit that the elasticity of the pulmonary tissue must lead it at once to recede as soon as there is anything to take its place, until it has become collapsed to at least the same extent as when air is admitted into the serous cavity after death. But, further, Lichtheim has proved by certain experiments, quoted above in the chapter on bronchitis (p. 102), that the elasticity of the lung does not become exhausted until the alveoli are completely emptied of all their gaseous contents. The reason why a lung is not rendered altogether airless when the pleural sac is laid open in the dead body seems to be mainly that the walls of the tubes presently fall together and offer a resistance to the further escape of air, which the elasticity of the pulmonary tissue is unable to overcome. And during life an additional force is in operation to empty the alveoli of air, namely, absorption by the blood-current circulating in their walls. Consequently it is not enough to say that a lung undergoes compression by pleural fluid until it is absolutely free from air alone; beyond this point it undoubtedly may become compressed until the blood is also driven out of its substance. It appears advisable to mark this distinction by separate names, and a lung which is bloodless as well as airless may be said to be *carnified*, whereas a lung which is merely airless may be spoken of as *collapsed*. Both terms have long been in use, but not with the precise shades of meaning here assigned to them. A carnified lung has a very peculiar appearance; it has a kind of slaty-grey colour and is best described as being mouse coloured. Its cut surface is smooth, very firm, and dry, showing the flattened orifices of air-tubes and vessels closely packed one against another. Sometimes, however, the bronchial tubes in it contain pus, and if its substance should happen to be affected with pneumonia or œdema, its characters are necessarily modified. The position occupied by a lung entirely compressed by liquid effusion is, as a rule, determined by its root; it becomes flattened against the mediastinum and backwards towards the spine, and if covered by a mass of false membranes its very presence may be altogether overlooked. Probably the notion of patients living on after having "entirely lost one lung," which is now so often applied to those who have phthisis, had its origin in autopsies made in cases of pleurisy of long standing. But when

the organ has previously been firmly fixed by adhesions, it cannot thus be driven inwards, and it may lie in the summit of the pleural space or be irregularly pushed to one side or even downwards. The most common deviation from the rule is doubtless when the upper part of the lung is affected with tubercular disease, and there is consequently a solid mass occupying a corresponding extent of the pleural cavity.

When pleural effusion is insufficient in quantity to cause the lung, as a whole, to become empty of air, and when therefore *compression* of the entire organ is a long way from having begun, the effect on the pulmonary tissue is very different from what is generally supposed. I think that everyone would anticipate that the elasticity of the organ would lead to a gradual and uniform shrinking of its substance, so that all parts of it should contain less air than before, without any part becoming completely airless. But the exact contrary is in fact the case. Dr Moxon long ago pointed out, when he was Demonstrator of Pathology at Guy's Hospital, that the presence of even a few ounces of liquid in any part of the pleural cavity causes a total collapse of the pulmonary tissue which ought to occupy that space. The writer has since repeatedly verified his observation; and Cohnheim, at p. 190 of vol. ii of his 'Vorlesungen,' makes a precisely similar statement. One often sees a small triangular area of collapsed lung-substance at the posterior inferior corner of the organ or a thin strip of it running up along its posterior margin. Nay, a mere enlargement of the heart, without there being any pleural effusion, may give rise to complete airlessness of the inner surface of the left lung; and distension of the abdomen, thrusting up the diaphragm, may cause a similar affection of the inferior surface either on one side or on both. The explanation of these remarkable facts can hardly be understood except in connection with the mode of origin of collapse of the pulmonary tissue, which has been discussed in the chapter on bronchitis (*supra*, p. 101). It depends upon the general principle, that whenever a part of the lung fails to be acted upon by the inspiratory forces, it becomes airless, notwithstanding that the tubes which serve it remain patent.

The production of local collapse of the lung-substance as the result of the effusion of moderate quantities of liquid into the pleural cavity has important clinical bearings. It accounts for a circumstance which has long attracted the attention of clinical physicians, namely, that temporary changes of posture on the part of the patient often fail to alter the position of the liquid within the chest, so far as one can tell by percussion. The statement was once made by a great teacher of medicine that whereas in a chronic pleurisy the effusion could be made to "gravitate" to a different part of the pleural cavity, this could not be done in acute pleurisy, because it was held in the meshes of fibrinous exudation. But surely it is quite the exception to be able to elicit evidence of gravitation, even in cases in which paracentesis is directly afterwards followed by a very free flow of liquid through the trocar, and Niemeyer agrees in this opinion. Even if one can alter the level of dulness a little by making the patient sit up the alteration does not amount to more than a finger's breadth or two. Now, if a certain part of the lung is rendered altogether airless by pleuritic effusion, it is easy to see how the fluid may (as it were) be *held up* in a fixed position in opposition to the force of gravity.

Even when the quantity of liquid is not very large, one can often in thin patients make out that the intercostal spaces are less depressed and

offer more resistance to the finger than on the sound side, and when the pleural cavity is full of effusion the ribs may be obviously wider apart, and the spaces between them may bulge or (occasionally) yield fluctuation. This, however, is but seldom observed. On measurement one generally finds, if there is much liquid, that the affected side is enlarged, and sometimes the difference between the two halves of the chest is considerable. In determining this Dr Gee's cyrtometer is very useful. The play of the ribs in respiration is greatly impaired, much more so than during the plastic stage of the disease. Moreover, in consequence of the extent to which the sternum is carried forwards, the movement even of the unaffected side during breathing may be much diminished.

Long before the lung has undergone complete compression in a case of pleurisy, other adjacent structures feel the pressure of the effused fluid upon them. The mediastinum is pushed over to the opposite side, the elasticity of the unaffected lung no doubt assisting to displace it. Thus if the pleurisy be on the right side, the apex-beat of the heart is felt and seen during life to be situated further to the left than usual; it may lie some distance outside the left nipple. If the disease be on the left side, the heart may pulsate in the epigastrium, between the sternum and the right nipple, or even to the right of the nipple, while in the normal position of the apex-beat no sign of the presence of the organ can be detected. Some observers have supposed that in such cases the heart swings over as a pendulum might, and that its long axis is now directed downwards and to the right, so that the part which beats against the chest wall is still the point of the left ventricle. But nothing seen in the *post-mortem* room would support this opinion. It is probable that the displacement of the organ is attended with little change in the inclination of its axis, and that the impulse is given by some part of the right ventricle.

It is to be noted that displacement of the heart is much more obvious and extensive when the effusion which causes it is situated in the left pleura than when it is in the right.

The cardiac sounds are sometimes altered in character under these circumstances. Dr Hope heard a systolic murmur over the aorta, which disappeared when the pleuritic fluid subsequently underwent absorption. Dr Walshe met with a case in which each sound of the heart was more or less masked by a blowing murmur for several successive days, while effusion into the left pleural cavity was at its height. He thinks that the diastolic murmur must certainly have depended upon malposition of the heart, probably through tension of the aorta.

It is of some importance to know what amount of liquid is required in order to produce a perceptible cardiac displacement. Fräntzel says that effusion scarcely ever reaches up to the third rib without affecting the position of the apex-beat to a greater or less extent, and that even smaller amounts of liquid often suffice. He also observes that in children the heart is more easily thrown out of place than in older persons. Another point mentioned by him is that when there have been previous adhesions between the pericardium and the left lung, pleurisy on the left side may cause the heart to be carried backwards away from the chest wall, so that no impulse whatever can be felt or seen. But whenever the amount of effusion is moderate, the normal apex-beat may be absent without there being any discoverable impulse elsewhere, the reason probably being that the sternum conceals it. In all cases of this kind the stethoscope must be used, with

the object of determining the spot at which the cardiac sounds appear the loudest.

The diaphragm is pushed downwards whenever the amount of pleuritic effusion is at all considerable. The displacement of the liver or of the spleen may be recognised by percussion, or one or the other may be felt projecting below the ribs. Fräntzel remarks that in the female the diaphragm resists pressure less than in the male, except when the abdomen is occupied by a pregnant uterus or when the intestines are over-filled with gas. According to this writer, when the distension of the pleura is extreme, it is sometimes possible to detect an elongated, tense, fluctuating swelling, which protrudes below the costal cartilages, and which is nothing else than the front part of the diaphragm itself.

Physical signs of effusion.—In the clinical recognition of pleuritic effusion physical examination of the patient plays the most important part. Several points already mentioned must be carefully sought for and noted; these are enlargement of the affected side, impairment of its movements, an altered state of the intercostal spaces, and displacement of the thoracic and abdominal organs. There remain the results of percussion and of auscultation. Now, of those two methods the former is in this stage of the disease by far the more valuable, as was long ago maintained by Piorry, in opposition to Laennec.

Dulness on *percussion* is, in fact, the main sign of pleuritic effusion. The percussion-sound becomes altered long before there is any evidence of pressure upon adjacent viscera. A circumscribed collection of serum and pus may of course cause dulness of any part of the chest, but when fluid lies free in the pleural space, the dulness is to be made out first at the base behind. One must not, however, suppose that small quantities of fluid ought always to be discovered by this means. Wintrich long ago declared that eight or ten ounces could scarcely be detected with certainty, and a considerably larger amount may escape recognition.

Much depends upon the habitual posture of the patient. If he is sitting upright in bed, the diaphragmatic surface of the lung, rather than its posterior surface, becomes compressed, and the percussion-sound may at first be scarcely altered. If he is lying down, the fluid is more or less spread out over the back, and is much more readily discovered. Perhaps, after all, Wintrich's dictum applies not so much to simple pleurisy as to the secondary effusions which occur in persons suffering from other complaints. For, in an ordinary uncomplicated case, if the percussion-sound down to the very bottom of the lung is perfectly resonant, one may in general assert that effusion, if present at all, is in such quantity only as to be clinically unimportant.

The dulness caused by fluid in the pleura differs from that produced by consolidation of the lung in being more absolute, and in the far greater sense of resistance which it conveys to the finger. Another important distinction is impairment or loss of vocal fremitus. The area over which these signs are to be detected of course varies with the amount of effusion. If this is considerable, the whole of the back and sides of the chest may be devoid of resonance, and may yield no vocal fremitus whatever. But now comes a remarkable circumstance. In such cases it usually happens that over the front of the chest—below the clavicle, or downwards nearly to the nipple—the percussion-note is sub-

tympanitic. Skoda first pointed out this fact, which will probably always remain associated with his name. The reason of it, however, is still doubtful. German writers are content to ascribe it to "relaxation of the pulmonary tissue," but there is no evidence that this is adequate to account for it. Dr Walshe thinks that it depends upon the presence of air in the multitude of minute tubes within the lung, so that a condition essential to its production is that these tubes should not have undergone compression as well as the lung-substance. What appears to be a satisfactory explanation is suggested by Dr Bristowe, namely, the diminution of the vibrating area formed by the thoracic walls.

When the whole of the pleural cavity is filled with fluid, there is absolute dulness in front up to the clavicle as well as behind. The dulness also passes over across the sternum to the opposite side.

With pleural effusion the tactile fremitus felt when the patient speaks is diminished or absent, a sign which is often of value in distinguishing this condition from solidification of the lung. Nevertheless, in exceptional cases this fremitus is palpable or exaggerated.

Auscultation is of less assistance than percussion in the detection of pleuritic effusion. In many cases one finds that the breath-sounds on the affected side of the chest are enfeebled, indistinct, or altogether wanting. But as often, perhaps, tubular breathing is audible over at least a part of the compressed lung, and in some exceptional cases this can be heard so extensively that one might well suppose the air to be entering freely into every lobe.

Nor is there any fixed rule with regard to *auscultation of the voice*. Very often one finds that it is conveyed to the surface more feebly than on the healthy side of the chest, but sometimes there is well-marked bronchophony, and this may occasionally be extremely loud. Or a peculiar modification of the voice may be transmitted to the ear, constituting what was called by Laennec *ægophony*, from its resemblance to the bleating of a goat. He also compared it to the voice of Punch, and in its most typical form it has a tremulous squeaking character, which is very curious. Dr Stone's explanation of it has been already given (p. 91, note). With regard to the frequency of this sign, widely opposed statements have been made by writers in consequence of their differing as to the definition of the term. Almost all the best observers are agreed that what may be called *pure* ægophony is very uncommon, and that it rarely lasts more than a few days. But between it and bronchophony there are all degrees of transition; and if one is to speak of the voice as ægophonic in every case in which it reaches the surface of the chest with more or less of a twang, there are very few instances of pleuritic effusion in which this change in its character is altogether absent. In one particular region this sign is observed far more frequently than anywhere else, namely, about the inferior angle of the scapula, and round towards the axilla. But sometimes it is heard in front, near the nipple, or even close to the clavicle. Its production is believed to depend upon the presence of a rather thin layer of liquid between the lung and the parietes. Consequently it generally disappears as the effusion increases, unless there are adhesions which keep the lung fixed at a certain distance from the surface of the chest. But in some exceptional cases, which are not always explicable by the existence of adhesions, ægophony may persist in spite of abundant accumulation of fluid, and according to some of the most experienced of modern auscultators, it is sometimes present when there

is no fluid effusion at all. Dr Walshe points out that one source of fallacy lies in the possibility of overlooking the fact that the ordinary voice of the patient is shrill and tremulous, as it so often is in women of advanced age. He also remarks that bronchophony may acquire an ægophonic character if a person speaks with the nostrils closed.

General symptoms.—The symptoms of pleurisy vary widely in severity in different cases. They are sometimes so marked as directly to suggest to the least experienced observer the nature of the case with which he has to deal; they are sometimes almost, if not quite, absent.

Foremost among them is pain in the side, the *point de côté* of French writers. This is often very violent, and of a sharp tearing or cutting or stabbing character. It is increased both by movement and by pressure. The patient therefore breathes in a shallow, jerky manner, for he fears to draw a deep inspiration. His cough, if he coughs at all, is short and half suppressed for the same reason, and he abstains as much as possible from laughing or sneezing. He lies during the early part of his illness on his back and on the unaffected side, and he shrinks from percussion and even from pressure with the finger upon the intercostal spaces of the inflamed side. Sometimes the pain is augmented by each ensuing exacerbation of pyrexia. As to its immediate cause, there have been discussions which have not led to any very definite result. Some have thought that it might depend upon an extension of inflammation from the parietal pleura so as to involve the neurilemma of adjacent intercostal nerves. Cruveilhier attributed it to the friction of the opposed serous surfaces upon one another, and in this way he accounted for the fact that its seat is so often limited to the lower part of the chest—about the nipple, or between the fifth and the eighth ribs—since there is so much more movement of the visceral upon the costal pleura there than higher up. But the pain is sometimes felt in the shoulder, in the armpit, beneath the clavicle, or along the sternum. In some cases, too, it is referred to the terminal branches of the intercostal nerves: to the hypochondrium, so that a mistaken diagnosis of hepatitis has sometimes been given; to the loins, so that the case has been called one of lumbago; or to the neighbourhood of the umbilicus, so that peritonitis has been suspected.* Laennec, and afterwards Gerhardt, declared that the pain of pleurisy was sometimes seated upon the opposite side of the chest to that which was inflamed, but this is questionable. In some cases pain is altogether absent, and then a large quantity of effusion may accumulate in the pleural cavity without its presence being thought of. Fräntzel says that this is especially apt to occur in children, in very old people, and in lunatics. It often happens that this symptom subsides or entirely disappears towards the end of the first week, or even after two or three days.

Next to pain, *dyspnœa* is the most striking symptom of pleurisy. The breathing is short and jerking, but it is also increased in frequency, especially when the patient exerts himself, as in lifting anything or in going upstairs. Sometimes the scaleni and the other muscles of forced inspiration are brought into action; and the nostrils dilate each time air is drawn into the chest. As effusion accumulates, the patient begins to find that he can

* I have myself had a patient whose sole complaint was of pain in the crista ilii; I feel sure that if it had not happened that a short while before my attention had been specially directed to this question, I should have failed to discover that he had pleurisy, although on applying my stethoscope I at once heard a rub.—C. H. F.

lie over towards the affected side more comfortably than in any other position, because the weight of the fluid is then removed from the mediastinum. He often has orthopnœa, for the diaphragm works more freely while the upright posture is maintained. Sometimes there is lividity of the cheeks and lips. It is worthy of notice that the dyspnœa of pleurisy is generally more marked in robust plethoric patients than in those who are anæmic and wasted.

As Andral long ago pointed out, it sometimes happens that this disease scarcely at all interferes with a person's comfort. He had a patient who went on with his work as a carter, in spite of an enormous effusion into his pleura; and Sir Thomas Watson speaks of a butcher, who under similar circumstances could not be persuaded that he was otherwise than well, and fit to leave the hospital.

Cough is seldom entirely absent in pleurisy; and there appears to be no doubt that it may occur independently of any affection of the lung or of the bronchial tubes. Sometimes it can be excited by percussion or pressure over the painful intercostal spaces, or by changes of posture. Kohts also says that during the operation of paracentesis he has seen cough produced by movements of the trocar, disturbing the serous membrane. In his experiments on animals he found that it was caused by irritation of the parietal, but not of the pulmonary, pleura. The cough of pleurisy is not usually attended with expectoration; but sometimes the patient gets rid of a small pellet of mucus or of a little muco-purulent material, in which case one must suppose that a slight bronchial catarrh is also present.

The *pyrexia* of pleurisy is generally moderate. The disease, when uncomplicated, seldom sets in with a violent rigor. But slight chills returning day after day are observed in many cases, especially when the patient remains out of bed during the day. The temperature commonly ranges at about 101° or 102°, but in the most severe forms of the disease it may reach 104°, or even a still higher point. In persons suffering from cancer, or from a chronic affection of the kidneys, there may be no pyrexia at all. The pulse is accelerated. Fräntzel insists upon the importance of watching it with care. For as effusion goes on, although the temperature may fall, the pulse not only becomes more rapid, but its volume and tension diminish, in consequence of the obstruction to the flow of blood through the pulmonary vessels. This affords a valuable indication of the degree of danger to the patient's life; and during paracentesis the physician may be able to feel the pulse becoming fuller, and at the same time slower, under his finger.

Diagnosis of empyema.—Can we determine by symptoms whether the effusion in a case of pleurisy is serous or purulent? One of the chief indications of the formation of pus is the continuance of high evening temperatures after the first two or three weeks; the pyrexia in many instances assumes a regularly hectic type. Œdema of the subcutaneous tissue of the affected side of the chest has long been mentioned as a sign of empyema, but it is often absent where suppuration is going on; and Fräntzel cites a case observed by Traube, in which it was present, but in which a fibrino-serous liquid was withdrawn by paracentesis. The best way of detecting the œdema is to pinch up a fold of skin, and to compare its thickness with that of a similar fold on the opposite half of the body. Fräntzel and Peter have each found in certain cases of pleurisy that the temperature of the skin was persistently higher

by about a degree Fahr. on the diseased than upon the healthy side of the chest, and almost every instance the effusion proved to be purulent. As practical rules, we may bear in mind that empyema is far more common in a child than in an adult, that rheumatic pleurisy never is purulent and renal pleurisy rarely, but that tubercular is often so, while traumatic commonly, and pyæmic pleurisy always, causes an empyema. Among adults, empyema is more common in men than in women.

It has been thought that empyema, and pleuritic effusion in general, is much more common on the left hand than on the right. Dr Eddison, of Leeds, in 40 cases found 20 right to 20 left. Dr Richards, of Birmingham, 3 right to 7 left. Dr Godfrey Carter, of Leeds, 19 right to 26 left. Of 42 cases under the editor's care, 14 were right and 28 left. Adding these numbers together, we find that of 137 cases of empyema 56 were right and 81 left.

Course and event.—The course of pleurisy varies widely in different cases. In the most severe form of all, which is very rare, but of which Fräntzel says that he has seen three examples, the patient quickly falls into a typhoid state, with delirium, stupor, and a dry fissured tongue: the dyspnœa and lividity increase, so as to threaten his life by the end of the first week; and though paracentesis be performed and repeated, the effusion collects again so rapidly that the fatal issue is scarcely retarded.

Even when the disease seems to be attended with no alarming symptoms, one must never regard it as free from danger, if the amount of effusion is large. Death sometimes occurs quite suddenly and unexpectedly. In 1874 this happened to a patient in Guy's Hospital with double pleurisy, who a short time before had been seen quietly asleep. For some days previously this patient had had much dyspnœa, and it seems not unlikely that the immediate cause of death was exhaustion of the respiratory centre (cf. vol. i, p. 10). But in another case, which ended fatally after an hour's extreme distress of breathing on the evening after admission, it was observed that the pulse ceased before the respiration. In that instance the effusion was on the right side; but there was œdema of the left lung, and it is probable that this really killed the patient. For the occurrence of fatal syncope, when the left pleura is the one affected, a special explanation has been suggested by Bartels, of Kiel, in the 'Deutsches Archiv' for 1868. It depends upon the anatomical fact (which has been verified after death on several occasions by him and by Fräntzel) that when the heart is pushed far over to the right the mouth of the inferior vena cava becomes bent almost at a right angle, just above the quadrilateral aperture in the diaphragm, the wall of the auricle forming a fold which covers a large part of that aperture. This is supposed to interfere with the due supply of blood to the heart, especially if at any moment the diaphragm is suddenly curved upwards in a fit of coughing, or if a sudden muscular effort is made.*

But pleurisy is not always attended with such risks. The inflammation need not go beyond the exudation of lymph, and after a time it may subside, leaving adhesions which fix the lung to the chest wall for the rest of life. Whether dry pleurisy necessarily ends in closure of the affected part of the pleural space, or whether it may not sometimes pass off without permanent damage to the serous membrane, is a question to which it would

* Trousseau had previously attributed the occurrence of sudden death in cases of pleuritic effusion to "twisting of the aorta and large vessels," as a result of displacement of the heart; but he does not seem to have had the inferior vena cava particularly in view.

be difficult to give a positive answer. What is well known to every pathologist is the fact that an adherent pleura is so often found when there has been no history of any chest affection. Dr Gee gives a tracing, made by the aid of the cyrtometer, which shows that in a child the chest may be markedly contracted on one side, as the result of a universal closure of the pleural space, without there having been any symptoms to suggest the presence of such a morbid condition; the patient died after an operation. There is no clear evidence that the adhesions left by a dry pleurisy at all affect the health. Some auscultators have thought that such adhesions may account for the occurrence of creaking sounds over the lower part of the lung on inspiration, especially when such sounds are constantly heard over a period of weeks or months.

The *duration* of an attack of dry pleurisy is sometimes exceedingly brief. I was once asked to visit a student who had been seized the same day with violent pain in the side, and who told me that he knew he had pleurisy, because he had had the disease before. I heard a very well-marked rub on auscultation, and told him that I should come to see him on the following day. But when on the morrow I walked into his room he assured me that he was well again; and on listening I could detect nothing abnormal.

When pleurisy gives rise to effusion it sometimes happens that the amount of liquid remains small, so that one can express a confident opinion that absorption will after a time occur, and that the compressed pulmonary tissue will expand and perfectly resume its functions. And even if the quantity should be very large, there is always a possibility that the patient's ultimate recovery may be complete, although one is not justified in predicting it.

Percussion usually affords the earliest indication of the subsidence of pleural effusion. The dulness becomes less extensive and less absolute, not only in front, but also over the upper part of the lung behind. A little later, the displaced organs may return to their proper situations, and the side may fall back to its proper dimensions. A feeble vesicular murmur may then be heard where none had been discoverable before. But with regard to this, there is a source of fallacy which must be borne in mind. At a certain stage of pleurisy, even while the affected lung still remains completely flattened and airless (as subsequently appears from an autopsy), the inspiratory sound from the opposite lung is very apt to be carried across the spine in such a way as to suggest that air enters both sides of the chest. Why this should be the case when the disease is advanced, and not at its commencement, is not clear.

Another physical sign which commonly attends the absorption period of pleural effusion is what is termed a "redux rub." This exactly resembles the friction-sound of the earliest stage of the disease, differing (if at all) only in being still louder and in being heard over a more extensive area. It often remains audible for several days or even weeks together, and may be accompanied by a return of pain in the side, without there being any reason to suspect fresh inflammation.

Even when the attack has so far subsided that the patient feels well, and perhaps resumes his occupation, it often happens that the side still remains more or less dull on percussion, and that the breath-sounds over the affected part are much feebler than natural.

But there are other cases in which pleural effusion, if left to itself, instead

of undergoing absorption, perforates the serous membrane, and makes its way out of the body. The liquid is then always purulent; the disease is an *empyema*. Sometimes it escapes through the lung and into the bronchial tubes. If this should occur while the patient is asleep, or if he should be in an exceedingly feeble state, so as not to be able to expectorate, he may be instantly suffocated. But surprisingly large quantities of pus are sometimes ejected, with much less distress than might have been anticipated. If the opening leads directly into the bronchial tubes, air passes into the pleural cavity to take the place of the liquid, and a "pyopneumothorax" is established, the effects of which will be discussed further on. But in many cases no such result follows. This appeared so remarkable to some of the older physicians, that they invented for the purpose of explaining it the theory that pus was capable of undergoing absorption from the pleura, and of being afterwards excreted from the bronchial mucous membrane. The true way of accounting for it seems to be one which was first suggested by Traube in 1872 ('Ges. Abhandlungen,' vol. iii, p. 44). He pointed out that if the pleura alone is eaten through, the alveolar texture of the compressed lung may allow pus to be forced through it by violent coughing, while it yet fails to afford a passage to air in the opposite direction, especially as there is little or no movement of that side of the chest during inspiration. In one case he found *post mortem* that the visceral layer of the pleura had been destroyed over a surface an inch broad by two and a half inches long, but that the pulmonary tissue was simply laid bare, there being no discoverable opening into a bronchial tube.

Such cases often end in recovery, as Hippocrates knew, when he wrote: "Those in whom a pleurisy ends in suppuration, may be cured if they bring up the matter within forty days from the rupture into the pleura." ('Aph.,' lib. v, cap. xv.)

In other cases, but far less often, an empyema makes its way outwards through the parietal pleura. An intercostal space is then usually perforated; and a soft elastic swelling, of greater or less size forms beneath the skin, which ultimately becomes reddened, ulcerates through, and allows an enormous quantity of pus to escape. At the present day one seldom has an opportunity of observing this result of pleurisy, because scarcely any practitioner now fails to diagnose a large pleuritic effusion, and to treat it surgically. The point at which perforation is most apt to occur has recently (in the 'Lancet' for 1882) been accurately defined by Mr Marshall, as being in the fifth space, below the nipple; here there is a weak spot in the chest wall, covered only by the internal intercostal muscle and a thin layer belonging to the great pectoral and the external oblique muscles. But the orifice may be elsewhere, and is sometimes as high as the second space. Or again, the diaphragm may be perforated by an empyema. It does not in this case always happen that an acute diffused peritonitis is set up—which would of course prove rapidly fatal. In 1865 a man died in Guy's Hospital of an empyema, which had been tapped eleven days previously. A hole, which had a diameter of a quarter of an inch, was found in the fleshy substance of the diaphragm, and below it was a large circumscribed abscess.

Again, the pus may make its way backwards and point in the loin. In 1858 a boy nine years old was in the hospital for pleurisy, and was discharged convalescent. Afterwards he came back with a swelling which, as it pulsated and was situated in the left lumbar region, was at first suspected of being an aneurysm. However, it proved to be an abscess and was punctured. Two

months later the boy died of tubercular meningitis, and an autopsy showed that the left lung was still contracted, and that a sinuous channel, six inches long, extended down from the pleural space behind the diaphragm to the external opening. Cases have been recorded in which the pus from an empyema has burrowed until it actually reached the popliteal space.

Except in those cases, already alluded to, in which the pus escapes through the pulmonary tissue, the spontaneous discharge of an empyema is almost always followed by a protracted illness, and very generally ends in the death of the patient. The entrance of air into the serous space often renders the fluids contained in it putrid; and this leads to irritative fever, and to more or less rapid emaciation and exhaustion. Otherwise, a pleural fistula may go on discharging for five, ten, and fifteen years, or even longer, until lardaceous changes develop themselves in the abdominal viscera, and cause death by renal dropsy. The only chance of recovery in cases of this kind seems to be that the whole cavity should be gradually obliterated by the abundant formation of granulation tissue, and by the contraction of the dense fibrous material which becomes developed from it. This indurated substance sometimes reaches the thickness of an inch. At the same time all the structures which surround the pleura become dragged inwards so as to reduce it within the smallest possible limits. The ribs fall in and may almost come into contact with one another; they remain motionless during inspiration, or (as was once observed by Dr Gee) their anterior parts may actually recede and move backwards each time that the healthy half of the chest expands and draws the sternum forwards. The dorsal spine becomes curved, so as to present a concavity towards the affected side. The shoulder sinks, the diaphragm is dragged upwards, with the abdominal viscera beneath it; the mediastinal structures are pulled over, the heart especially being often brought so widely into contact with the inner surfaces of the ribs that its impulse can be seen and felt over a far more extensive area than under normal circumstances.

Diagnosis.—As a rule, the recognition of pleurisy is easy, being based directly upon the characteristic physical signs. But some medical men are far too ready to set down to this disease cases in which there is no symptom except a *pain in the side*, perhaps of a neuralgic or myalgic character. Hospital practice teaches us to distrust the statements of patients, when they tell us that they have been under treatment for pleurisy. It has sometimes happened that an eruption of *shingles* has escaped notice, from not having been looked for. Costal *periostitis* and abscess of the chest wall are other affections the possible presence of which must not be forgotten. The only case in which it is allowable to diagnose pleurisy without positive evidence from percussion or auscultation is when violent pain in the lower part of the chest is accompanied by pyrexia. It is then reasonable to suppose that there is inflammation of the upper surface of the diaphragm and of the corresponding surface of the lung.

Even when one thinks that one hears a *pleuritic rub*, there is sometimes need of caution. Dr Gairdner has recorded an instance in which a sound which he describes as having a shuffling character, attended with a tactile sensation as of a jerking movement, produced by something rubbing up and down against the walls of the chest, proved to be due to emphysema of the lung. And Guttmann cites a case of Jürgensen's, in which a similar effect was produced by tubercles projecting above the surface of the pulmonary

pleura. Frequently what has been called a pleuritic rub becomes obviously a mucous râle.

A much more common mistake is that of attributing to pericarditis a friction-sound which is really pleuritic.

Pleuritic effusion has sometimes been diagnosed when the disease (if on the right side) has been a *hydatid* in the liver, or a hepatic *abscess*, or a hypophrenic abscess, or when (if on the left side) it has been an abscess connected with the spleen. Again, it is remarkable that all the examples of very large chronic *pericardial effusion* which have occurred at Guy's Hospital have been set down to pleurisy. But it is probable that one might always be put on one's guard by carefully mapping out the area of percussion-dulness, and by noting exactly how far it extends in front, at the side, and behind. To say that the disease might not be a circumscribed empyema would, indeed, be impossible; but at least one would be saved from imagining that the fluid lay free in the pleural cavity.

When there is a very large effusion of pus into the left side of the chest, pulsation synchronous with the heart can sometimes be felt in the intercostal spaces near the nipple or above it and towards the clavicle, so that the presence of an *aneurysm* may be suspected. A case of this kind was recorded three centuries ago by Baillon; its real nature was cleared up by the bursting of the swelling with discharge of pus from it. In our own time Dr Walshe has studied "pulsating empyema," as he terms it; and Traube threw out the suggestion that the existence of pericardial (in addition to the pleural) effusion might perhaps aid in giving it its peculiar character by facilitating the movement of the heart from left to right during the systole, and so increasing the force of its impact against the structures adjacent.

Again, all the physical signs may point to the presence of a large pleuritic effusion occupying the lower and back part of the serous cavity, and yet the diagnosis may not be absolutely free from possible sources of error. Traube has related a case of acute thoracic disease in which he imagined that there were both hepatisation of the left lung and exudation upon its surface, but in which an autopsy showed that the serous cavity had been closed by former adhesions; the great diminution of tactile vibration in this instance was attributed to plugging of the smaller bronchial tubes by lymph. Most English observers follow Laennec so far as to think that the detection of ægophony is conclusive evidence that there is at least some fluid effusion into the pleura. Fräntzel, however, declares, as the result of careful observation directed to this question for some years, that this opinion is incorrect, as had, indeed, long ago been asserted by Skoda.

But in chronic cases a more serious error may be committed; that of mistaking for pleuritic effusion a mass of *malignant growth*. Every physician of experience must either have seen this mistake made by others or have made it himself. One should therefore never give an opinion without having thought of such a possibility; the points to be especially noticed are whether the area of dulness corresponds in shape with that caused by a distended pleura, and whether tactile vibration is or is not still to be felt in certain positions. It must be borne in mind that a new growth situated in the mediastinum or in the lung is often accompanied by effusion into the pleura, so that a very strict differential diagnosis may be, after all, sometimes less accurate that a more doubtful opinion. Fräntzel relates a converse case, in which a large hæmorrhagic effusion was for a time supposed to be a solid tumour.

Even when the presence of liquid in the pleural cavity is positively and correctly diagnosed, one must not assume as a matter of course that the effusion is due to pleurisy. Passive dropsy of the serous cavity, or *hydrothorax*, may give rise to precisely similar physical signs, except that, being probably never altogether unilateral, it is not likely to displace the heart.

Again, pure blood may fill the pleura, constituting what is termed *hæmatothorax*. Apart from surgical injuries, the chief cause of such an affection is the rupture of an aneurysm of the aorta. Sir Thomas Watson mentions a case in which caries of a rib led to destruction of the wall of the intercostal artery and to distension of that side of the chest with blood, a large part of which was clotted in concentric layers.

In children, the difficulties of diagnosis of pleural effusion are greater than in adults, especially between it and broncho-pneumonia with extensive collapse; and the temperature affords little help in distinguishing a purulent from a serous effusion, for it is so readily raised in a child. Dr Thomas Barlow and Mr Parker (in a paper read at the British Medical Association in Manchester, 1877) quote a case of serous pleurisy with a temperature of 103·4, and of empyema with the highest evening temperature 101·5. They look upon anæmia with clubbing of the fingers as the best signs of the purulent character of an effusion, but depend on the practical decision of the hypodermic syringe, which has for this purpose replaced the grooved needle.

The frequency of empyema in children is illustrated by the following figures quoted in the above paper: out of 44 consecutive cases of pleurisy in the Great Ormond Street Hospital, 27 were purulent, and in another series of 16 cases 14 were purulent.

Complications.—Pleurisy does not lead to pneumonia, nor directly to phthisis. Empyema is generally attended with more or less swelling of, and exudation into, the subserous connective tissue. In a case which occurred at Guy's Hospital in 1872 the surface of the lung was covered with reticulated lines, due to the presence of pus in the lymph-channels beneath the visceral layer of the pleura. In another case, in 1869, pus was found outside the parietal layer of the pleura, infiltrating the intercostal muscles; there was also in the mediastinum a diffused abscess as large as a walnut. In a third case, in 1873, the mediastinal tissues were infiltrated with a mass of puriform lymph, three quarters of an inch thick. All three were examples of double pleurisy, accompanied by pericarditis. Cases differ very much as regards liability to the spread of inflammation from the pleura to adjacent serous membranes. The pleurisy which so constantly arises in phthisis seems never to extend to the pericardium. But the more intense and violent forms of the disease are exceedingly apt to set up pericarditis. Moreover, there are cases in which both pleuræ, the pericardium, and the peritoneum seem to become simultaneously inflamed, or at least in which it cannot be determined that any one of them was affected earlier than the rest (cf. vol. i, p. 68). As a rule, such cases present very acute symptoms and run a rapid course. But this is not invariable. In 1876 a woman, aged twenty-three, was admitted into Guy's Hospital with what was supposed to be enteric fever. After a fortnight fluid effusion was detected in the left pleura, and forty-two ounces were drawn off by the aspirator. Her febrile symptoms continued; she became emaciated and died. As may well

be understood, it was thought towards the last that she was suffering from some obscure tubercular disease. However, on making the autopsy, we could discover no tubercles nor any primary visceral lesion. Beside the pleuritic effusion on the left side which had been diagnosed, there was lymph over the whole of the right pleura; the pericardium was adherent by a recent plastic exudation; the liver and the spleen were fixed to the diaphragm; and the lower part of the abdominal cavity contained a small amount of purulent fluid. It may be that the disease was of rheumatic origin, for the girl was said to have had a painful affection of her joints about two months before; but there was no evidence of endocarditis, present or past.*

It has been laid down that, whenever the two pleuræ are attacked with inflammation simultaneously or in succession, one ought to suspect that there is some underlying condition such as Bright's disease or latent tuberculosis. But this is not always the case.

Ætiology.—(*a*) *Of idiopathic or primary pleurisy.*—Foremost among the causes of pleurisy is *cold*. This has been so commonly set down as giving rise to all forms of internal inflammation, and often with so little reason, that one cannot be surprised that many physicians are reluctant to recognise its operation. But the clinical evidence of the direct dependence of pleurisy upon cold is most striking and cannot possibly be explained away. Fräntzel cites, for example, cases of persons who have been attacked after having exposed one side of the body to a draught in changing their clothes while heated, or after having got one side wet through in a driving rain. Other cases have immediately resulted from sitting near an open window or a badly-fitting door, especially during convalescence from some acute illness or (it is said) after a course of mercurial treatment.

Injuries to the chest often give rise to pleurisy. Not only does this occur when the ribs have been broken, but also when there is no evidence of damage to the walls of the chest.

(*b*) *Of secondary pleurisy.*—Certain general diseases are very apt to be attended by pleurisy as a complication. This is the case with acute *rheumatism;* but inflammation of the pleura, when it is of rheumatic origin, occurs only during the fever, not independently, as occasionally happens with pericarditis or endocarditis. Scarlet fever must also be mentioned. Fräntzel lays down the rule that enteric fever never becomes complicated with pleurisy during its early stage, when the morning temperature is not below 102°.

Among chronic affections, none is so commonly accompanied by pleurisy as Bright's *disease of the kidneys;* so that the urine should always be carefully and repeatedly tested for albumen.

Secondary pleurisy is often due to the direct extension of inflammation from some adjacent structure. To the pathologist there is probably no condition which is so familiar as this; but the affection is in many cases found to be quite recent and early at the time of death, so that it has no clinical significance. Among the less obvious starting-points for a severe or even fatal pleurisy may be mentioned abscesses in the armpit, operations

* The case is of interest, not only pathologically, but also on account of its obscurity during life. For the thoracic serous cavities and the upper part of the peritoneal space have been repeatedly found all closed by old adhesions, when there had been no history of any chest affection; and it would now seem that such a result may arise from an illness which clinically might be taken for fever.—C. H. F.

upon the breast, suppuration of the cervical connective tissue after tracheotomy, caries of the ribs, mediastinal abscess, cancer of the œsophagus, and caries of the dorsal vertebræ. Or its origin may be below the diaphragm, the connecting lymph-channels discovered by Recklinghausen probably conveying the inflammatory process from one serous cavity to another. Thus pleurisy which was the immediate cause of death, has been found to be due to extension from a peritonitis of puerperal origin, or following ovariotomy, or arising from disease of the rectum. Again, a very acute inflammation of the pleura, with fœtor of the pus, has started from the upper end of a psoas abscess, or from a localised abscess behind the stomach due to a perforating gastric ulcer. Mediastinal growths without inflammation frequently produce pleuritic effusion; so also do malignant tumours of the walls of the chest or of the mamma.

But by far the most common causes of secondary pleurisy are *affections of the lungs*. Indeed, it may be said that pleuritic effusion is in older persons most often due to renal disease, and in young patients to tubercle.

It is unnecessary to repeat that dry pleurisy occurs in every case of acute pneumonia, and in almost every case of heart disease in which pulmonary infarcts are formed. In pyæmia (especially when resulting from thrombosis of the cerebral sinuses, itself consequent upon disease of the temporal bone) pleurisy is sometimes the most conspicuous feature of the case, and may be mistaken for the primary disease and the cause of all the patient's symptoms. Still more important is it to bear in mind that what seems to be an uncomplicated and simple attack of pleurisy may really be dependent upon extensive pneumonia, of which there is sometimes little or no clinical evidence. When pleural effusion has once taken place, it may be impossible to discover by physical signs the presence of hepatisation of the corresponding part of the lung. It often happens that the characters of the sputa afford the only clue to the real nature of the case, or a correct diagnosis may depend entirely upon one's having seen the patient at an earlier period, before the fluid was poured out into the serous cavity. Again, it is not improbable that a very limited patch of pneumonia, involving the surface of the lung, may sometimes be the starting-point of a diffused and severe pleurisy.

It is in regard to *phthisis* that this question is of the greatest importance, on account of its bearing on prognosis. One is frequently seeing patients who, having favourably passed through an attack of pleurisy, are shortly afterwards seized with hæmoptysis, or develop signs of tubercular disease of the lungs. Sometimes, no doubt, the presence of such disease can be detected even while the serous inflammation is in progress, if one is careful to examine the upper lobes thoroughly. But in many cases all the clinical evidence points to the conclusion that the pulmonary affection has been of later development. Those who adopt Buhl's infective theory of tubercle can then, of course, maintain that it is really secondary, and due to the absorption of caseous matter into the blood. But the primary pleuritic inflammation is almost always of a serous not a purulent character, and it is far more probable that it is due to a few pulmonary tubercles on the surface of the lung setting up irritation and effusion.

It is a curious question whether pleuritic effusion, while it continues to compress one of the lungs, favours a fresh development or a further growth of tubercles in that organ, or whether it may not rather be adverse to such an occurrence, even though it may increase the susceptibility

of the other lung, which has to perform extra work. The following six cases bear on this question. In one there were no tubercles except in the opposite lung; in another the tubercles were much less numerous on the side of the pleurisy; in two others it was observed that on that side they were all of old date and inactive. On the other hand, there was one case in which they were more abundant in the compressed lung than in the opposite one; and once a lung entirely airless was full of tubercles (some grey and others caseous) in its lower lobe, where their presence, the apex being healthy, is an occurrence so exceptional that one could hardly doubt that the pleurisy had determined their formation.

Prognosis.—This depends chiefly upon the answer to the question as to the origin of the pleurisy set forth in the preceding paragraphs.

Acute idiopathic pleurisy from exposure to cold in a healthy subject is scarcely ever fatal. With modern "antiphlogistic" treatment, it is speedily relieved, and cured without leaving sequelæ behind. Even when neglected, and when one side of the chest has been allowed to fill with serum, it is remarkable how well such cases do after aspiration, with warmth, rest, and restricted diet, aided, perhaps, by blisters or diuretics.

We must remember, however, the exceptional cases recorded by Frantzel (*supra*, p. 172), which must have been due to some undiscovered complicating cause.

A catarrh of the pleura, which half fills the chest without pain or fever, and is only discovered by physical examination in search of a cause for the patient's dyspnœa,—this should make one suspect tubercle or Bright's disease. But when there is no underlying morbid condition, the prognosis still is good.

To leave a patient, however, with one side of the chest full of fluid is never safe. He may die suddenly from syncope, or œdema of the other lung may suffocate him before help can be brought.

Pleurisy which forms part of acute pneumonia adds to the pain, but not to the danger of the primary disease, and the same is true when pleurisy is secondary to lymphoma, or other mediastinal growths. In the latter case it is usually latent, and often as much dropsical as inflammatory.

Tubercular pleurisy does not usually add to the dangers of phthisis, and often appears to retard its progress by limiting the amount of blood as well as of air in the diseased lung, for caseous inflammation does not flourish in anæmia and airless pulmonary tissues. On the other hand, pleurisy secondary to Bright's disease, whether dry or purulent, or combined with hydrothorax, is a very serious complication, only less so than the pericarditis which supervenes under the same circumstances.

When pleuritic effusion is purulent, the prognosis is more grave. Formerly the event was often fatal, by pyæmia, by hectic fever, by secondary tubercular phthisis, or by lardaceous disease; and in the best cases much deformity was the usual result, as described above (p. 175).

But the bolder treatment on which modern surgery safely ventures, has wonderfully improved the prognosis of empyema. In the case of children a single free opening made and a drainage-tube inserted has repeatedly been followed by re-expansion of the lung and complete recovery, without the least sign of the disease remaining except the scar of the operation. With adults the result is not so often perfect, but here also complete cures are not infrequent, and complete failure is the exception.

Treatment.—It is evident from what has been stated in the last few paragraphs that the treatment of pleurisy is a very important matter. There are cases which, left to themselves, run a course as favourable as could possibly be desired. There are others which, even if they do not end fatally, leave the patient crippled and deformed, worn out and emaciated by the drain of a constant purulent discharge. The problem is to modify the progress of these latter cases so as to make it resemble that of the former ones.

Except in the mild adhesive form of the disease, a person affected with pleurisy should be kept strictly in bed during the early stage. For the relief of pain, the application of a blister is perhaps more generally serviceable than anything else. It may be used with advantage at any period, even when the inflammation is advancing. Cupping, however, may sometimes be first employed, if the patient is robust, while many physicians hold that for prompt and grateful relief of the acute pain, as well as of the dyspnœa, no remedy is so efficacious as the application of half a dozen leeches to the side.

Among drugs, those most commonly prescribed when effusion has taken place are purgatives, diuretics (such as digitalis, acetate of potass, nitrate of potass), and absorbents (especially iodide of potassium internally, and diluted mercurial ointment externally). Marked results have followed the application of mercurial ointment, a rub which had been heard day after day for weeks ceasing almost as soon as it was used, or fluid effusion clearing away after a somewhat longer period. The diet should be light and spare. The plan of allowing the patient very little to drink, was often practised at Guy's Hospital by Sir William Gull and by Dr Moxon, and is advocated by Niemeyer. Fräntzel remarks that a very rapid absorption of fluids from the chest has been noticed when the patient has been attacked by cholera.

Paracentesis thoracis.—Whenever the physical signs indicate that there is considerable liquid effusion into the pleural cavity, the question of removing it by tapping must be carefully considered. This procedure, it is interesting to know, dates back to Hippocrates. But of course nothing that could be written about it before the time of Laennec is of any practical value now. And although Trousseau advocated it very strongly as far back as 1843, scarcely any physicians, until within the last twenty years, understood its importance or the desirability of frequently resorting to it.

If percussion shows that one side of the chest is full of fluid, paracentesis should be performed at once, on account of the danger of sudden death which attends this condition, as pointed out at p. 172. One should never wait even until the following day; and it is important to remember that the absence of obvious distress of breathing affords no proof that the operation may safely be postponed. Whether the temperature is high or not makes no difference whatever.

If percussion shows that the quantity of fluid is moderate, it is often advisable to wait for a few days before performing paracentesis. The presence of pyrexia is, at an early stage of pleurisy, a reason for delay, since it may reasonably be hoped that, by the end of two or three weeks from the commencement of the attack, the temperature will fall. When one decides to put off the operation, one must of course watch the patient very closely, observing whether the amount of effusion undergoes increase or diminution as time goes on. Unless it begins to diminish in the course of a fortnight, no further postponement of tapping is, as a rule, permissible. For, even though the quantity of liquid should remain quite unaltered, the probability that the lung will quickly expand and regain its functions after paracentesis

must obviously become less and less, in proportion to the length of time during which it has been compressed; for the layers of lymph that bind it down have been allowed to become fibrous and to contract. Another point of great importance is that the withdrawal of a part of the fluid by operation seems often to facilitate the absorption of the rest. The supposition is that the subpleural lymph channels are mechanically pressed upon by the liquid and that the flow through them is interfered with. Moreover, in cases of Bright's disease when one lung is half carnified by fluid, sudden œdema of the other may prove fatal before help can come. At an advanced stage of the disease, the fact that the temperature is high is only an additional reason for tapping the chest without further delay, especially if there is fever of a hectic type. For, as Kussmaul originally pointed out in the 'Deutsches Archiv' for 1868, the removal of pus from the pleural cavity, especially if fœtid, is often at once followed by the cessation of pyrexia.*

When it is decided to tap the chest, some physicians recommend that a preliminary puncture should be made by means of an empty hypodermic syringe, into the chamber of which some of the pleuritic liquid may be drawn. But it often happens that this procedure leads to no result, even though the diagnosis is correct. On the whole, the use of the instrument seems to be undesirable, at least when the condition of the patient is so serious as to make the removal of the effusion a matter of importance. For either liquid enters the syringe or it does not. In the former case a larger trocar is at once employed, and the patient might as well have been saved the slight pain of the preliminary puncture. In the latter case one is very much hampered in taking any further step, which is yet perhaps essential to his safety.

The aspirator is greatly to be preferred to an ordinary trocar for paracentesis of the chest, for through a trocar fluid will only escape from the pleural cavity when the pressure there is greater than the atmospheric pressure. Several pints may be present, and yet sometimes there may be only a momentary flow of it during the act of expiration, or when the patient happens to cough. Moreover, there is great danger of air being sucked back into the pleural space by a deep inspiration.

Some of those who advocated the operation a few years ago maintained that the introduction of air is a matter of but little importance, on the ground (which cannot be denied) that it has often occurred without ill effects. But at the present day few surgeons would deny that it must involve the risk of giving a septic character to the inflammatory process. However,

* Of this I saw a most striking instance in 1881 in a man who had pleurisy as a sequel of enteric fever. His temperature rose every afternoon to between 102° and 103°, falling in the night and morning. I had made one unsuccessful attempt to draw off the effused fluid, the reason of my failure being that I used a very fine aspirator needle, because the area of percussion dulness was not in the usual position behind, but at the side of the chest, just outside the situation of the pericardium, so that I felt some hesitation in acting upon my diagnosis. However, six days later, when I visited him, I found him in a most critical condition, with great anxiety of face and with extreme distress of breathing. As the only chance of saving his life I had a somewhat larger trocar plunged into the chest at exactly the same spot as before. Aspiration was then performed, but at first no fluid appeared. However, I took the instrument and pushed it inwards, feeling, as I did this, that the end of it encountered and seemed to pass through a resisting membrane. A quantity of rather viscid blood-stained liquid at once escaped, and of this four and a half ounces were withdrawn. The patient was instantly relieved, and from that time went on to recovery without a bad symptom. His temperature, which was rising at the time of the operation and had reached 100·8°, fell from that very moment; three hours later it was normal, and during the next three days it only once reached 99·4°.—C. H. F.

one can without difficulty prevent it by surrounding the mouth of the trocar with a piece of moistened gold-beater's skin, which acts as a valve; or, as has been usual in England, one can fit on an elastic tube and make it dip beneath the surface of water containing carbolic acid, in a basin on the floor.

In tapping the chest for simple pleuritic effusion Mr Davies-Colley thinks the best plan is, after introducing the aspirating cannula, to attach a long rubber tube to it, and to evacuate the cavity by means of the siphon-action exerted by the fluid in the pendent tube. If a stoppage occurs in the flow, the aspirator may be used to remove it, but otherwise it is better to avoid the strong action of this instrument, as it occasionally gives rise to hæmorrhage.

Even when the aspirator is used, there is often difficulty in getting out any considerable quantity of the liquid. Pieces of lymph are drawn against the inner orifice of the tube, or its channel may be occluded by viscid or curdy pus. Sometimes one can restore the flow by moving the cannula in various directions. If this fails, it may be necessary to withdraw it and to make a second puncture at a different spot. It should be a rule without exception never to employ for tapping the chest the hollow needles which are commonly sold with the aspiratory apparatus. One expects the lung to be expanded as the fluid is withdrawn, and there must be a very great risk of its being wounded if there be a sharp point in the way. A perfectly safe instrument consists of a trocar and cannula, the cannula having a lateral opening, to which the tube of the aspirator is fitted; there is also a stopcock which can be turned at the moment when the trocar, having punctured the chest, is being withdrawn. Another advantage of this instrument is, that should the cannula become obstructed, a blunt probe, fitting loosely to the cannula, can be passed in, so as to clear it out, without having to detach the aspirator tube.

The best spot for puncturing the chest is generally said to be about two inches outside the edge of the pectoralis major, and just above the edge of the sixth rib on the left side or of the fifth rib on the right side. At Guy's Hospital it has been usual to select a lower intercostal space, and a point much nearer to the posterior fold of the axilla. Dr Bowditch recommends that the instrument should be introduced between the ninth and the eleventh ribs. The reason for keeping close to the upper edge of a rib is that one is then not likely to wound the intercostal artery. In 1855 this accident happened during an operation performed at Guy's Hospital; the patient (who had phthisis) became faint at the time, and died the same evening; a pound of clotted blood was found in the base of the chest. Fräntzel speaks of the use of a "capillary" trocar as obviating all risk of such an occurrence; but instances of it are very rare, and from the difficulty of extracting the fluid in many cases, it is injudicious to employ too small an instrument.

It is not desirable, in performing paracentesis, to attempt to empty the pleural cavity. Fräntzel says that not more than about two pints and a half should be withdrawn at once. In many cases, during the operation or immediately afterwards, paroxysms of cough occur, which are best relieved by a hypodermic injection of morphia. As Fräntzel remarks, they are doubtless set up by the re-entry of air into the tubes of the lung which had been compressed. That they are not due to the contact of the cannula with the surface of the lung is, he says, shown by their being sometimes produced when the quantity of fluid is still so great that such contact cannot have

taken place. Moreover, he has repeatedly felt the orifice of the instrument rest against the pulmonary pleura without any cough resulting. Since he has used the aspirator and drawn off pleuritic effusion very slowly, he has very seldom observed these severe fits of coughing. Once, in a phthisical patient, he has known thoracocentesis to be followed by fatal hæmoptysis after an interval of eight hours; this was due to the rupture of an aneurysm in a vomica. It is easy to see that the increased activity of circulation in the pulmonary vessels, which must be one result of the operation, is very likely to lead to the giving way of any weak spot in their walls. A much more frequent occurrence is œdema of the pulmonary tissue on the affected side. This seems to be the cause of a phenomenon which attracted much attention in Paris about ten years ago—the expectoration after thoracocentesis of large quantities of a frothy liquid containing much albumen. The patient to whom this accident happens may have experienced the usual relief from the operation; but after an interval of from ten minutes to an hour his breathing becomes distressed, he begins to cough, and he may turn lived, and die in a quarter of an hour. Twenty-one instances of it were collected by Terrillon in a monograph published in 1873; most of them, however, ended in recovery, and some were comparatively slight. The close resemblance between the fluid discharged from the air-passages in such cases and that withdrawn by the trocar so short a time before seems to have led some distinguished French observers to suppose that the expectorated albumen was dependent upon the presence of a communication between the cavity of the pleura and the interior of the lung, their notion being either that the lung was wounded during the thoracocentesis or that a perforation existed previously, which became opened out as the pressure was removed from the surface of the organ. But, as Terrillon had no difficulty in showing, such opinions are quite untenable, and the only reasonable explanation is that there is suddenly produced an active hyperæmia, which leads to œdema of the pulmonary tissue.

Among the objections that have been urged against the performance of thoracocentesis, except in cases of absolute necessity, one, which had the support of Dr Stokes, of Dublin, and of Sir Thomas Watson, is that it may lead to the conversion into pus of an effusion originally sero-fibrinous. And although this suggestion is altogether rejected by Trousseau there is probably truth in it. Nothing is more likely than that the vessels in an inflamed pleura are often weakened and dilated to such an extent that a sudden and great removal of pressure from them may cause increased exudation. And it is certain that in many cases in which a clear fluid is withdrawn at a first operation, pus appears on the second or third occasion. Thus, although it accords with the natural tendency of the disease that as it advances the proportion of leucocytes in the exudation should become greater, one cannot but admit that this process may sometimes be hastened by the performance of an operation. Fräntzel speaks of a burning pain in the seat of puncture and of an indefinite sense of pressure on the affected side as being generally present, and as lasting in some cases for a day or two. He also says that thoracocentesis is usually followed by a slight elevation of temperature, and that until the second, third, or fourth day a more or less considerable increase of effusion may be observed, after which a somewhat rapid process of absorption sets in, attended with diuresis. How such statements are to be reconciled with the fact that an existing pyrexia is often suddenly cut short by the operation is not apparent. Certainly most

patients experience marked relief from the operation, and are in every way more comfortable after it than before.

In certain cases the fluid quickly accumulates again after paracentesis, so that the patient's condition becomes as bad as before, and the operation has to be repeated. When this happens two or three times in succession, perhaps at intervals of only a few days, Fräntzel advises that one should desist from further interference, which will only precipitate the downward course of the case.

Paracentesis in empyema.—If the fluid obtained by paracentesis is purulent, the further treatment requires to be modified. At the time the best course is generally to close the puncture with lint and plaster, for sometimes what remains of the effusion afterwards undergoes absorption. Cheesy masses, and even thick hard deposits of calcareous matter, are now and then found after death lying between adherent pleural surfaces; and there can be little doubt that such residues have usually, if not always, had their origin in an empyema. In other cases a collection of liquid pus, enclosed in a dense capsule, has been discovered in the pleural cavity, when the fatal illness was of an altogether different nature. Dr Moxon alludes to such an instance, in which there was a history of pleurisy three years before, and in which physical signs of fluid in the chest had been observed during the intervening period. Thus it is not a matter of course that one should give a very grave prognosis whenever one has tapped an empyema. But in most cases the effusion quickly reaccumulates, a second operation is required, and after this a third. It is right to allow at least two chances for the subsidence of the disease; and, as Fräntzel remarks, there is the further advantage that, each time one removes fluid, adhesions may possibly form between the opposed surfaces of the pleura, so as to narrow the cavity. But when a third puncture becomes necessary it is advisable to alter one's procedure, and to provide a channel by which for the future the cavity may go on draining itself, so as to give the best possible opportunity for its gradual closure by granulations.

In children we have sometimes attained admirable results by making an aperture just large enough to admit one end of a long elastic tube, of which the other end is carried beneath the surface of carbolised liquid in a jar placed beneath the bed. The elastic skin of a child grasps the tube firmly, and does not ulcerate. The negative pressure of the column of liquid acting hydrostatically seems gradually to raise the compressed lung. Within a surprisingly short time the flow of pus may cease, and a permanent cure may be obtained.

With older patients similar success is rare. The plan usually adopted at Guy's Hospital is to make rather a free incision, under the carbolic spray, into one of the lower intercostal spaces, to introduce a drainage-tube, and to allow the pus to escape into an antiseptic dressing. Injections of iodine, or of permanganate of potass, are often used to wash out the cavity at intervals of twenty-four or forty-eight hours.

The incision should be made along the upper border of the rib, a short distance in front of the angle. A large tube should be introduced, and care should be taken to fix it in such a way that only a small portion of the tube enters the pleural cavity. In children it may be necessary to excise part of a rib to allow of the introduction of a sufficiently large tube.*

* A somewhat different method is recommended by Fräntzel. Having, at the time of the operation, got rid of as much as possible of the pus, he pushes a long catheter

The operation of washing out the chest is not altogether free from danger. In 1876 Dr Cayley read before the Clinical Society a case which had occurred to him, and in which, while a solution of iodine was being injected, the patient suddenly became pale, unconscious, and convulsed; the temperature rose to 107°, and death followed in sixteen hours. He cites three cases recorded by French observers, in each of which like symptoms appeared, though one of them ended favourably. In 1874, at Guy's Hospital, a girl, aged sixteen, died in precisely the same way. She had had a drainage-tube inserted into the right chest for an empyema five weeks before, and was going on well. One day she was sitting up, and her chest was being washed out with carbolic acid, when she suddenly ceased to breathe, and, although artificial respiration was set in action, remained unconscious, with muscular twitchings, until death. Nothing has been found, on *post-mortem* examination, to account for such accidents. A suggestion that thrombi in the pulmonary veins may have been dislodged and have formed emboli in the heart or in the cerebral arteries seems to have been shown to be without foundation. It is noteworthy that in every one of the cases the chest had been washed out many times before without any ill-effects occurring; the only difference being that in two instances a somewhat larger quantity of fluid was being injected than usual. Fräntzel hints that it is important that the stream should not be directed towards the pericardium. In the girl who died at Guy's Hospital it was noted that there was a very slight basal pericarditis, and that the septum between the ventricles of the heart was ecchymosed.

The process by which the sac of a discharging empyema becomes gradually obliterated seems to consist in the formation of granulation tissue, and in the union of the two opposed surfaces; in most cases this union probably begins at the root of the lung, and spreads from one point to another until it reaches the external orifice. Even before this has obliterated the cavity, absorption of the air in the pleura takes place by solution in the lymph of the serous membrane, and, the wound being closed, the negative pressure in the pleura soon allows of the lung expanding if not bound down by adhesions. Fräntzel suggests, with much probability, that the compressed lung is re-inflated with air from the opposite lung during the act of coughing, or as the result of simple expiratory efforts with closed glottis.

The results of treatment of empyema by free incision and drainage, with modern methods and antiseptic precautions, are most encouraging.

downwards towards the spine, and slowly injects through it distilled water, at a temperature of 100°, until the space is full. He then draws off the water by another catheter with an exhausting syringe, and he repeats this procedure three or four times until what returns is quite pure. Masses of fibrin, sometimes as large as the palm of the hand, generally appear in the wound while this is being done, and are carefully removed. This is one of the advantages of Fräntzel's plan, for sometimes much trouble is caused at a later period by such masses, which may become putrid. Finally, he fixes in the aperture a flat silver cannula, with a broad plate fitting upon the surface of the chest, of such a size that two catheters can be passed through it side by side; outside this are placed antiseptic dressings, and over them a bag of ice. Each day afterwards the pleural cavity is twice washed out by means of the catheters, of which one is introduced with great care to the farthest possible point, so as to prevent any accumulation of pus from taking place. Another detail, which he deems very important, is that the patient should lie in such a position that the wound is at a higher level than any part of the pleural space, so that the fluid may gravitate into every part of it. After two days a solution of ½ per cent. of common salt is substituted for the distilled water; and later still a very dilute solution of iodine or of carbolic acid. He says that of eleven patients treated in this manner five were completely cured; five died, but most of them from causes which had little or nothing to do with the operation; one was under observation when he wrote, and was doing well.

Even in adults one may again and again see complete recovery of the lung with no resulting deformity. In children the practice is still more successful. Thus, Dr Goodhart reports that of 50 cases under Dr Frederick Taylor's and his own care 42 completely recovered, a sinus remained in 3, and only 5 died; one from suppurative pericarditis, one from septic pneumonia, and one from measles and broncho-pneumonia.

But in a certain number of cases, the cure of an empyema remains incomplete. The cavity may have shrunk to very narrow dimensions, the chest may have regained a fair amount of resonance over a large part of its surface, air may enter the lungs pretty freely, but there is a fistulous opening from which small quantities of pus continually drain away. In such cases, and generally when other treatment has proved unsuccessful, it has recently been the practice to excise portions of one or more ribs, so as to allow the side of the chest to fall in and meet the lung. This operation seems to have been first performed by Dr Peitavy. In the 'Birmingham Medical Review' for 1880 Dr William Thomas has recorded several cases which were so treated, and in almost all of them the result seems to have been highly satisfactory, the wound healing in a few weeks, and the lung rising completely. The rib, too, was restored by a new growth of bone. It is to be observed, however, that the patients were all children under eight years old. In 1877 Mr Howse excised portions of three ribs from a child, aged six, a patient of Dr Taylor, in the Evelina Hospital, with results which are detailed in vol. xiii of the Clinical Society's 'Transactions.' An interesting point was that the seventh and the eighth ribs were found at the time of the operation to be nearly united together by bridges of bone, which had formed round the track of a drainage-tube that had been lying for a considerable time between those bones. Some improvement followed, but the cavity of the empyema did not close, and ultimately the child died with lardaceous organs.

PNEUMOTHORAX

Origin and pathology—post-mortem characters—Physical signs—Diagnosis and symptoms—Prognosis—Treatment.

Some of the older pathological anatomists, including Morgagni, made mention of the fact that air sometimes accumulates in the cavity of the pleura; but the word pneumothorax was first used in 1803 by Itard in his 'Thèse de Paris;' and it was left to Laennec to give a full description of this affection.

Ætiology.—Most writers have admitted that in exceptional cases gases may be formed in the pleural cavity as the result of chemical decomposition of liquid effusion, and perhaps by direct secretion (or rather exhalation) from the lining membrane. Such notions, however, accord ill with the general doctrines that are now held held by almost everyone. And as neither of these supposed causes of pneumothorax has in its favour the slightest clinical evidence, we may now, guided by the experience of more than half a century, reject them altogether, and assume that air is never found in the interior of the pleural space except as the result of a breach in the continuity of its surface, placing it in more or less direct communication with the external atmosphere.

Seeing how delicate are the structures which separate the pulmonary alveoli from the surrounding serous space, one cannot be surprised that in the immense majority of cases pneumothorax is a consequence of perforation of the visceral layer of the pleura, allowing air to escape from the lung. Very often this arises from direct violence. Broken ribs are exceedingly apt to wound the lung, and in persons who are run over or severely crushed the organ may be torn without there being any fracture of bone or laceration of the costal pleura. So, again, pneumothorax may be produced by powerful muscular efforts, even when the lung has not been previously diseased. Thus, Fräntzel relates in 'Ziemssen's Handbuch,' how a youth of nineteen, who was exerting all his strength to push a heavy cask, felt something give way in his chest, and became suddenly short of breath and powerless. As he recovered entirely within six weeks, without any affection of the lungs having been discovered, it may probably be assumed that none had existed before. A similar accident sometimes happens during the violent straining which attends the paroxysms of whooping-cough. The fact that the bullæ in emphysema of the lungs often have the thinnest conceivable walls might naturally lead one to anticipate that pneumothorax would occasionally arise from their rupture. But this if it ever happens must be exceedingly rare.

As a general rule, the entrance of air into the pleural space is the result of some local inflammatory affection of the lung, leading to ulceration or sloughing of the pulmonary pleura. Sometimes, though very seldom, the disease is acute pneumonia running on to gangrene. Much more often it is a slough, dependent upon infective emboli, such as are carried to the lungs in cases of ear disease, or in a great variety of surgical affections. Some-

times it is fœtid bronchitis, with sacculated dilatations of the tubes. Or it may be an abscess starting from the glands at the root of the lung, and making its way first into a bronchus and thence into the pleural space.

But such cases are after all exceptional. By far the most common cause of pneumothorax is the giving way of a superficial vomica in phthisis. Walshe estimates that nine out of ten cases arise in this way, and Fräntzel carries the proportion still higher, putting it at fourteen to one. It would occur very much oftener than it does but for the adhesive pleurisy, which generally advances *pari passu* with chronic pulmonary disease, and slowly seals up the serous cavity. Indeed, even when there is no evidence of phthisis either before or afterwards, it is a question whether the spontaneous development of pneumothorax, independently of any violent muscular effort, should not generally be attributed to the rupture of a small tuberculous cavity, by which neither physical signs nor symptoms have been produced. A case in point was related by Prof. Vogel, of Dorpat, in the second volume of the 'Deutsches Archiv.' A woman, aged twenty-nine, became suddenly the subject of pneumothorax one morning at nine o'clock. All that could be made out as to its possible causes was that some months previously she had a slight loose cough, and more recently a little pricking pain in the region of the liver; when the attack began she was engaged in turning over her child's bed, and just before she had been lifting its bath, which was rather heavy. Vogel himself was inclined to think that she had had latent tubercular disease.

Another way in which pneumothorax may arise is by perforation of the visceral pleura from without, as when an empyema discharges itself through the air-passages. In medical practice this cause comes next to phthisis in order of frequency. Perhaps it is also possible for pleurisy to give rise to pneumothorax at an earlier stage, if the inflammation is sufficiently intense to lead to sloughing of the visceral layer of the serous membrane. Thus, in 1869 a man, aged forty-two, was brought into Guy's Hospital with a very severe chest affection, under which he was said to have been labouring for a fortnight; he died half an hour after his admission. He was found to have acute pericarditis, mediastinal inflammation, and early pleurisy on the left side. But the principal seat of disease was the right pleural cavity. This contained fœtid gas, and four and a half pints of dirty purulent fluid. In the upper lobe of the lung there were two openings, and through these air had doubtless entered. But the pulmonary pleura was gangrenous over an area of two square inches, and the substance of the lung beneath it to a depth of half an inch, the affected part being bounded by a yellow border. That the pleurisy was of exceptional severity was also evident from the fact that there was suppuration outside its parietal layer, involving the intercostal muscles.

There still remain cases in which the air is not derived from the lung at all, but directly from the outside of the chest, or from some part of the alimentary canal. As a consequence of perforation of the thoracic walls, pneumothorax is scarcely ever seen by physicians except when an empyema has broken through spontaneously, or has been let out by operation. And when the pus points of its own accord, the channel by which it reaches the surface is commonly oblique and indirect, so that air fails to find its way along it. Dr Moxon has drawn attention to the possible occurrence of double pneumothorax as the cause of death after tracheotomy, subcutaneous emphysema extending down from the wound so as to fill the mediastinal

connective tissue with air, which then bursts into both serous cavities. One such case occurred in a woman, aged thirty-three, who died in less than twenty-four hours after the operation. Emphysema had spread over the neck, chest, and arms as far as the fingers. Both lungs were found collapsed and almost airless.

The part of the alimentary canal which is most often the starting-point of pneumothorax is the œsophagus; a malignant growth may eat its way into the pleural cavity, or the ulceration due to a foreign body may have a like result. But sometimes a gastric ulcer, after setting up a circumscribed hypophrenic abscess, has led to perforation of the diaphragm; and a hydatid cyst of the liver has been known to open communications in two opposite directions, with the bowel below, and with the pleural space above.*

Anatomy.—The recognition of pneumothorax is not always a perfectly simple matter, even in the dead body. In making an autopsy, at the moment when the knife is first plunged into the thorax, the air can sometimes be heard to rush out; or, if a puncture is made with a trocar, it may escape in a jet, so as to blow out a lighted match. But this occurs only

* *Physical causes of pneumothorax.*—Few hospital physicians can have failed to meet with cases difficult to explain, in which communication of the pleura with the outer air, through the parietes of the chest or through the lung, has not been followed by pneumothorax. In most cases, pleural adhesions at or in the near neighbourhood of the perforation afford a probable account of the difficulty, and their presence has been often confirmed after the patient's death. But certain cases seem incapable of this explanation. In a lecture reported in the 'British Medical Journal' for June 4th, 1887, Dr Goodhart—after some instructive observations on the fact that pleuritic effusion may be "held up," so to speak, over the lung instead of gravitating to a uniform depth in the bottom of the cavity—proceeds to comment on the rarity of pneumothorax after fracture of the ribs, particularly in young patients, and on the free re-expansion of the lung even after a large opening has been made into the chest by paracentesis; and seems to think that there must be some forces at work to allow of the lung becoming inflated again by inspiration different from those known to physics. Mr Godlee replied in a subsequent number of the same journal from the surgical point of view, arguing the expediency of excision of the ribs.

The subject was dealt with by Dr Samuel West in his interesting Bradshaw Lecture before the College of Physicians (*ibid.*, August, 1887). He showed experimentally that there is considerable power of cohesion between opposed surfaces of serous membrane, and attributed to this cause the fact that pneumothorax does not occur so readily as one might have supposed. There can, however, be no doubt in the mind of any physiologist who has seen the unfailing and immediate collapse of the lungs which follows a free incision into the pleura in the case of an animal, that when the pressure on the inside and outside of the lung is equal it at once shrinks to the bulk which the elasticity of its tissue permits. This is proved experimentally by Donder's "schema," and by a similar arrangement of the dead thorax with manometers to gauge the pressure. The terrible result of tapping a healthy pleura when the other lung is incapable of expansion (cf. *supra*) confirm the conclusions derived from theoretical considerations and from more frequent and harmless experiments.

Apart from any mechanical obstacle to separation of the two layers of pleura, either from adhesions or from their natural cohesion, it is obvious that when a pleuritic effusion is tapped and runs out, the intrathoracic pressure must be greater than the barometric pressure of the air at the time. When it becomes greater with expiration, the pus or serum runs with a jerk; when it lessens with inspiration, the flow lessens also; when the pressure on the chest becomes negative, the flow ceases; or, if there is not enough fluid to close the trocar, air is sucked in. But when the orifice is closed, after a little air has been admitted no more will enter, and that already in the pleura will be quickly dissolved by the remaining fluid or by the lymphatics of the pleura—first the oxygen, then the nitrogen and carbonic dioxide. As the air is thus absorbed, the pressure in the pleura diminishes and the lung again expands. Even if a large opening is made, the lung is not emptied of air,—it is only reduced to the condition observed after the thorax is opened, after death from some disease which has not affected the thorax. There is no carnified (*i.e.* completely airless) lung to be re-expanded, unless pleuritic effusion has compressed it for a long time. As soon as the pressure on its surface becomes a little less than that in the trachea, the compressed lung will begin to expand again, if not mechanically prevented by thick adhesions.

when its pressure is greater than that of the atmosphere, which is by no means generally the case. In all probability the existence of air in the pleural space is very often overlooked in ordinary *post-mortem* examinations, especially in the bodies of phthisical patients, in whom, from their having extensive adhesions, the collapse of the lung has been only partial. The best way of making sure whether there is pneumothorax or not is to puncture the chest under water, which may be done either by dissecting off the tissues from the ribs so as to form a pouch that can be filled with water, or by pouring water into the abdomen and then perforating the diaphragm with a trocar. Or, if pleuritic effusion is present, it may be sufficient to shake the body before opening the chest, after which, if there is any air, the liquid will be frothy.

When the pneumothorax arises from perforation of the visceral pleura, the aperture by which the air entered is sometimes plainly visible; it may be as large as a threepenny piece. Much more often it is covered by recently formed lymph, and the only way of detecting it is to inflate the lung with bellows through the trachea. Or it may have become completely closed by adhesions during the interval that has elapsed between the occurrence of the pneumothorax and the death of the patient, so that there may be no possibility of discovering its position. It is most commonly situated upon the lateral surface of the lung, in the upper lobe near its lower border or in the lower lobe near its upper border.

The chemical nature of air withdrawn from the pleural space was investigated by Dr John Davy many years ago,* and analyses have since been made by other chemists; it has always been found to consist mainly of nitrogen, and the amount of carbonic acid in it has generally been greater than that of the oxygen; sulphuretted hydrogen has been present when the other contents of the cavity were putrid. Dr Walshe explains the difference of this pleural from the atmospheric air by pointing out that it traversed the lung before reaching the pleura. But it can hardly be said to have passed through pulmonary tissue, and, moreover, its composition is far more altered than that of normally expired air. Obviously, therefore, it must have undergone change while in the serous space, either as the result of the solvent action upon it of liquid effusion, or in consequence of the absorbent energy of the pleural membrane (*i. e.* of the lymph which fills its stomata and lymphatics, and of the subpleural veins), which is very considerable, as we shall presently see.

The secondary pleurisy.—When death occurs within a few hours after the development of pneumothorax, the cavity of the pleura is of course found empty, there having been no time for the occurrence of effusion. But in other cases, at least such as are seen by physicians, an empyema is, as a rule, formed within a few days. Dr Walshe has even discovered signs of liquid effusion within twenty-four hours. A most striking instance to the contrary is afforded by Vogel's case, already quoted (p. 190). He repeatedly examined his patient during the month after she was attacked, and could never detect the slightest indication of pleurisy.

Even when effusion does take place it is not always purulent. Fräntzel speaks of sero-fibrinous exudations as of not infrequent occurrence. In one patient the pleural space gradually became full of fluid without any pyrexia developing itself, until there was no longer any pneumothorax; a puncture showed that the fluid was sero-fibrinous. In another case the same thing happened notwithstanding that a large opening through the lung kept up

* 'Phil. Trans.,' 1823; and in his collected 'Researches,' vol. ii, p. 249.

during a period of three months a free communication between the serous cavity and the external atmosphere. So far as the air itself is concerned, one must indeed suppose that its power of setting up inflammation depends upon its containing septic bacteria or their germs. But, as we have seen, pneumothorax is, in a very large majority of cases, due to the rupture of a phthisical vomica into the serous space. The contents of the vomica must generally escape with the air, and they may well be regarded as the cause of the pleurisy which follows. When the original affection is a sloughing block in the lung, or when the pleura is perforated by a malignant œsophageal growth, or by a hypophrenic abscess communicating with the stomach, the consequent inflammation is peculiarly severe and rapid in its course. On the other hand, in surgical practice, when a healthy lung is wounded by fractured ribs, we are told that pleurisy is often absent. Probably very much depends upon whether or not the aperture in the lung becomes quickly closed again. For the risk of the entrance of germs must be greatly diminished if no air is admitted beyond that which immediately fills the serous cavity.

It is clearly impossible for subcutaneous emphysema to be produced by fracture of the ribs without there being also pneumothorax, unless the pleural space at the seat of injury happens to have been closed by former adhesions. But in cases of this kind we have often failed to detect any signs of the presence of air in the serous cavity when a day or two had passed before an opportunity of examining the patient was afforded. So that air must often disappear from the pleural space by absorption very rapidly. This conclusion is quite in accordance with the results of experiments on animals. Cohnheim says that in rabbits it is not possible by injection of air into the pleura to cause compression of the lung, so as to study the effects of that condition, because the air is so quickly absorbed. Exceptional instances of pneumothorax occur in medical practice in which a lung, previously healthy, is ruptured during a straining effort, or in a paroxysm of whooping-cough.

Locality.—Dr Walshe states that of eighty-seven cases of tuberculous perforation of the lung collected by him from various sources, fifty-five affected the left and thirty-two the right pleura. But among twenty-six cases of pneumothorax extracted from the *post-mortem* records of Guy's Hospital without selection, the number on each side of the chest was exactly equal.

Physical signs.—Clinically, it depends upon a variety of circumstances whether pneumothorax is easy or difficult of diagnosis. The recognition of this affection must always be based directly upon the results of a physical examination of the chest, although we shall hereafter see that the patient's symptoms, and the way in which they develop themselves, often enable one to form a shrewd guess as to the real nature of the case. As regards physical signs it should be a fundamental rule that pneumothorax is to be suspected whenever, over a large part of the chest, but on one side only, a marked deficiency or absence of vesicular murmur is associated with an alteration of percussion-sound in the direction of over-resonance or of tympanitic quality.

Enfeeblement or absence of vesicular murmur is a very important indication of the presence of air in the pleural space; and sometimes the sound

which accompanies the breathing gives one an impression of being conveyed from a distant part of the chest. But in many cases there is marked amphoric breathing. Sometimes, no doubt, this is due directly to the passage of air backwards and forwards into the serous cavity; but it is often present when the aperture is closed and when, as Dr Gee remarks, it must in some way acquire its peculiar quality by transmission through the pneumothorax.

The voice may either be less audible than on the healthy side, or it may be conveyed so as to produce bronchophony or even pectoriloquy. As a rule, vocal fremitus is either absent or greatly diminished.

The percussion-sound may be "altered in the direction of over-resonance or of tympanitic quality." When air escapes into a healthy pleural sac, the sound is, as a rule, purely tympanitic. But if the air should accumulate so as to cause extreme distension, it may, in the words of Dr Walshe, at length become "muffled, toneless, almost dull," like that of a drum tightened to the highest possible point, and with all escape of air from its cavity prevented. Much more frequently, the reason why the percussion-sound in pneumothorax is imperfectly tympanitic is that the pleura itself is thickened; in all probability not only do the chest walls themselves fail to vibrate, but they are even incapable of transmitting the sound made in percussion to the air within, so as to throw it into anything like free vibration. In such cases one may obtain any one of the modifications of percussion-sound mentioned at p. 80—osteal, tracheal, or subtympanitic.

There still remain certain physical signs of pneumothorax, which when present are extremely striking and characteristic. They are therefore apt to impress inexperienced auscultators with an undue sense of their importance, for they must after all be regarded as accidental rather than as essential indications of the existence of pneumothorax. They may be grouped together as "metallic" phenomena.

By Laennec most of them were included under the name of *metallic tinkling*,* a sound which he compared with that "produced in a metal cup, or in one made of glass or of porcelain, by gently striking it with a pin, or by dropping into it a grain of sand." He described it as being heard when the patient either breathed, or spoke, or coughed. There was afterwards much discussion as to the origin of this sound, but writers seem now to be generally agreed that the main cause of it is the bursting of bubbles of fluid in a large space, which is filled with air and has a smooth surface. In other words, metallic tinkling is a moist sound, or râle, modified by the vibrations of the walls of a cavity of great size, and by those of the air contained in the cavity. One way in which the bubbling necessary to give rise to such a sound may be produced was noticed by Laennec himself; namely, by the dropping of liquid from the upper into the lower part of the pleural space when it contains air as well as pus. Thus metallic tinkling may be due to the patient's changing from the recumbent to the sitting posture; but one is hardly likely to hear this unless one has the stethoscope applied to his chest before he begins to move. Again, there is no difficulty in understanding how the sign may be produced by coughing, as well as by drawing in the breath, especially if there be a free communication between the space in which it is found and a bronchus.

But it does not seem clear that it can arise as a mere result of speaking. What is heard under these circumstances is rather an *echo* of the

* *Fr.* Tintement métallique—*Germ.* Metallklang.

voice, which acquires a metallic quality from the conditions under which it is produced. So also, the heart-sounds, and even the sound produced by percussion of the chest, may be reverberated with a similar character. To these phenomena it would be better to give the name of "metallic echo;" reserving that of "metallic tinkling" for sounds which in their origin resemble râles. Coughing, as is obvious, may either be attended with echo, or with tinkling.

A particular kind of metallic echo has been specially described by Trousseau under the name of *bruit d'airain*. Among all metallic phenomena it has the advantage of being completely under the control of the observer. Metallic tinkling is well known to be exceedingly capricious, accompanying certain respiratory movements, and being absent with others, according as bubbles happen or do not happen to burst. Even a metallic echo of the patient's voice may probably fail to be heard unless he speaks distinctly and with a certain pitch and loudness. But the *bruit d'airain* can not only be determined as to the time, but the sound which is to produce it can be varied, until one obtains the best possible result. The method of eliciting it was originally given in the 'Gazette des Hôpitaux' for 1859. It consists in applying one's ear to the back of the patient's chest, while a third person strikes the front of the chest, either with the hammer upon the plessimeter, or with one coin upon another. The metallic echo which results is sometimes wonderfully distinct, and there are probably few cases of pneumothorax in which it is absent. Traube, however, has pointed out that one may sometimes fail to obtain a metallic echo by percussion during life, and yet have no difficulty in eliciting it from the dead body of the same patient. He attributes this fact to lowering of the tension of the air in the pleural space, as the result of *post-mortem* cooling of the tissues.

Another sign of pneumothorax which is of considerable practical, and of extreme historical interest, is that which is termed the *succussion-splash*. It was well known to Hippocrates, so that it is sometimes spoken of as "Hippocratic succussion."* To obtain it, one may shake the patient's body while one has one's head pressed against his chest. But sometimes it can be heard at a little distance off, and the patient himself may be conscious of it every time he makes any abrupt movement, as in stepping downstairs, or in riding on horseback. It is literally nothing else than the splashing of pleuritic effusion against the sides of the serous cavity, and of course it is never audible unless there is liquid present as well as air.

In such cases the signs of pleuritic effusion are of course to be observed, as well as those of pneumothorax; it may also be noted that alterations of the level of dulness when the patient changes his posture are generally very conspicuous, whereas in uncomplicated pleurisy they can seldom be made out satisfactorily.

Lastly, in most instances pneumothorax is attended with lateral dis-

* "Another malady. When the time grows long (after an inflammation of the chest has appeared), then the fever becomes higher and the cough increases, and the patient's side pains him, and he can no longer bear to lie on the sound side but only on the diseased one, and his feet swell and the hollow of his eyes. Then, when fifteen days have elapsed since the rupture (*i. e.* the bursting of an abscess into the pleura as the result of peripneumony, for that was the Hippocratic pathology of empyema), give the patient a warm bath (or, possibly, bathe the affected side with hot water) and set him upon a good steady stool. Then, while a friend holds his hands, do you shake him by the shoulder and listen, so as to tell on which side of the chest there is a splash (*ψοφέϊ—ψόφος*, is a noise, *strepitus*, as opposed to a musical or an articulate sound: it is applied to doors banging, armour clanging, and streams splashing)." Hipp., 'De Morbis,' lib. ii, cap. xvi.

placement of the heart. Dr Douglas Powell has pointed out, in vol. lix of the 'Med.-Chir. Trans.,' that the mere elasticity of the opposite lung drags the mediastinum over whenever air has free entrance into one pleural space, without there being of necessity any excess of pressure above that of the atmosphere. He remarks, however, that, in some cases of phthisis, consolidation of the lung on the side opposite to the pneumothorax prevents the mediastinum from being thus displaced, and probably a like effect is also produced by consolidation and adhesion of any considerable part of the lung on the side of the pleural affection, or, again, by the rigidity and thickening of the pleura, which so often occur in cases of empyema before perforation takes place. Thus one must not expect to find the heart beating in an abnormal position in those cases of chronic disease of the chest in which it is sometimes so difficult to determine whether pneumothorax has or has not developed itself. Yet, even in such cases, it is quite possible for the pressure of the air in the pleural cavity to become considerably increased. The way in which this is brought about is now well understood to be by the action of a piece of false membrane lying over the aperture; this plays the part of a valve, and allows air to enter the cavity during inspiration, but hinders its escape during expiration. Cohnheim, indeed, declares that for air confined in the pleural space to retain for any length of time a high pressure after closure of the opening by which it entered is impossible, on account of the rapidity with which it undergoes absorption. But however this may be, it is certain that among seventeen cases collected by Dr Powell there were twelve in which, after death, the pressure was found to be above the atmospheric pressure, the difference amounting in these cases to that of a column of from five and a half to seven inches of water. When the adjacent organs are capable of yielding to it, one cannot be surprised that the elastic force exerted by air in the pleural cavity should displace them even more than they are displaced by liquid effusion. Thus Dr Gee speaks of the diaphragm as being pushed down so that the upper surface of the liver lies altogether below the level of the anterior costal margin, and percussion yields a tympanitic sound within the ordinary confines of the abdomen. The intercostal spaces, too, may be flattened or bulging; and the affected side of the chest may be enlarged and motionless.

Diagnosis.—If it be asked what affections there are of which the physical signs may be mistaken for pneumothorax, or *vice versâ*, there is but little to answer.

The limitation of the signs to one side of the chest obviously suffices to exclude the possibility of their being due to pulmonary emphysema, which from the time of Laennec has been given in text-books as the disease chiefly needing distinction from pneumothorax, although in practice the two are not in the least likely to be confounded.

When distension of the pleura with air is very extreme, the percussion-sound may become muffled and toneless; but this probably never reaches such a point that the case could be supposed to be one of liquid effusion.

In vol. xi of the 'St Bartholomew's Hospital Reports,' Mr Butlin has recorded an example of rupture of the diaphragm, with escape of the distended stomach and colon into the left pleural cavity; it was the result of a severe crush between the buffers of two railway coal-waggons, and was diagnosed during life as traumatic pneumothorax.

But in general the only cases which are attended with doubt are those

in which, if air is present in the pleura at all, it is confined to a limited portion of the serous space. Thus at the upper part of the chest it might very likely be impossible to diagnose a localised pneumothorax from an exceedingly large vomica. It is doubtful, however, whether limited pneumothorax ever occurs in that position, and in all probability the cases that have been admitted as open to question have always been really examples of vomicæ attended with unusual signs, such as metallic tinkling or Hippocratic succussion-splash. On the other hand, at the base of the chest, a cavity within the lung of sufficient size to be mistaken for pneumothorax is a thing almost, if not quite, unknown. But it must not be forgotten that during the contraction of the sac of an empyema the diaphragm, with the stomach below, may on the left side be drawn upwards so far that percussion may yield a tympanitic sound over a considerable area, when complete dulness might otherwise have been expected. A similar condition may also arise when the lung is affected with cirrhosis. Probably one might avoid an error of diagnosis by re-examining the patient after having made him swallow a large quantity of fluid. In a case of Wintrich's a subdiaphragmatic abscess, which arose from a perforating ulcer of the stomach, and consequently contained air, was mistaken for pneumothorax.

It is necessary to bear in mind the fact, mentioned at p. 134, that in some instances the percussion-note is tympanitic over a part of the lung affected with pneumonic hepatisation. For whenever this is observed the suspicion cannot but arise that there is air in the pleural cavity.

But, after all, the mistake which is most apt to be made in regard to pneumothorax is not that it is taken for any other affection or any other affection for it, but that its presence is overlooked. This is due to the fact that the symptoms of pneumothorax, which are sometimes of the most striking character, are sometimes altogether absent.

Symptoms.—The amount of dyspnœa produced by the escape of air into a pleural sac depends upon two conditions ; first, upon whether the patient's vital functions are or are not being actively carried on at the time ; and, secondly, upon whether he has or has not been accustomed to make full use of the lung on that side in breathing. A healthy person always experiences great distress when attacked with pneumothorax. Among those who are the subjects of disease the distress is greater in proportion as they are well nourished and able to take food and to bear exertion. It is also greater among those who have chronic pulmonary disease, in proportion as the lung on the side of the pneumothorax took a greater share of the work of respiration before the supervention of the accident. It accordingly reaches its maximum when a man who has one lung extensively diseased, but whose health is nevertheless pretty good, becomes attacked with pneumothorax on the opposite side. A directly fatal result is then inevitable. And thus pneumothorax has to be remembered among the possible causes of sudden death in persons who are walking about and earning their living. One morning, in the year 1874, there was brought into Guy's Hospital the body of a man who had fallen dead while on his way to his work ; he was found to have pneumothorax on the right side, and chronic tubercular disease of the lung on the left side.

On the other hand, if air escapes into the pleura of a person who is wasted, and whose functions were already at a low ebb, and especially if the lung on that side had before been rendered almost useless by advanced tubercular mischief, the supervention of the pneumothorax may give rise to

no symptoms whatever. This fact was pointed out in the 'Medical Gazette' for 1844, by Dr Hughes, who was one of the best auscultators of that day.

But even in persons who are already in the last stage of phthisis, it is possible for pneumothorax to produce a shock that may be directly fatal. The patient is perhaps found dead in bed without anything having occurred to attract the attention of the nurse.

Between the two extremes described in the last paragraph there are all degrees of severity in the symptoms of pneumothorax. The most typical cases are those in which the patient is suddenly seized with an agonising pain in the side, and has a sensation of something having given way or even of a stream of air or of fluid trickling down within his chest. His dyspnœa is extreme; the respirations may reach forty or even sixty in the minute, while the beats of the heart, although accelerated, are not so to any proportionate extent. The pulse is small, the radial arteries being imperfectly filled as a consequence of the deficient flow through affected lungs. The hand, the foot, the cheeks, the lips, and the visible mucous membranes become cyanosed; the extremities and even the tongue feel cold; a cold sweat breaks out over the body; the temperature, even in the rectum, falls considerably. The voice is weak, and may even be altogether toneless. There may be complete inability to cough. The patient is usually obliged to sit up in bed; sometimes he finds it more comfortable to incline towards the affected side, sometimes towards the healthy side.

Prognosis.—In some cases of pneumothorax the symptoms continue with unabated severity until the death of the patient, which may take place after a few hours, or in a day or two. But in other cases they subside as the shock of the accident passes off; the breathing may remain rapid and yet the patient may experience little or no distress, as in a case in which Dr Walshe counted the respirations at fifty-two in the minute. In some very exceptional instances the air gradually undergoes absorption, and complete recovery takes place. Vogel's patient, referred to at p. 190, got quite well within four weeks, being from that time able to work as well as ever.

But, as we have already seen, what usually happens is that after a few days pleurisy sets in. Even then it is not impossible for the disease rapidly to subside. Dr. Walshe says that he has seen two cases in which in the course of two months all signs of air and fluid in the pleura disappeared; in all probability the exudation was sero-fibrinous. As a rule, when an empyema is developed, one can give the patient a chance of recovery only by making a free external opening, so as to allow the cavity to become obliterated by the process of granulation. The same treatment is of course necessary when the entrance of air into the pleura is secondary to pleurisy. But in cases in which there is already advanced phthisis it is scarcely ever right to perform such an operation, as the resulting inflammation of the pleural space is apt to assume a putrid character, so as to carry off the patient very rapidly. On the other hand, Czernicki pointed out ('Gaz. hebd.,' 1872) that in some phthisical patients the supervention of pneumothorax with consequent spontaneous pleuritic effusion actually leads to an improvement in the general symptoms and to cessation of expectoration, effects which can only be ascribed to anæmia of the affected lung, resulting from its collapse. As a rule, however, death occurs within two or three weeks after perforation of the pleura. Traube insisted on the rapidity with which

emaciation advances in many cases. Œdema of the limbs, and even of the face, sometimes develops itself. The urine may be scanty and sometimes albuminous.

On the other hand, it is surprising how long a pyo-pneumothorax is sometimes tolerated, and how little discomfort it causes. The patient is sometimes able to take horse exercise and thus, as already mentioned, may hear the fluid splashing within his chest while he is riding.

Traube relates a case of pneumothorax occurring in a woman who had been attacked by it some years before he first saw her, and in whom seven years later scarcely any physical signs were discoverable. She looked well, and could even walk uphill without discomfort. The history appeared to indicate that the affection arose as a complication of phthisical mischief in the lung; she had previously had a febrile illness, with night sweats, cough, expectoration of yellow matter, and hæmoptysis.

Treatment.—In cases of pneumothorax a great deal can be done to diminish the patient's sufferings and even perhaps to avert a fatal termination. Cupping, dry or wet, often gives remarkable relief, and venæsection is probably still more efficacious. A small dose of morphia should be injected subcutaneously, or, as Dr Walshe recommends, a very little chloroform may be given by inhalation from time to time. He also says that he has seen musk in five-grain doses afford much relief.

If great enlargement of the side and depression of the diaphragm suggest that the pressure of air within the thorax is greater than the atmospheric pressure, paracentesis should be performed with a fine trocar. But displacement of the heart alone is not evidence of increased pressure; it may be due to the elasticity of the mediastinal tissues. Fräntzel appears to have tapped the pleural cavity rather frequently; he speaks of a dissertation by Bärensprung in which are recorded a number of his cases treated in this way with success. He says that, if possible, it is well to postpone the operation until three or four days have elapsed, so that the aperture in the pleura which allowed the escape of the air may have closed. Between the fourth and the eighth day it may be advisable to introduce a trocar, even when the symptoms are not very urgent. He has often found the pressure of the air such that a considerable quantity passed out through the instrument; and even when this is not the case, it is easy to close the wound, and no harm is done. An aspirator ought never to be employed, on account of the risk of reopening the original aperture. Fräntzel uses an ordinary trocar with a valve of gold-beater's skin. If cough arises during the operation, which is not uncommon, he either gives an injection of morphia and waits for a time before withdrawing the trocar, or else he keeps up pressure upon the seat of puncture for a little while afterwards until the cough has ceased. By either of these methods the escape of air along the track of the trocar may be prevented, which would otherwise lead to subcutaneous emphysema.

PHTHISIS*

History and definition—Pathology—Unity of phthisis—Localisation—Phthisis always tubercular—Histology: miliary, caseous and infiltrating tubercle—Tubercular pneumonia and ulceration—Vomicæ—Adhesions: involution—Symptoms: wasting, pyrexia, cough, sputum—The bacillus—Hæmoptysis—Physical signs of the three stages and of involution—Diagnosis—Course—Mode of death—Prognosis—Ætiology—The question of contagion—Hereditary taint—"Diathesis"—Conformation—Overcrowding, &c.—Inhalation of dust and other irritants—Damp soil—Treatment: preventive, curative, and palliative—Diet and hygiene—Climate—Drugs.

From an early period in the history of medicine it has been known that progressive loss of flesh is often dependent upon a destructive affection of the lungs. And thus, among the various forms of wasting disease, *phthisis pulmonum* has ever held the chief place. But the other affections which were once included under the same generic designation now have names of their own. Consequently phthisis, with its English equivalent "consumption," has come to mean a chronic *pulmonary* disease only. This term is now applied even to such exceptional cases as happen not to be attended with emaciation; and, on the other hand, it is never used for cases in which the lungs are believed to be healthy, even though the bodily tissues may be reduced to the utmost.

There is, however, a secondary meaning of the word. Many pathologists hold that in all cases of phthisis the affection of the lungs is tuberculous. This doctrine, which dates from Laennec, has led to the occasional employment of such expressions as "renal phthisis," "intestinal phthisis," "laryngeal phthisis," to imply that the kidneys, the bowels, or the larynx present tuberculous lesions. It will be observed that such a use of the term is altogether different from that which long ago prevailed, when other forms of phthisis beside pulmonary were recognised. For the old idea was that bodily wasting might be due to affections of various organs other than the lungs, whereas Laennec's view was that other organs are liable to the same lesions which, when they occur in the lungs, constitute phthisis. But to make the clinical term phthisis synonymous with the histological term tuberculosis is most undesirable.

Phthisis one disease.—The appearances presented by the diseased lungs in cases of phthisis differ exceedingly; and its clinical symptoms and course are subject to no less wide variations. One cannot be surprised, therefore, that both pathologists and physicians have strenuously endeavoured to discover points which might serve to split it up into several diseases, fundamentally distinct from one another. Addison led the way in this

* *Synonyms.*—Phthisis pulmonalis—Tabes pulmonum—Consumption of the lungs—Decline. *Germ.* Lungenschwindsucht, *Fr.* La phthisie. Φθίσις (from φθίνω, I waste = *tabes*, consumptio) occurs frequently in the Hippocratic writings.

direction by insisting that much of what was commonly regarded as tubercular disease in the lungs was in reality pneumonic, and that softening of the organ with excavation of its substance might occur without any tubercle being present. But it is to be observed that he was very far from maintaining that an absolute distinction could be drawn; in his well-known essay, read before the Guy's Hospital Physical Society in 1845, he described first a "pneumonic" and then a "tuberculo-pneumonic phthisis;" and the final sentence of this work is, that "in every form of phthisis, inflammation constitutes the great instrument of destruction." Thus, after all, it may be said that Addison's teaching bore upon the theoretical question whether tubercles should be regarded as distinct from what Laennec used to term tubercular infiltrations quite as much as upon the practical question whether a separate kind of phthisis is to be recognised apart from the tubercular. Since his time many pathologists have asserted, in the most positive and dogmatic manner, that a "catarrhal" or a "caseous" pneumonia is the essential morbid change in many, if not in most, cases of phthisis. Another form which has also been declared non-tuberculous is the so-called "fibroid phthisis." But there is no real consensus of opinion among the most advanced histologists with regard to these questions. Rindfleisch, who at one time maintained that the "tubercular granulations of Laennec" consisted in an inflammatory infiltration of the alveolar parenchyma round the smallest bronchi, now teaches (in 'Ziemssen's Handbuch') that they are true tubercles.

For my own part, I believe that all ordinary cases of phthisis are essentially of the same nature. The varied appearances which may be found in the lungs after death seem to me to depend mainly upon whether the tubercles and the tuberculous infiltration become caseous or undergo fibrous changes. This, to a great extent, rests upon the degree of rapidity with which the disease has advanced during life. Thus, pneumonic phthisis is generally equivalent to phthisis which has advanced quickly; fibroid phthisis to one of which the course has been slow.

The decision of this question would seem to rest upon whether the tubercle bacillus (vol. i, p. 86) is or is not present in cases of phthisis supposed to belong to different forms. In his earliest communication on the subject Koch stated that he had found bacilli in twelve cases of caseous bronchitis and pneumonia; and these appear to have been all the examples of that variety of phthisis he had then examined. In fibroid phthisis one would doubtless have to look for them in the parts of the lungs most recently affected. For in the caseous form of the disease they were generally limited to the edge of the infiltrated tissue, where, however, they were very abundant. Sometimes nests of bacilli were met with, even in the interior parts of the lungs which had undergone infiltration. In most vomicæ they were present in great numbers. The little cheesy fragments, which are so commonly found in vomicæ, consist, according to Koch, almost entirely of masses of bacilli. It is also worthy of notice that, after speaking of the presence of the bacillus in the "Perlsucht" of cattle (cf. vol. i, p. 90) Koch goes on to say that he also detected it in cases in which there were round smooth-walled nodules filled with a cheesy pulp, such as are not generally reckoned to belong to Perlsucht, but are regarded as due to bronchiectasis. It would thus appear that pathologists have too narrowly confined the conception of tubercular diseases of the lungs, in animals as well as in man.

In discussing the subject of tubercle in general (vol. i, p. 79) I endea-

voured to show that histology opposes no insuperable difficulties to the doctrine of the unity of phthisis; but my own opinions with regard to phthisis have been based not so much upon microscopical investigations as upon the results of careful study of the appearances seen in the *post-mortem* room of Guy's Hospital during a long period of years. And I cannot help thinking that any unprejudiced observer would inevitably be driven to the same conclusion. Without wishing to detract from the importance of histological inquiries, I am under the impression that the practice of setting aside minute fragments of diseased organs for study at a future time, when the general morbid anatomy of the case has been forgotten, is very apt to lead to one-sided and partial views. What I have found is, that in the same body lesions which would be universally admitted as tubercular are associated inextricably with other lesions, of which the tubercular nature would by many pathologists be denied.

Locality.—Phthisis affects both lungs. Clinically we constantly meet with cases, particularly early cases of consumption, in which the physical signs are confined to one side of the chest; but, as the disease advances, the opposite side becomes also involved. Occasionally too we find clinical evidence of obsolete phthisis, or even of a large vomica and greatly thickened pleura on one side, while the other shows no traces of past disease. But the evidence of autopsies proves that it is the rarest event for a patient to die of phthisis with one lung only affected. Nevertheless the disease is not perfectly symmetrical; it is almost always earlier and more advanced in one lung than in the other. It is much more symmetrical than pneumonia or cirrhosis or pleurisy, rather more so than tubercular disease of the testes or adrenals, and less symmetrical than chronic tubal nephritis or than psoriasis.

It has long been known, both to physicians and to pathologists, that the upper parts of the lungs are almost invariably affected with phthisis, in whatever form, before the lower parts; and that in all but the most exceptional instances, the disease spreads downwards from apex to base, often with almost perfect regularity.

It is difficult to find a satisfactory explanation of this proclivity of the upper lobes of the lungs to phthisis. The same thing is observed in miliary tuberculosis, in which disease the pulmonary affection is believed to be due to an affection of the tissues at a number of different points through the blood-stream. This fact is opposed to Dr Hamilton's view, expressed in the 'Practitioner' for 1880, that the proclivity of the apices depends upon their being the driest parts of the lungs, so that caseation of catarrhal products is more apt to take place there than elsewhere. He also maintains that there is less expansion of the apices during breathing, and that catarrhal products are consequently more likely to accumulate in them than in other parts of the lungs. Almost exactly the same line of reasoning is adopted by Rindfleisch in 'Ziemssen's Handbuch.' He insists that the upright position of the body in man and in the Quadrumana causes the weight of the shoulders and arms to fall upon the upper ribs, and so interferes with their play and leads to a deficiency in the movement of air in the apices as compared with that in the lower lobes. On the other hand, it is certain that the proclivity of the apices is no greater in men than in women, who use those parts of the lungs far more than men do. According to Dr Moxon the regions which become the earliest seats of tubercle in persons who are confined to bed are the anterior edges, a fact for which he found an

explanation in the supposition that under such circumstances these parts are more, not less, active than any other in the function of respiration.

The general rule of the proclivity of the apex is liable to some other exceptions. In certain cases the tubercles appear a little lower down, leaving one or two cubic inches at the extreme summit of the upper lobe free from them. But sometimes the middle of the organ is first affected, or even the lower lobe, the upper angle of which is frequently the seat of a vomica in ordinary phthisis. But the tubercular process seems never to spread upwards from the base of a lung into and through the upper lobe. And it is certain that what has sometimes been called "basal phthisis" is a distinct affection, which has been described above under the name of "chronic pneumonia" or cirrhosis of the lung (*supra*, p. 152).

Phthisis tubercular.—According to what has been stated in the preceding paragraph, we shall scarcely ever be wrong, when we find a lung extensively affected with phthisis, in assuming that the disease is oldest at or near the apex, and that its most recent stage is situated towards the base. Now, a very common appearance is that the upper lobe presents a dense fibroid mass (perhaps containing more or less numerous cavities), that in the middle of the organ there are cheesy patches, and in the lower lobe grey tubercles, scattered or in groups. Or, again, the affection in one lung may appear to be typically fibroid, or typically pneumonic, throughout; yet in the opposite lung, in which the disease is of more recent origin, there may be clusters of tubercles; and these may themselves be caseating, whatever the character of the change in the organ first affected. Lastly, the pulmonary lesion may appear to be pneumonic, or to be fibroid, not a single tubercle being discoverable, even in a state of caseation; yet in some distant part of the body there may be tubercular lesions of the most characteristic description. What is most frequent is for the small intestine to present the transverse hard-edged ulcers, with tubercles in the peritoneum beneath and around them, of which the nature is so unmistakable.

There are other facts, all of a similar kind. Thus in 1876 I examined the body of a girl, aged sixteen, who died of what was regarded as pneumonic phthisis; in one kidney, traversing its cortex from the surface to the medulla, was a single linear tuberculous mass. In 1878, in a case of pneumonic phthisis, in which the affected part of the lung showed only a cheesy infiltration breaking down into sinous cavities without any distinct walls, there were not only small caseating points and ulcers in the intestine, but in the liver several tubercles as typical in their characters as it would be possible to conceive. So, again, in 1876 I made an autopsy upon a child, aged six, whose lungs presented a remarkable example of fibroid phthisis; in her intestine there were a large number of ulcers, with most abundant subserous tubercle. In 1879 I examined a typical case of fibroid phthisis of the apices of the lungs, with indurated tubercles lower down; in the kidneys and the prostate of the same patient there were caseating vomicæ. In 1878, in a woman of thirty-three, I met with an instance of phthisis, consisting mainly in grey induration of the pulmonary tissue, there being very little tendency to caseate; both adrenal bodies contained cheesy nodules, and there were yellow tubercles in the liver.

A few years ago it might have been objected that some of these cases

were useless as evidence, inasmuch as tubercles, whether in the lungs or in other organs, when they appeared to be of more recent origin than a pneumonic or a fibroid phthisis, could be supposed to have arisen secondarily from it by a process of infection. In particular, some pathologists thought that tubercular ulcers in the small intestine were frequently caused by swallowing matters expectorated from the lungs, when the disease in these organs was itself of a non-tubercular kind. But as soon as it is proved that the infection of tubercle depends upon the presence of a specific virus, all such objections fall to the ground. It must, however, be observed that many of the instances cited in the previous paragraph fail to adjust themselves readily to any infective theory. When, in a case of phthisis, an isolated tubercle, or a cheesy mass, or a vomica, is found in some distant organs, such as the kidneys, or the liver, or even the testis, it is more natural to suppose that it arose independently and by a repetition of the same process which caused the pulmonary lesion; and this conclusion is not invalidated by the presence of a number of tubercles in close proximity to one another in a single organ, even though we suppose their multiplication to have been due to a kind of local infection within the limits of its area.

Again, the way in which phthisis begins confirms the belief that it is at the outset a tubercular affection. In 1881 a man, aged thirty, died in Guy's Hospital of caries of the spine with psoas abscess; in the apex of each lung there were grey translucent tubercles, some scattered, some in clusters, occupying about a square inch of the cut surface; they were more numerous on the right side than on the left; the other parts of the lungs were quite free. In the same year a man, aged thirty, died of delirium tremens; in the upper parts of both lungs there were miliary tubercles in groups. In 1879, a youth of nineteen died of spinal disease, with a scrofulous kidney; in the apex of the right lung there was a single cluster of the most typical firm grey tubercles, none of which showed any tendency to caseate. In the same year a man, aged thirty, died of "sacro-iliac disease," which, although the result of injury, was accompanied with tubercular affections of the prostate, kidney, spleen, and lymph-glands; the extreme apex of the right lung contained a number of scattered grey miliary tubercles, without the slightest caseation, and with no induration of the surrounding pulmonary tissue. In 1877, a boy, aged ten, was killed by fracture of the spine; he appeared to have been strong and healthy, but in the apex of each lung there were miliary tubercles. Dr Moxon's experience, when he taught pathology at Guy's Hospital, was very similar. In 1869 a child, aged two years and three months, died of croup; in the left lung, below the apex, there were found several clusters of grey tubercles, one of them with some cheesy material in its centre. In 1867 a man, aged twenty-seven, was killed by accident, with fracture of the skull; at both apices, especially the right, there were recent miliary tubercles, in smaller or larger clusters. In the same year a man, aged twenty-two, died of typhus; in the right upper lobe there were many clusters of miliary tubercles, some already softening. In 1868 a woman, aged twenty-one, died after amputation of the thigh for disease of the knee-joint; in each apex there was early phthisis with clustered tubercles, some caseating; there was also a small vomica. Even when the appearance of an incipient pulmonary lesion is such that its tuberculous character might fairly be doubted, one may discover elsewhere morbid changes the nature of which is indisputable. Thus, in 1874, in examining the body of a girl,

aged eighteen, who had died after excision of the knee-joint, I found in the apex of the right lung a mass, the size of a marble, consisting of a cluster of yellow softening granules, which might naturally have been set down to a catarrhal pneumonia; the bronchial glands were caseating, but one of them contained the most typical grey tubercles. At the time this appeared to me to be a most instructive case.

Seat of the tubercles.—If we consider that there is scarcely a structure in the human body which is not liable to the growth of tubercles, we shall surely think it very improbable that in the lung their development should be limited to any one, rather than another of the various tissues which make up the organ. Rindfleisch, however, maintained that the morbid process in phthisis begins definitely just where the bronchioles open into the alveoli, the earliest change being a "tuberculous infiltration of all the edges and processes" which exist at these points, and which contain muscular and elastic tissues, as well as fibrous. The occurrence of such a change at the extremities of several adjacent tubes, and its extension along the walls of the tubes themselves, would no doubt account satisfactorily for the "racemose" distribution of pulmonary tubercles on which Carswell used to insist, so that the phrase "Carswell's grapes" was invented to keep it in recollection. But it has always appeared to me that Carswell's drawings illustrating this point are highly artificial and untrue to nature, and that it requires an eye of faith to perceive, except perhaps in some rare cases, anything like a "peribronchial" distribution of clustered tubercles. And Dr Hamilton, in the 'Practitioner' for 1880, describes tubercle in the lung as generally beginning in a little cellular projection on one side of an alveolus, which afterwards becomes somewhat pedunculated and hangs into the alveolar cavity. When these alveoli lie adjacent to one another it may project into all of them at once. At first it pushes before it the epithelium and even the alveolar capillaries. But soon it breaks through and destroys the alveolar wall, so that a uniform rounded mass results, in which the outlines of the original air-vesicles are barely recognisable. The cells of the tubercle may, Dr Hamilton thinks, be derived either from the connective-tissue elements of the alveolar wall, or from the endothelium of certain of its capillaries, or from both sources at once. Sometimes a tubercle sprouts from the inner coat of a branch of the pulmonary artery, starting perhaps from the endothelium, but soon involving the rest of the *tunica intima*, and almost occluding the channel of the vessel. Other tubercles lie in the course of the pulmonary lymphatic vessels contained in the periarterial and peribronchial sheaths, the interlobular septa and the deep layer of the pleura.

In some instances tubercles, scattered or in clusters, spread slowly through one lung, or even through both lungs, with little or no change in the intervening portions of the pulmonary tissue. But, as a rule, this undergoes consolidation at an early period, so that the tubercles come to be embedded in a more or less homogeneous mass. Sometimes, the substance of the lung is involved uniformly from the apex downwards, the edge of the consolidated area having a festooned outline, not unlike that of the border of a malignant new growth. Much more frequently, even when part of the upper lobe is universally affected, there are more or less numerous independent nodules of various shapes and sizes, lower down; and between and below these again, scattered tubercles may generally be seen in abundance. The character of the infiltrating material varies widely in different cases.

It may be a soft semitranslucent pinkish substance, the less recently formed parts of which are found to have passed into a state of caseation. Or it may be firm, dark, and tough, constituting one form of Addison's iron-grey induration. Or it may have a "marbled" aspect, crossed by bands and seams of well-developed fibrous tissue, and of all degrees of depth of pigmentation, up to perfect blackness. In general, the softer kinds of infiltration are associated with caseating forms of tubercle, the harder with tubercles which themselves become tough and fibrous. But there are comparatively few cases in which some cheesy masses are not to be found in one part of the lungs or another; and where the parts which were earliest affected are fibrous and of an iron-grey colour, or black, it often happens that recently involved parts are soft and yellow.

Vomicæ.—In all but very exceptional cases of phthisis the process of consolidation is followed more or less quickly by one of ulceration, leading to the formation of cavities or *vomicæ*. The tubercles themselves doubtless soften in their centres, as is the case in every other organ in which tubercles occur, and as, indeed, can often be seen in the more recently affected parts of the lungs. Sir Robert Carswell used to declare that sections of bronchial tubes with pus in their interior were very frequently mistaken for softening tubercles. In this I feel confident he was wrong. In two or three exceptional instances I have thought, on first glancing at the cut surface of a lung, that I saw tubercles, when there were really only the open mouths of swollen tubes; but an instant afterwards I have perceived my error, because the slightest pressure below has made pus well up from them in large quantities. There seems to be no reason why a vomica of considerable size should not result from the breaking down of caseous material derived by extension from a single original tubercle. But, as a matter of fact, tubercles are generally scattered too thickly to admit of such an occurrence, and there can be no doubt that formation of cavities ordinarily involves the destruction of infiltrated lung-substance as well.

One very characteristic appearance, which is exactly analogous to what may be observed in various other organs besides the lungs, is the presence of a caseous zone of definite thickness, which on one side bounds a vomica, while on the other side it is embedded in the pulmonary tissue. Such a zone is generally an indication that the affection is still actively spreading; it is one of the chief means by which vomicæ increase in size. As they enlarge, cavities originally distinct are very apt to open into one another and so to coalesce. In this way a single cavern of very irregular form may be produced. Sometimes the destructive process remains limited by the lobar septum; sometimes this becomes ulcerated through, so that both the whole of the upper lobe and a large part of the lower lobe are included in one wide open sac. Sooner or later, however, the further extension of every vomica becomes arrested; that is if the patient should survive long enough to allow time for it. The indication of this change is that the interior of the cavity ceases to be rough and shaggy with adherent portions of cheesy débris. A fibrous wall of greater or less thickness becomes developed round it, and its inner surface gradually assumes a smooth polished appearance, exactly like that of a mucous membrane.

Such smooth-walled vomicæ are often crossed by fibrous bands or *trabeculæ*. Each consists of a mass of condensed pulmonary substance, with fibrous tissue that perhaps originally belonged to interlobular septa. In all probability

some trabeculæ are remains of partitions that at one time separated vomicæ which have since coalesced. But other trabeculæ often contain obliterated branches of the pulmonary artery, and sometimes several of them can be seen to spread away from a single point situated on that side of the cavity which is nearest the root of the lung, so that their formation has obviously been the result of the resistance offered by the arterial walls to the process of ulceration. Ultimately the trabeculæ themselves often give way and rupture, and their loose ends may then be seen hanging into the interior of the vomicæ. In very large cavities a bundle of such ruptured trabeculæ may sometimes be seen, the relation of which to the pulmonary artery is at once shown if a probe is passed into that vessel from the heart. Sometimes a pervious channel persists for some little distance along the interior of a trabecula, a fact which we shall hereafter see to be of considerable clinical importance.

According to many observers of authority certain smooth-walled cavities have an entirely different origin from that just assigned to them, being dilatations of bronchial tubes, instead of being formed by ulceration. Tubes do, indeed, almost invariably open into them more or less freely, the branches of the bronchial tree possessing no such power of resisting ulceration as belongs to the arteries. The idea of regarding the cavities in question as "bronchiectases" seems to have originated with Laennec. In all probability what first suggested it was the difficulty of understanding how a vomica formed by ulceration could acquire anything like a mucous membrane. But this goes for very little, now that we know how readily such a structure can be pushed forwards over a raw surface from an edge of skin or of mucous membrane, as, for example, in the case of a rectal fistula. As a matter of fact, however, it is doubtful whether smooth-walled pulmonary cavities ever have a continuous epithelial lining. Dr Ewart, in his Gulstonian Lectures for 1882, says that this is wanting, except where there are "scattered islets of mucous membrane," the remains of "outlying bronchi intersected by the cavity wall." But Dr Hamilton, in the 'Practitioner' for 1879, declares that cavities, which one cannot accept as dilated bronchial tubes, often have an epithelium which is "most typically columnar and ciliated." This pathologist attempts to explain the occurrence of bronchiectasis, and the sinuous and irregular outlines of the cavities which he believes to be of such a nature, by referring it to the traction of bands of fibrous tissue radiating away from the sides of the cavity at different points. What has always convinced me in the *post-mortem* room that the cavities in question were really vomicæ is that one never gets an opportunity of observing the earlier stages of the process of dilatation. If the view adopted by Dr Hamilton were correct, one ought, towards the margin of the affected part of the lung, to see tubes which could still be traced on to their extremities, but the sides of which were beginning to bulge out here and there. On the other hand, what one does commonly find are all possible transitional varieties between smooth-walled cavities and unmistakable vomicæ. The former are seen towards the apex, where the mischief is of oldest date; the latter lower down where it is of more recent origin. Moreover, smooth-walled cavities often riddle the substance of a diseased lung in all directions, communicating freely with one another on every side, so that an ulcerative process must clearly have been concerned in their formation.

The contents of vomicæ vary widely in kind and in amount. When they are recent they often show masses of cheesy débris. Such masses, even if they

are at first too large to pass out through a bronchial tube, probably crumble into fragments in the course of time and are expectorated. Cavities of old date usually have pus in their interior and sometimes they are quite full of pus. This of course implies that there is no very free communication with the bronchial tubes. And the fact is that in old vomicæ there is a tendency for the orifices of the tubes to contract until they become very narrow. One often finds that a tube of considerable size, into which a large catheter might be passed, has an opening into a cavity that will but just admit a probe. When such is the case, a turgid condition of its lining membrane may easily block it altogether.

It is remarkable how seldom the contents of phthisical cavities putrefy; they often have a faint sickly odour, but they very rarely become actually fœtid, nor do they often undergo that peculiar acid fermentation which is so apt to arise in cases of chronic bronchitis with dilatation of the tubes. When there is free escape of pus from vomicæ their lining membranes may continually pour out fresh quantities of it, so that a large amount of expectoration may occur from day to day. But sometimes the walls are found at an autopsy perfectly dry, and the interior is quite empty; in such cases there may during life be no expectoration whatever. In 1854 Dr Bristowe showed to the Pathological Society a specimen of such a quiescent cavity having adherent to its inner surface a soft greenish powdery mass of fungus, consisting of a branching mycelium, and of a fructification, in which the spores were arranged upon rounded heads raised on thick stalks.

Adhesions.—A very constant attendant upon phthisis is a local pleurisy, which leads to the gradual closure of the upper part of the serous space on the affected side of the chest. As a rule this affection is non-tubercular, and Rindfleisch insists on the extreme vascularity of the fibrous tissue which unites the two surfaces, as contrasting with the deficiency of vessels in the substance of a fibroid lung. Not infrequently the adhesions are of enormous thickness and density, so that after death it is impossible to remove the lung without free use of the knife.

Involution.—To complete the morbid anatomy of phthisis we have still to discuss the processes by which tuberculous lesions in the lungs become obsolete, so that they cease to threaten the patient's life, or even to impair his health.

Relics of former mischief are discovered very frequently in the lungs of persons who have died at various periods of life and of every kind of disease or injury. In May, 1880, Dr Heitler, of Vienna, brought before the Medical Society of that city an analysis of all the cases of this kind that had been met with in a series of 16,562 autopsies between the years 1867 and 1879. Excluding all cases in which death was due to pulmonary tuberculosis (among which there must of course have been many other instances of a previous attack of the same affection) he found that there were no fewer than 780 (or almost exactly 5 per cent.) in which obsolete tuberculous masses were present. Of the patients 503 were males, 277 females. The number of those who died of tuberculous affections of other organs was 101. A point of great interest is that the proportion of cases at different ages went on regularly increasing for each decennial period up to sixty years of age. Among persons aged from ten to twenty there were 12; from twenty to thirty, 105; from thirty to forty, 131; from forty to fifty, 156; from fifty to sixty, 157; from sixty to seventy, 36; from seventy to eighty, 153. It is true that no positive conclusion can be drawn from this fact, in the absence of information as to

the proportion of persons at different ages in the total number of autopsies, but it is difficult to escape the inference that the time at which the pulmonary lesions were originally developed must, in a considerable number of instances, have been during adult life. In no fewer than 651 cases both lungs showed signs of past disease, though generally to an unequal extent; in sixty-eight the right lung was alone affected, in sixty-one the left.

Some observers have shown extreme reluctance to admit that such appearances really indicate the remains of phthisical disease, or that such disease can be said to be ever curable. Strictly speaking, perhaps, phthisis is "cured" only when its favourable issue has been mainly due to medical treatment, whereas in all probability the subsidence of the morbid process in the cases in question was often spontaneous.

There is a definite history of a former pulmonary affection in a case cited by Rindfleisch in 'Ziemssen's Handbuch.' It occurred in a man over fifty years of age, who died in hospital of enteric fever, fourteen years after having been treated in the same institution for an attack of serious disease of the lung, attended with hæmoptysis and with infiltration of the right upper lobe, down to the level of the third rib. He completely recovered, resumed his former occupation, and remained well until he took the fever a week before his death. The part of the lung that had been diseased was found to be indurated and shrunken, with surrounding emphysema and dilatation of bronchial tubes.

That the relics of long-past pulmonary mischief belong to the same affection which, when it goes on and destroys life, is called phthisis, ought not to be doubted, even by those who hold the narrowest views with regard to tubercle. Almost invariably the seat of such relics is in or near to the apex of the lung. The affected part is more or less indurated; it is often puckered on the surface and adherent to the chest wall. On section it presents fibrous bands, or tough masses of fibrous tissue, parts of which are generally deeply pigmented, and in which there are often embedded cheesy or calcareous nodules, of greater or less size. The cheesy nodules may look very like gummata; they are enclosed in fibrous capsules; not infrequently they are gritty from the deposition of lime-salts in them, or this process may have gone on until they have become converted into hard, smooth bodies, apparently made up almost entirely of mineral constituents. Those pathologists who hold that a caseous pneumonia is often the commencement of phthisis ought logically to accept even these appearances as conclusive evidence of the former existence of a morbid condition which, if it had advanced further, would have terminated in that disease. But in many instances one also observes grey or black indurated tubercles, which obviously have themselves long been obsolete, and which may very probably have been of the same date as the cheesy masses. When the fibrous bands have given rise to much puckering of the pulmonary tissue they often look very like cicatrices, and at one time it was actually taught that they represented former vomicæ which had undergone obliteration. But, as almost all observers seem now to be agreed, there is no proof of this and it is not likely that a pulmonary cavity is capable of thus completely disappearing. There is no doubt at all that its walls may shrink, so that in course of time it may become much reduced in size. Dr Theodore Williams has pointed out that this process of contraction of a vomica is often attended by a shifting of its position. Unless its anterior surface is closely in contact with a firmly adherent pleura, the more fixed part of its

wall is that which contains the openings of bronchial tubes; consequently it often shrinks away from the front of the lung towards the root. Dr Ewart, in his 'Gulstonian Lectures' for 1882, gives diagrams showing that the pulmonary pleura, if not too extensively fixed by adhesions, may be drawn inwards over such a receding cavity until it forms a deep chink or fissure. The space created by the shrinking of a vomica may be filled up by an enlargement of the adjacent pulmonary tissue. Even when there are merely solid relics of former mischief at the apex, without any evidence of excavation having occurred, the surrounding lung-substance is sometimes found to be highly emphysematous, the bullæ having been probably formed during inspiration, after the manner suggested by Dr Gairdner (see p. 107). More frequently, however, the lower part of the upper lobe of the lung, or (in the case of the right lung) the fore part of the middle lobe, is uniformly enlarged, so that one might perhaps almost regard it as hypertrophied. Or, if there is a considerable amount of mischief, the upper lobe of the opposite lung may increase in size until it passes across the median line. Other organs at the same time undergo displacement. The liver or the stomach is dragged upwards, according as the right or the left lung is the one which is diseased, and the heart may be pulled over either to the right, or beyond its natural position to the left. Lastly, the upper ribs are drawn inwards, so that the chest wall, especially below the clavicle, appears flattened or even hollowed.

We may conclude our account of the anatomy of phthisis by saying that one of its characteristic marks is its multiformity. In every case there is more or less bronchitis, in every case more or less pleurisy, in every case discrete tubercules, grey or yellow, and infiltrating caseous tubercle. In every case there is catarrhal inflammation ("pneumonia"), with softening and destructive ulceration, causing more or less developed vomicæ; and in almost every case there is some attempt at repair, shown by fibrous induration, contraction, and cicatrisation.

On the other hand, pleuritic effusion is rare except occasionally in the earliest stage, and empyema is still rarer. True lobar fibrinous pneumonia is a late and apparently an accidental complication; and neither abscess nor gangrene are met with. Pneumothorax sometimes occurs, and must always be remembered as a possible event. Emphysema, chiefly of the anterior edge of the lungs, is present in almost all chronic cases.

Symptoms.—The clinical recognition of phthisis, as of pulmonary diseases in general, is based partly upon symptoms, partly upon physical signs. But there is no other disease in which diagnosis depends so completely upon the concurrence of the two kinds of evidence. Symptoms alone, when no signs can be detected, may justify a strong suspicion that phthisis is present; but, unless it is confirmed by their subsequently appearing, this suspicion never reaches certainty. On the other hand, when one discovers physical signs of the disease in a person whose health appears perfect—as sometimes happens, for example, in a candidate for life insurance—the proper inference is, surely, that they depend upon a lesion which, although it was phthisical, yet is now obsolete, at least for the time. Probably physical signs never develop themselves in phthisis without symptoms being also present, although no doubt the patient may fail to notice or may wilfully conceal them. Evidently, therefore, a description of the symptoms of the disease should precede that of the signs.

The symptoms of phthisis fall into two groups. One group includes those which point directly to the lungs; the other those which concern other organs, or belong to the body as a whole.

(1) *Emaciation.*—Of the symptoms which belong to the body as a whole, one, which is very important, is progressive loss of flesh. This often occurs with extreme rapidity. Rühle mentions the case of a very bulky woman who had weighed 240 lbs., and who lost 40 lbs. in the four weeks before she came under his care for hæmoptysis, at which time no physical signs of mischief in the lungs could be detected. Ultimately phthisical patients commonly become reduced by a quarter or even a third of their weight. The explanation of the wasting is often by no means obvious. There is sometimes a great loss of appetite, and especially a distaste for fat in every form; or the occurrence of vomiting may appear to account for it. But other patients emaciate who eat well, and appear to digest what they eat. Nor does the loss of flesh seem to be constantly proportionate to the degree of pyrexia or to the amount of the sweating. It probably affects all the tissues more or less, but a point worthy of notice is that the heart becomes much less reduced in size in phthisis than in some other wasting diseases, as, for example, malignant tumours. The reason seems to be that the right ventricle has so much work thrown upon it by the destruction of blood-vessels in the diseased parts of the lungs. Among the minor alterations in nutrition which accompany phthisis are those which affect the hairs. The straight lanky whiskers and beard of consumptive patients often suggest to one the nature of their disease. The hair, too, may become prematurely grey. Dr Walshe speaks of having often noticed in males that the hair on the chest has become quite white when the change was only just beginning in the hair of the head and in the whiskers. Along with the emaciation of phthisis goes a more or less marked failure of strength and energy. The patient becomes no longer able to walk far without fatigue. If he has duties to perform they tire him in a way to which he is not accustomed. After his day's work is over he is glad to get home as quickly as possible, and to lie on the sofa until he goes to bed; and in the morning he gets up feeling weary and unfit for the labour which is before him. Anæmia is another early symptom of phthisis. The face becomes pale, the hands are white and bloodless. In women scantiness or suppression of the catamenia may be one of the first indications that the health is failing. Œdema of the ankles often occurs as the disease advances, but it is seldom considerable, unless there be venous thrombosis.

Pyrexia.—When one is consulted by a person who has thus become thin and weak, the first thing to do is to ascertain whether the temperature is raised. And it is important not to be contented with taking a morning temperature; during two or three days the thermometer should also be used at bedtime, or in the evening. One should notice whether the palms of the hand feel hot, and whether the cheeks are flushed, and the patient must be asked whether he has noticed any unusual tendency to perspire, especially in the latter part of the night. The pyrexia of phthisis is altogether atypical, and in different cases it varies widely in character and in degree. It is scarcely ever altogether absent. Dr Theodore Williams, however, in vol. lviii of the 'Med.-Chir. Transactions,' says that in several of his cases in which active disease was going on in one or both lungs, no rise of temperature took place. And he gives details or an instance in which, although

five observations were made every day for a week, the thermometer was never found above 99°.

In the most acute cases of all, which in Germany have the name of "phthisis florida," the pyrexia may be continuous throughout the twenty-four hours; the temperature may reach 104°, and may never fall below 102°, unless profuse sweating should occur, when it may be lowered for the time to about 100°. It is rather a remarkable circumstance that, even when there is such high fever, delirium and other cerebral symptoms are often altogether absent, but they may occur, and sometimes the patient passes into a "typhoid" condition, with stupor, sordes on the lips, and a dry brown tongue. Another point which may be noted is that phthisical patients often retain a much better appetite than would be present in most other diseases attended with a like degree of pyrexia. Nor is there generally much complaint of thirst. Scarcely less acute is the course of other cases in which the daily range of the thermometer is very wide, the maximum perhaps reaching 103° or 104°, while the minimum may be 98·4° or even lower still. Rühle says that the occurrence of a subnormal temperature, alternating with a high temperature at different periods of the day, is more unfavourable than when the fall is nearly to the normal point. Sometimes the patient experiences a slight rigor, or a sensation of chilliness, and then passes through hot and sweating stages, very like those of a paroxysm of ague, for which disease phthisis has actually been mistaken by a careless observer. In other less severe cases the range of the temperature is comparatively slight; the thermometer may indicate 100° or 101° towards evening, but during the rest of the day it is perhaps scarcely, if at all, above the normal point. In the same patient there may be all possible variations in the thermometric readings. Even when pyrexia is generally present, it sometimes happens that none can be detected during intervals of days or weeks. Rühle says that it has not yet been made out whether the sweating of phthisical patients is invariably preceded by a rise of temperature. This comes on especially during sleep, and some individuals cannot doze for half an hour during the day without their clothes becoming soaking wet. In all probability a paroxysm of cough, of which the patient himself may be unconscious, is often the starting-point of such outbreaks of perspiration. Of the cause of the differences in degree of pyrexia in different cases of phthisis no satisfactory account can as yet be given. Dr Wilson Fox ('Med.-Chir. Transactions,' vol. lvi) thinks that it is generally largely proportioned to the extent of the intercurrent "inflammation." But he seems to admit that there are many exceptions, and to be not unwilling to accept a conclusion which Lebert drew from a very elaborate series of investigations to the effect that the temperature-course is more influenced by individual idiosyncrasy than by anything else. This, of course, is no explanation at all.

Pulse.—The heart's action is nearly always accelerated in phthisis, and its rate is almost as valuable an indication of the activity of the disease as the temperature itself. Like the pyrexia, it is highest in the evening. It is apt to be much affected by slight exertion, and even by a change of position from sitting to standing. It is generally soft and feeble in quality. Sometimes its rapidity is out of all proportion to the degree of fever; in all probability this depends upon the patient's being anæmic.

Vomiting is sometimes a conspicuous and early symptom of phthisis, and instances are known of medical men being thus led into the grave error of supposing that the patient's complaints were all due to disorder of the

stomach. The suggestion was many years ago made by Mr Hilton that vomiting in phthisis is due to interference with the trunk of the pneumogastric nerve by tuberculous bronchial glands. At a later period of the disease, cough often leads to ejection of the contents of the stomach.

Diarrhœa is another symptom which often attracts attention in phthisis. It is generally due to the presence of tubercular ulcers of the small intestine, or rather, perhaps, to a catarrhal state of the mucous membrane in general accompanying such ulcers. Sometimes diarrhœa from this cause persists for many weeks before any physical signs of pulmonary disease can be detected. In advanced cases another cause of diarrhœa is the development of a lardaceous change in the mucous membrane.

A minor point, on which French writers have insisted, is the presence of a pink line on the gums close to the teeth. Whether it is seen more often in persons who are consumptive than in others is doubtful. Nor can much value be assigned to the existence of patches of *tinea versicolor* on the chest and elsewhere, although some writers still mention this as suspicious.

Aspect.—A phthisical patient often betrays the nature of his disease to the experienced physician at the first glance. Apart from the question (to be considered presently) of there being a special configuration indicative of a phthisical diathesis, a bright eye, and a flushed cheek, associated with a wasted frame and lanky hair, at once suggest consumption.

It is a curious peculiarity of consumptive patients that they generally remain all along hopeful as to the result of their illness. As Rühle says, they order new shirts within the last few weeks of their lives.

(2) Of the symptoms which point directly to the lungs, *cough* is naturally the first to be mentioned. Indeed, it is often the earliest indication that the patient is otherwise than well. At first it may be very slight, hardly more than a clearing of the throat; or it may occur only in the early morning, or when the patient happens to exert himself in the course of the day. It sometimes disappears for a time, to return later on. But ultimately it becomes more and more frequent, until it may cause great distress. It is when cough has been the first symptom noticed that the disease, as is so often the case, is said to have arisen out of a "neglected cold."

There may be no *expectoration*, or a greater or less quantity may be ejected of a frothy fluid, either watery or slightly viscid in character. Or the sputa may consist of a glairy greyish material, in which the microscope shows large round granular cells, being fatty epithelium derived from the pulmonary alveoli. Streaks and spots of blood are in most cases present from time to time. As the local process advances, the expectoration becomes muco-purulent; and it may ultimately be almost pure pus, or pus so intimately mixed with blood that it has a uniform brick-dust red colour. This, when abundant, may accumulate in the spit-jar as an almost homogeneous mass. But in other cases the expectoration consists of pellets that remain distinct from one another, even after they have settled upon the interior of the vessel. It is then usual to speak of "nummular sputa," from their resemblance in size and shape to coins. If received into water they are seen to have a loose flocculent surface, as if they were portions of wool, or as if they had been "nibbled at," to copy an expression employed by German writers. They generally contain no air, and therefore fall rapidly to the bottom, unless they are held up by stringy mucus.

Their characters seem to show that they have been formed in a space of some size, not in a narrow tube through which air was constantly passing backwards and forwards. Accordingly, the opinion, which is generally held, that nummular sputa are distinctive of phthisis, is not without reason. But it must be remembered that the necessary conditions for their production are afforded by dilated bronchial tubes, as well as by pulmonary vomicæ. This is probably the explanation of a case mentioned by Sir Thomas Watson, in which he wrongly diagnosed phthisis when chronic bronchitis was the patient's disease.

It has long been known that the sputum of phthisical patients often contains *fragments of pulmonary tissue*, the nature of which can be identified microscopically, for the shape of the alveoli is still plainly visible. Dr Fenwick, in the 'Med.-Chir. Trans.' for 1866, showed that their detection is much facilitated by boiling the sputum with an equal part of a solution of pure caustic soda (gr. xv to ℥j). This dissolves the mucus in three or four minutes. The resulting liquid is then poured into a conical glass which is filled up with pure water; and the deposit which forms is carefully examined in a very shallow cell. Dr Fenwick in one case found 800 fragments in the expectoration of twelve hours. He did not discover them in any case which was at so early a stage that there were no physical signs, but he often succeeded when the signs were such as might have led to the opinion that ulceration or softening of the lung had not begun. The method is also of great value in cases in which phthisis supervenes upon chronic bronchitis and emphysema, when the physical signs are apt to be ambiguous.

At an advanced stage, when cavities were obviously present, Dr Fenwick never failed to find *elastic fibres* in the expectoration, even though the disease appeared to be quiescent, both from the general improvement in the patient's condition, and from his coughing only in the morning and spitting up only a little semitransparent mucus.

More recently, the detection of the *bacillus* of tubercle in the sputum has become an important means of diagnosing phthisis. According to a paper by Dr Heneage Gibbes in the 'Lancet' for 1882, the best method of preparation is the following, which is a modification of those of Ehrlich and Weigert. A thin layer of the sputum is spread out upon a cover-glass, and is allowed to dry. The glass is then passed through the flame of a small Bunsen burner, and afterwards cooled. It is next placed face downwards in a watch-glass upon two or three drops of a freshly filtered solution of magenta crystals (two grm.) and pure anilin (three grm.) in equal parts of alcohol (sp. gr. 830) and distilled water (twenty cc. of each). After fifteen to twenty minutes the cover is removed, and washed with a dilute solution of nitric acid (one part to two of water) until all colour has gone. Then it is washed with distilled water, when a faint colour reappears. Next it is placed face downwards upon a few drops of a saturated solution of chrysoidin, until it has taken a brown colour. After being removed, it is allowed to become perfectly dry in the air, and it is then mounted.*

Dyspnœa is a much less marked symptom than might perhaps have been expected. The gradual onset of the disease and the development of anæmia keeping pace with the destruction of pulmonary tissue, probably accounts for the fact that a patient, even with advanced phthisis, is often able to

* See Dr Gibbes's paper in the 'Lancet' of August 5th, 1883; and Dr Klein's account of Koch's original method, with those of Ehrlich and Weigert, in his 'Micro-

breathe quietly, and to carry on conversation with comfort, so long as he is sitting still. As Sir Thomas Watson observes, nothing is more common than for persons who fear, but will not believe, that they are consumptive, to fetch a deep breath, and bid us remark how thoroughly they can distend their lungs. But any effort or exertion is almost always attended with obvious hurry of breathing in patients who have passed beyond the earliest stage of the disease. And towards the last, orthopnœa is sometimes present in the most extreme degree, so that the patient gasps for breath, while his face and hands are livid and bathed in sweat.

It is no doubt as a consequence of obstruction to the pulmonary circulation that persons affected with chronic phthisis so often have clubbed finger-ends with incurved nails (*ungues adunci*). This affects the toes also, and is the same condition which is seen in chronic bronchitis and in heart disease.

Pain is not commonly distressing or troublesome in cases of phthisis. There may be pain in the shoulder, or beneath the collar-bone, or lower down. But in many cases even this seems to be muscular rather than deeply seated. The pleurisy which invariably fixes the lung to the surrounding structures as the disease advances must be supposed to be painless, for otherwise pain would scarcely ever be absent. But pleurisy lower down, where there is more movement of the parietal upon the pulmonary layer, is not uncommonly attended with sharp and piercing pain.

Hæmoptysis.—This important symptom may be said to be present whenever mucous or purulent sputa contain streaks of blood, or are uniformly discoloured by admixture with it. But in practice it is necessary to distinguish from such conditions the expectoration of blood in a pure state, or frothy with air. In a very considerable proportion of cases this occurrence is the first thing which suggests that there is anything wrong with a patient's lung, or indeed that he is otherwise than perfectly well. He perhaps feels a little tickling in the throat and finds that his mouth contains a fluid which has a salt taste. He looks at his handkerchief and is horrified to see that it is stained with blood. He may either bring up a large quantity at once, or he may remain free from further hæmorrhage for some hours.

From the days of Hippocrates it has been thought that the hæmoptysis is in such cases the cause of the consumption which ultimately develops itself; and two centuries ago Dr Richard Morton included a *phthisis ab hæmoptoë* among his species of that disease.* Recently the same doctrine has been revived by Niemeyer. But only the most overwhelming evidence should lead to the acceptance of the opinion that the extravasation of blood into a lung is ever the starting-point of disease spreading through its substance and

organisms and Disease,' 3rd ed. p. 163: also a full account by Dr Cruikshank in his 'Bacteriology' (1886) pp. 162—167. The advantage of the methods now in use is that by double staining the tubercle bacilli stand out distinct in colour from the rest of the sputum on the slide. Like all aniline dyes, the colour is apt to fade, but if the slides are thoroughly washed and treated with nitric acid this may be overcome. Specimens still show perfectly well which have been made three or four years ago.

* "Hoc tamen perpetuo fere observare licet: quoties scilicet hæmoptoë præcedit, phthisin pulmonarem subsequi solere."—'Phthisiologia,' lib. iii, cap. v (1789). He gives three illustrative cases.

Herodotus narrates the following case, the earliest on record, of phthisis ex hæmoptoë. One of the generals of cavalry in the great host with which Xerxes invaded Europe B.C. 480, was Pharnuches, but he never crossed the Hellespont. For as the army was defiling out of Sardis a dog chanced to run under his horse's feet, and the horse being frightened, reared and threw Pharnuches. After his fall he brought up blood, and the sickness ended in consumption (πεσὼν δὲ αἷμα ἤμεε καὶ ἐς φθίσιν περιῆλθε ἡ νοῦσος), lib. vii, cap. 88.

destroying it. Under various other conditions—as, for example, after injuries to the chest, and in chronic heart disease—we have frequent opportunities of observing the effects of pulmonary hæmorrhage and hæmoptysis ; and few pathologists will assert that they have ever seen it give rise to phthisis. Nor do *post-mortem* appearances lead to the belief that blood extravasated into the air-passages is capable of being inhaled into the pulmonary tissue, so as to form solid nodules, as was maintained by Dr Reginald Thompson, in the 'Med.-Chir. Transactions' for 1878. Cases are often met with in which inhalation of blood into the lung has obviously taken place ; and what is observed is mottling of the cut surface of the organ with red or purple spots, impalpable, devoid of induration, and offering not the slightest resistance to the finger when passed over them. The formation of infarctus or nodules of pulmonary apoplexy is quite a different matter, and occurs only when the pulmonary circulation is in an abnormal condition. Hence it seems most probable that the cheesy congested bodies to which Dr Thompson refers are really relics, not of hæmorrhages, but (at least in most cases) of tubercular lesions. According to Dr Thompson, however, they are most often found in three situations,—in the upper lobe, in the axillary region, and towards the base, but not posteriorly,—which, he says, are notably those where inspiration produces the greatest expansion of the lungs.

A point mentioned by Niemeyer, and to which a certain importance has since been attached by some of those who have discussed this question, is that in one case, four weeks after an attack of hæmoptysis, he found a bronchial tube filled with adherent softening clot, giving it exactly the appearance of a vein obliterated by thrombus. A similar case has since been recorded by Dr Weber in vol. ii of the Clinical Society's 'Transactions.' It is to be noted, however, that in each instance the tube so affected was situated in the lower lobe of the lung. Now, as Traube remarked, such an appearance is so exceptional that very little significance can be attached to it. As a rule, unless a patient has actually been suffocated by hæmoptysis, one does not find any clots in the bronchial tubes after death. Sometimes, a clot of considerable size, with branches that had evidently extended into a number of the bronchi, is expectorated a few days after an attack of pulmonary hæmorrhage. In the museum of Guy's Hospital there is such a specimen.

The other evidence brought forward by Niemeyer in support of the existence of a *phthisis ab hæmoptoë* was mainly clinical. It consisted partly in the fact that hæmoptosis in patients who subsequently die of consumption often takes place at a time when no signs of mischief in the lungs can be detected on the most careful examination ; partly in the fact that the hæmorrhage is frequently followed by fever, acceleration of the pulse, and signs of inflammation of the pulmonary tissue and of the pleura.

The first point is surely worth nothing. We shall presently see that auscultation and percussion frequently fail to reveal lesions which are really present in the lungs, if they happen to be situated deeply or to be scattered widely apart from one another. Take, for example, the case of a patient who is attacked with hæmoptysis, but who recovers from it completely without the subsequent development of any disease, so that the origin of the hæmorrhage remains a mystery. The author recently had under his care an old lady, about seventy years old, who on two successive occasions brought up several ounces of blood, but who got quite well afterwards and is now living, and who has at no time had any signs of mischief

in the lung. The probability is that she really had, and still has, a small cavity or other relic of former phthisis and that this was the seat of the hæmorrhage Dr Weber has remarked that some of the patients who appear to get a *phthisis ab hæmoptoë* have had a tendency to epistaxis, and suggests that there is no reason why blood should not come from the mucous membrane of the bronchi in such persons as well as from that of the nose. But this appears unlikely. It often happens that hæmoptysis is directly traceable to some violent effort or strain, such as rowing, running a race, or lifting a heavy cask. But of course that fact is quite compatible with the existence of disease in the lung at the time.

Niemeyer's other point was that hæmoptysis is often followed within two or three days by an increase in the temperature of the body and in the frequency of the pulse, and by signs of inflammation of the lung and pleura. Traube remarks, in reference to this, that none of the cases cited by Niemeyer show the absence of pyrexia at the time when the hæmorrhage occurred. But a chart given by Bäumler in vol. ii of the Clinical Society's 'Transactions' does show a rapid rise of temperature from the second morning after the commencement of the bleeding until the sixth day, when it reached 103·8°, and then a gradual fall until the eleventh day, when it became normal. And it must be in the experience of every clinical physician that such a febrile attack of variable duration is of frequent occurrence after an attack of hæmoptysis, and that before it subsides one can often make out distinct signs of consolidation of one apex, which were absent when it began. Still Niemeyer's interpretation of these facts is not the most probable. It seems far more likely that the hæmoptysis is itself a direct effect of the development of tubercles in the pulmonary tissue. Hæmorrhage is no uncommon symptom of miliary tuberculosis of the lung, and may be immediately fatal at a time when there is neither ulceration nor obvious consolidation of the lung-substance, and when the only lesions found after death are recent miliary tubercles which had apparently produced no other symptoms whatever. There is no doubt a difficulty in saying how the bleeding is brought about, but it seems very likely that the growth of tubercles in the walls of the alveoli may be attended with an invasion and softening of the coats of many of their capillaries, while at the same time the blood-pressure in them is augmented in consequence of compression of other capillaries. Rindfleisch, in 'Ziemssen's Handbuch,' gives a microscopical drawing, showing the coats of a minute artery actually perforated by a tubercular cell-growth.

But in other cases of phthisis, hæmoptysis is due to a very different cause, namely, to the rupture of the wall of a branch of pulmonary artery crossing the side of a vomica or enclosed in a trabecula. Rasmussen, of Copenhagen, first made known the fact that in many instances of this kind the hæmorrhage is preceded by an aneurysmal bulging of the coats of the vessel. A translation of his paper appeared in the 'Edinburgh Medical Journal' for November and December, 1868, and for August and September, 1869. Since that time the occurrence of such aneurysms in vomicæ has been noticed by many observers. In the 'Pathological Transactions' for 1871 Dr Douglas Powell tabulated a number of cases that had been inspected by him. We have had a specimen in a child under three years of age. This instance is in itself sufficient to show that the formation of the aneurysm is not the result of atheroma, like that of an ordinary aortic or popliteal aneurysm. Rasmussen was inclined to attribute it to the un-

supported state of the walls of the vessel when one side of it is exposed in a vomica. But Dr Powell points out that the coats are much swollen, semigelatinous, and glistening; and it therefore seems clear that their yielding to form a pouch depends on a previous inflammatory change, more or less like that which causes aneurysm in an artery occluded by an embolus. This, indeed, was Rokitansky's account of the lesions which precede large bleedings in phthisis, in opposition to Laennec's theory of diapedesis and Andral's of bronchial oozing. The size of an aneurysm in a vomica is commonly from that of a pea to that of a nut; but Dr Powell speaks of one which was as large as a Maltese orange. The vomica in which it is found is usually an old one, with fibrous walls. The point of rupture is usually a little hole or fissure just large enough to admit a probe. Hæmorrhage may have recurred on several different occasions, at intervals of days or weeks, before the fatal issue. Indeed, death is not by any means always the direct result of an attack of bleeding, and the patient may sink exhausted after having ceased to spit any blood for several days. But in other cases he may die almost instantaneously, with a rush of blood from the mouth and nose. Or he may even be choked by the blood before any of it appears externally, so that the occurrence of hæmorrhage is not suspected until an autopsy is made. On the whole, it is remarkable how rarely even profuse hæmoptysis is the immediate cause of death in phthisis.

Instances are not uncommon in which, instead of having formed an aneurysm, the branch of pulmonary artery from which fatal hæmorrhage had occurred is found to be simply perforated by a process of ulceration. At Guy's Hospital the one condition has been as frequent as the other. Lastly, in some cases, even of advanced phthisis, in which the lungs contain many vomicæ, it is not possible, after the most careful search, to discover what has been the source of the hæmoptysis. No part of either lung may seem to be more deeply stained with blood than all the rest, even though death may have occurred almost immediately.

A point of some importance in regard to cases of ruptured aneurysm, or laceration, of a branch of the pulmonary artery is that the blood which is expectorated by the patient is usually of a bright red colour. For some writers have insisted that when the source of the hæmorrhage is doubtful, such an appearance must prove it to have been derived either from a bronchial artery or from a pulmonary vein. But it is extremely rare for blood from the lungs to be dark coloured. One such case is related by Niemeyer, in his 'Clinical Lectures.' The patient had brought up enough blood to fill three basins within a few minutes; it was found to have a thin frothy layer on the surface, but below this it was coagulated into a dark, almost black, cake. Anyone not acquainted with the facts might, says Niemeyer, have supposed that it came from a profuse venæsection. In all probability the bright red, arterial appearance, which is usually seen, depends on the blood having become aerated either after its expectoration or while it is in the bronchial tubes, where it certainly often is freely exposed to the air, as is shown by the frothy state in which it reaches the mouth. At any rate it is clear that in no case of hæmoptysis can the fact of the blood being bright red be taken as proving that it came from one kind of vessel in the lung rather than another.

A further point of great interest, on account of its bearing on the question of a *phthisis ab hæmoptoë* is that in none of Rasmussen's cases of

hæmorrhage from aneurysms of the pulmonary artery was any recent pneumonia found at the autopsy, even when the patient had lived for some weeks. If it should be found that no pyrexia develops itself when, in a patient previously free from fever, hæmoptysis results from ruptured aneurysm, or from laceration of a branch of pulmonary artery, it would give the *coup de grâce* to Niemeyer's view.

Hæmoptysis, like so many other hæmorrhages, has been supposed by some observers to be frequently *vicarious* of the catamenial function. Sir Thomas Watson, for example, says that this is not at all uncommon, and that it is not usually attended with any peril to life. He cites a case which was observed by Pinel at the Salpêtrière, that home of all that is marvellous in disease, in which a woman was said to have menstruated through her lungs from the age of sixteen to that of fifty-eight, often to the extent of two quarts of blood during a period of two days, while she nevertheless remained plump and healthy. A very different view of this question is taken by Rühle, who will only admit that in patients who already have lung disease suppression of the catamenia (or of a hæmorrhoidal flux) may be followed by vicarious hæmoptysis. He speaks of having seen cases in which this recurred at intervals of from four to six weeks, until a few leeches were applied to the anus with a corresponding regularity. Vicarious hæmoptysis, if it occurs at all, is so rare that we should look with great suspicion on a supposed case of this condition.

In almost all cases of hæmoptysis, if the bleeding should cease, there is for some little time afterwards a continuance of expectoration of a deeply blood-stained material—clotted blood, or mucus intimately mixed with blood. This is gradually found to alter in appearance, becoming reddish-brown, or brownish-black in colour. Such a change in it should be carefully noted, because it shows that the hæmorrhage is in reality no longer going on, and perhaps this may affect the treatment. But of course there is still reason to fear that fresh oozing may at any time occur.

Concretions.—When the tubercular process in a part of the lung has become quiescent, and calcification of some of the cheesy material has occurred, it not uncommonly happens that the patient ultimately spits up the concretions which are thus formed, and which may be of all sizes up to that of a pea. Sometimes their detachment from the tissues in which they had been embedded is attended with a little hæmorrhage, and Rühle seems to think that there must necessarily be at the time some fresh softening, so that a further advance of the disease may be anticipated. Indeed, that the expectoration of pulmonary concretions is unfavourable was long ago stated by Morgagni. But this occurrence in many cases has not been followed by any serious consequences. In one case it took place at a considerable interval of time after the subsidence of all active symptoms; and the patient in question is living at the present time. It must be remembered, too, that exactly similar concretions may come from the substance of a mediastinal gland, having reached the trachea, or one of the bronchi by ulceration. A case in point occurred at Guy's Hospital in 1874; the man, who had been spitting up pieces of calcareous matter every two or three weeks, was admitted into the hospital and died there; and at the autopsy it was found that round the affected gland there was an abscess which had opened into the œsophagus as well as into the right bronchus.

Physical signs.—The examination of the chest in a case of phthisis

reveals slow and progressive consolidation, followed by excavation of the affected parts of the lungs.

(1) *Initial stage.*—At the commencement of the disease the signs may be very slight and doubtful, and repeated examinations at intervals of some days, or even two or three weeks, may be required, before one ventures to express a positive opinion as to whether mischief is developing or not.

Among the earliest changes to be detected is diminished mobility of the upper part of the chest on one side. Standing behind the patient, with one hand placed lightly below each of his clavicles, the physician can feel that the expansion of the two sides is not equal; one lags slightly behind the other, or one stops in its movements while the other still continues to rise.

On percussing with great care, and comparing closely corresponding regions of the chest, he may make out that there is more or less decided deficiency of resonance, amounting perhaps to actual dulness, either in front or behind. A good plan is gently to flick the two clavicles in turn with the finger; the resulting "osteal" sound may then be mixed with unequal degrees of pulmonary resonance on the two sides. It is important to examine the spaces above the clavicles as well as those below them; and by employing different amounts of force in succession one may sometimes find that a particular kind of stroke elicits an impairment of resonance better than others. The suprascapular regions must also be carefully percussed; a firm blow is required to bring out differences of sound there.

On auscultation it may be found that the vesicular murmur is not alike on the two sides. If over one apex it is permanently deficient or even absent there can be no doubt that that is the lung which is affected, but it must not be forgotten that a temporary disappearance of breath-sound may be due merely to plugging of a bronchial tube with mucus. In other cases the presence of phthisis causes the vesicular murmur to be louder or harsher than natural. It is then difficult to determine by auscultation alone which of the two lungs is most likely to be the seat of disease, for an abnormally loud vesicular murmur, instead of indicating mischief when it is heard, may be compensatory of mischief on the opposite side. Various modifications in the character of the vesicular murmur may also be present in early phthisis. It may be interrupted or divided into two or three distinct parts corresponding with irregularities in the play of the chest walls. These interruptions may be so frequent that the inspiratory murmur has been compared with the sound produced by a revolving cogged wheel (*respiration saccadée*). This must not be taken as necessarily showing that disease is present, for Dr Walshe has "observed it at one or both apices, when free from consolidation of any kind." The case to which he alludes was that of a female, and in all probability the cogged-wheel rhythm was due to the action upon the healthy lung of an irritable heart. For the separate sounds which make up cogged-wheel breathing are synchronous with as many cardiac pulsations, as pointed out in the 'Revue mensuelle,' 1877. It seems likely that when a portion of the lung is partially solidified by tubercles the shock given to it by the beating of the heart, whether directly or through the blood-vessels, may produce a greater effect than normally on the air-cells which still receive air.

One of the signs of tubercular disease of the anterior edge of the lung is an increased loudness of the cardiac sounds in the corresponding subclavian region. In some cases, too, there is heard over the pulmonary artery a systolic murmur, which is supposed to be due to compression of that vessel by

the lung. A somewhat later sign of phthisis is the presence of non-consonating moist sounds at the affected apex. In some cases they are audible only just after the patient has coughed, so that one must never conclude that they are absent until one has listened over the apices while making him cough. If limited to the upper lobe moist sounds are of special diagnostic significance, since a simple catarrh is probably never thus localised.

(2) *Consolidation.*—No doubt a considerable amount of consolidation may take place in the apex of a lung without any physical signs being audible beyond those which are mentioned in the last paragraph, but as the process of solidification goes on it almost always happens before long that bronchial breathing is discoverable. At the same time dulness on percussion becomes more marked than before, and the voice is transmitted by the stethoscope with increased loudness, constituting bronchophony. These signs possess an importance which cannot be exaggerated; but at the same time it is essential always to bear in mind what has been stated at pp. 84 and 89, about the normal presence of bronchial breathing and of bronchophony in certain regions of the chest, especially in some people. And it should be added that throughout the right suprascapular, supraclavicular, and subclavicular regions the voice may in health be heard more loudly than in the corresponding left regions, though the difference is too slight to justify our speaking of the sound on the right side as bronchophonic. Moist sounds may or may not accompany the bronchial breathing of phthisical consolidation. When they are present they generally have a markedly consonating character. A very common combination is for the inspiration to be attended with râles, so that no bronchial breathing is noticeable, whereas immediately afterwards a blowing expiratory sound is heard but no râles.

From a very early period of the disease the regions above and below the clavicle on the affected side are commonly found to be flattened or slightly hollowed. Rühle lays stress on the fact that even when no dulness on percussion can be detected in the supraclavicular space one can often make out that resonance reaches upwards for only an inch or an inch and a quarter above the clavicle instead of an inch and a half or two inches.

(3) *Excavation.*—The quality of the bronchial breathing in a case of phthisis may be modified to tubular, without there being any further change in the affected part of the lung beyond consolidation; and, conversely, excavation may take place to a considerable extent, without the physical signs indicating it by any modification in their quality. But such cases are altogether exceptional. The formation of vomicæ is an occurrence so nearly universal that one is scarcely ever wrong in diagnosing the presence of a vomica where we hear well-marked "hollow" sounds.

It is not infrequent for a phthisical cavity to become so large that the recognition of its physical signs is the key to the diagnosis of the case. The corresponding part of the chest may in some rare cases fill out, and even bulge slightly, when the previous consolidation had given rise to a capacious hollow, as was once observed by Dr Walshe. One might perhaps have anticipated that under such circumstances the percussion-sound should become resonant again or even tympanitic. It has, however, long been known that this is not so; the thick adherent pleura and the condensed lung tissue round the wall of a vomica serve effectually to check the vibrations of the thoracic parietes, so that a toneless noise always forms a large part of

the sound which is elicited on percussion. But mixed with this are tones of varying quality, due to the vibration of the air within the vomica itself, and thus the sound as a whole may present all those modifications enumerated at p. 80, from osteal to tympanitic.

A frequent peculiarity of the percussion-sound over a large vomica is, that it resembles the noise produced by striking coins together, or by striking over one's knees the hands loosely clasped. Laennec called such a percussion-sound the *bruit de pot fêlé*, or in English "cracked-jar sound." For its production in a perfect form the walls of the cavity itself and the thoracic parietes must be elastic and yielding, the percussion-stroke must be heavy and forcible, and the cavity must communicate freely with the bronchial tubes, and these again with the external air through an opened mouth. The reason is that the *bruit de pot fêlé* depends upon the expulsion of air from the cavity, just as in striking the hands over the knee one drives air out through a chink between them. The most marked example of this kind of percussion-sound was a patient who had, outside the thorax, beneath the pectoral muscles, an abscess-cavity which contained air, and which communicated with the pleural space (itself filled with air) by a narrow hole through the intercostal muscles. But there are in fact many other diseased states of the respiratory organs in which this curious percussion-sound may occur. Thus, according to Dr Gee, it is sometimes obtained over the upper part of the front of the chest in cases of pleuritic effusion, sometimes over islets of unsolidified lung embedded in tissue hepatised in acute pneumonia, sometimes in cases of malignant tumour. But in cases of phthisis there is no likelihood of falling into error by taking it as significant of the presence of a cavity.

Precisely similar in its mode of origin to the *bruit de pot fêlé* is a phenomenon which sometimes attracts the notice of the patient himself as well as of other persons, namely, the transmission of the heart-sounds outwards so that they can be heard, like the ticking of a watch, at a distance of several feet from him.* This singular phenomenon would doubtless be much less rare than it is were it not that a cavity of sufficient size to have a quantity of air driven out of it by each pulsation of the heart very seldom exists, except in the upper lobe.

On auscultation over a large vomica one may obtain any modification of bronchial breathing up to the amphoric. Another modification, which appears to be heard only when a cavity has been formed, has recently received from Seitz the name of *metamorphosing murmur*.† It is probably not very different from what Laennec long ago described somewhat vaguely as the *souffle voilé*. It is said to be characterised at the commencement of inspiration by an unusually harsh sound, which lasts only during one third of the inspiratory period, and gives place during the remaining two thirds to bronchial breathing accompanied by a metallic echo, or to ordinary râles.

All kinds of metallic phenomena may present themselves in a very large vomica, exactly as when there is pneumothorax. The moist sounds are often very "large," so as to claim the designation of gurgling. Vocal resonance often amounts to pectoriloquy. On the other hand, Dr Walshe

* Many years ago my father showed me a case of this kind, which had come under his observation. The sounds were sometimes audible across a good-sized room, but I found that when the patient, a young woman, was made to close her mouth, I could instantly stop them by pressing together her nostrils. Just such a case was brought under the notice of the Clinical Society in 1880, by Dr Frederick Taylor.—C. H. F.

† See von Niemeyer's essay on 'Phthisis' (New Syd. Soc. transl.), p. 54, note.

insists on the fact that over a large cavity, at least at its upper part, there *may be dead silence*, both respiratory and vocal.

One very rare effect of excavation of the lung is the production of subcutaneous emphysema. A case in point occurred at Guy's Hospital in 1882. The patient had been slowly sinking for weeks, and shortly before his death there was a slight crackling below the clavicle and at the root of the neck. Fräntzel, in 'Ziemssen's Handbuch,' alludes to similar instances. As pneumothorax is not present, it must be assumed that ulceration extends through both layers of the pleura, the space between having been previously closed by adhesions.

Involution.—The physical signs of quiescent or retrogressive phthisis vary widely in different cases. Shrinking of the upper part of the chest may go on until the clavicle is seen to be obviously at a lower level than on the healthy side. Dr Walshe also says that the corresponding suprascapular region may be distinctly more hollow than its fellow. Unless excavation of the lung has gone on to a great extent the percussion-sound is usually very dull; indeed, Rühle remarks that extreme dulness in phthisis is usually a sign that the case is likely to run a favourable course.

The heart becomes uncovered by retraction of the lung, especially if the left is the one affected. Its impulse may be seen and felt over a much more extensive area than is naturally the case even as high as the third or the second intercostal space. The stomach also may be drawn upwards to the level of the sixth or the fifth rib. On the other hand, if the right lung is diseased, the heart's apex may be displaced to the right side of the sternum; and the liver may be dragged up as high as the fourth rib.

The opposite lung.—In all cases of phthisis it is, of course, very important that, while one is watching the changes that take place in the region first affected, one should also be on the look-out for signs of extension to other parts of the same lung as well as to the opposite lung. The frequency of excavation in the apex of the lower lobe makes it advisable to listen carefully below the spine of the scapula, or rather on that level, but with the shoulder-blade when it has been drawn outwards by the patient crossing his arms. In advanced cases, the question to which one should mainly direct one's attention is very often not what parts of the lungs are diseased, but what parts remain capable of carrying on the function of respiration. And it is surprising to how small an area, at the extreme base of one lung, one may find the presence of a vesicular murmur restricted. At this point, however, it is exceedingly harsh and loud, affording in fact a most typical example of "compensatory" or "puerile" breathing. On the other hand, one must not over-estimate the significance of crepitations and râles, when heard over the whole of the back of a lung, as proving, even if they are consonating in character, that the corresponding lung substance contains more than scattered or clustered tubercles. In cases of acute and general miliary tuberculosis it has appeared during life that large tracts of the pulmonary tissue were breaking up, and yet it appeared after death that the pulmonary tissue between the tubercles was still crepitant.

But in the majority of cases of phthisis, the discrepancy between physical signs and *post-mortem* appearances is in the opposite direction. Clinically disease is perhaps discovered in the upper lobe of one lung; the autopsy shows that nearly the whole of that lung is affected, and also the upper lobe of the other lung. This is only in part to be explained by the extension of the mischief in the interval that may have elapsed. Apart from

any such hypothesis we must frankly recognise the fact that the presence of well-marked disease in one apex adds greatly to the difficulty of detecting early mischief in the other apex. The reason obviously is that one has lost the standard of comparison on which one is accustomed to rely.

Diagnosis.—The recognition of phthisis, which must be based upon symptoms as well as signs, is often very simple and easy. But there are cases in which there is the greatest difficulty in arriving at a right conclusion, and in which, indeed, the only safe course is to reserve one's opinion, at any rate for a time. The diagnosis between phthisis and other pulmonary affections has been already referred to in previous chapters. In practice the doubtful cases are generally rather those in which physical signs are either wanting, or at least slight and obscure, so that one hesitates as to whether the disease is in the thorax, or whether there is not rather some deeply-seated new growth, or some lesion of the internal lymph-glands, or of the thoracic duct, or of the great abdominal nerve-centres, by which the patient is wasted and worn down. Very often, however, although the nature of the affection cannot be determined, it is clear that he is stricken by fatal disease of some kind. Under such circumstances inability to give an exact diagnosis is not practically of great importance.

But it is quite otherwise when, as is sometimes the case, the prognosis depends absolutely upon the opinion one may form. The doubt generally then is whether the patient, if a man, may not be merely suffering from the syphilitic cachexia, or be the victim of hypochondriasis and of aggravated dyspepsia; if a woman, whether she is not hysterical. In all cases of this kind the thermometer is of the greatest value. One hysterical affection, which has often been mistaken for phthisis—the "anorexia nervosa" of Sir William Gull—will be fully described elsewhere. But there are other cases in which the suspicion of pulmonary disease is based mainly upon the fact that the girl, as is said, "spits blood." A glance at the sputum is sometimes sufficient to remove all uneasiness about this. What is expectorated may be found to be a rather slimy liquid, uniformly tinged of a pink or purple colour, so that it looks exactly like the juice of plums or of some other fruit. It is, in fact, saliva or secretion from one part of the mouth; and the blood comes from the vessels of the mucous membrane. Rühle remarks that this sort of hæmorrhage often occurs in the night, from the patient making sucking movements of the lips and cheeks during sleep. Thus the pillow may show stains of blood, the origin of which seems at first to be inexplicable. Another variety of sanguineous expectoration, which is equally unimportant, is due to the rupture of small vessels at the back of the fauces during violent coughing, or "hawking up" of phlegm. Varicose veins in the pharynx may be the source of hæmorrhage.

Dr Walshe, insisting on the difficulty of diagnosis in early cases of phthisis, advises little weight to be laid on differences in vocal resonance, particularly at the right apex in women, nor harsh or jerking inspiration at the same spot; and urges the propriety of suspending judgment until a second examination of the chest has been made.

Course and events.—Phthisis varies greatly in the rapidity of its progress, but its duration is almost always a matter of several months, and sometimes of many years. Trousseau is no doubt right in saying that the only *phthisie*

galopante is miliary tuberculosis of the lungs. Traube, indeed, related in the 'Berliner klin. Wochenschrift' for 1867 the case of a man, aged twenty-eight, who died, after thirteen days' illness, of "acute tubercular (caseous) pneumonia." The attack began with rigors and fever; a few days later hæmoptysis set in, and became one of the chief symptoms. At the autopsy all parts of the left lung presented patches of lobular hepatisation, the centres of which were caseating, especially in the upper lobe. A similar affection, in an earlier stage, existed also in the right lung. Both apices, moreover, showed traces of old mischief. Eight cases occurred in Guy's Hospital, in each of which there was a definite history that the duration of the patient's illness, from its commencement to its fatal termination, was only from five to twelve weeks. In two instances the attack was attributed definitely to a chill. One man said that he got wet through while working in a potato field, after which he shivered and became hot, and was never well again; the other that on a particular occasion he slept with his window open. In almost every one of these cases vomicæ had formed before death, especially in the upper lobes, in the centres of the cheesy masses, which formed the most conspicuous lesions observed at the autopsy. It must, however, be borne in mind that the distinction from miliary tuberculosis may, sometimes at least, be not very apparent. The dissemination of the tubercular virus by the blood-current may, if the tubercles to which it gives rise in the lungs are not very numerous, have no apparent effect until they in their turn become starting-points of a local infection, when a disease exactly like ordinary phthisis may arise.

The sudden commencement of some of the rapidly fatal cases of phthisis is of great importance in regard to their diagnosis from cases of acute pneumonia of the upper lobe of a lung. The most serious errors of diagnosis have been made between the two diseases; the mode of onset usually affords a means of arriving at a right judgment. But it is evident that this is not always the case; and the only point on which one can fall back seems to be one to which Traube has drawn attention, namely, that in acute phthisis bronchial breathing is not discoverable until much later than in pneumonia of the upper lobe—not until the end of the second week, or even for a longer time still. It is a striking fact that when fibrinous pneumonia occurs in a person who already has phthisis it often seems to run as favourable a course as if it had arisen in one who was healthy. Thus Andral is said by Rühle to have seen a single phthisical patient pass through from twelve to fifteen successive attacks of pneumonia. If one finds very extensive consolidation in a case of phthisis when it first comes under one's observation, one should always think of the possibility that it may, in part at least, be the result of intercurrent pneumonia, and therefore that the prognosis may be far less grave than it otherwise would have been.

But even acute phthisis—"phthisis florida," as German writers term it—may, instead of running on straight to a fatal termination, become arrested, and afterwards run a chronic course. Rühle relates a case in a girl, who seemed to have but a short time to live when she was transferred to his charge from that of his predecessor at Greifswald, Niemeyer. Yet her symptoms subsided, and she was discharged from the hospital with signs of a cavity in the left upper lobe, and did not die until the following year, having in the meantime given birth to a child.

The progress of ordinary chronic cases is, almost without exception,

interrupted by intervals, during which the patient may seem to regain his health. Cough may almost disappear; even the evening temperature becomes normal from day to day, the appetite returns, the face is no longer pale, the weight of the body becomes as great as it used to be. It is of course true that this favourable change commonly takes place under medical advice, and we shall presently see how important it is that the advice should be well carried out. But sometimes it occurs even in those persons who are not able to do so, and who have gone on working in spite of their illness.*

Duration.—Different observers have made widely different estimates as to the duration of phthisis. Sir Thomas Watson cites Dr Gregory, of Edinburgh, as having stated that the "ordinary duration" of the disease was about six months. Laennec, Andral, Bayle, and Louis each put the "mean duration" at about two years. Dr Austin Flint, in America, found that, excluding acute tuberculosis, the average duration was thirty-three months. These estimates apply to the first thirty years of the century. Dr Pollock, analysing 3566 cases observed by him at the Brompton Hospital, found that the "average duration" of these cases while under observation was more than two years and a half, and in the course of that time only 127 ended fatally. What was the real average length of the disease among the whole number of cases he could not tell, but it must clearly have been much larger still. It is, however, very difficult to believe that Dr Pollock's cases fairly represent the ordinary course of the disease. There must have been an undue proportion of exceedingly chronic cases, and cases running a rapid course must in some way have been excluded. Still more extraordinary are the statements made by Dr Theodore Williams, in vol. liv of the 'Med.-Chir. Transactions,' with regard to the duration of life among 1000 cases of phthisis seen by Dr C. J. B. Williams in private practice between 1842 and 1864. Of the patients in question 198 were known to have died; in them the average duration of the disease was nearly seven years and three quarters. In the remaining 802 patients who were alive when last heard of, its average duration had already been more than eight years. Among these cases, however, none were included which had not been at least one year under observation, and this restriction, besides keeping out of the list all rapidly fatal cases, doubtless weeded it of the majority of those who failed to improve for a time under the treatment recommended.

That life is sometimes maintained for a great length of time after phthisis has developed itself has long been well known. Sir Thomas Watson alludes to a patient of Dr Gregory's who was at least seventy-two years old when he died, and who from the age of eighteen had never been free from symptoms, "being often hectic, and frequently spitting blood." In women the average course of phthisis appears to be shorter than in men.

Immediate causes of death.—The fatal termination of phthisis is not seldom

* In January, 1874, a hatter, aged thirty-seven, who said that he had been ailing for six months, came to me with signs of phthisis at both apices. His morning temperature was 101·4°. His father had died of consumption. Notwithstanding my urgent advice to give up work, he did not rest for a single day. The only difference he made was that instead of living away from his workshop in the Borough, so that he was exposed to changes of temperature in going backwards and forwards, he now slept in the same building. For a week or two the physical signs increased, moist sounds becoming audible all over the left lung. But his symptoms quickly improved, and by the end of May he was as stout as ever and said that he felt nearly well. The signs at the apices, however, still remained. In the following year I heard incidentally that he was in good health, with only a little occasional cough. But in 1879, his symptoms returned, and he ultimately died in September, 1881.—C. H. F.

sudden and unexpected. In 1866 a gentleman, aged twenty-six, who had long been ill, went up to London from Brighton one day to transact some business. At the London Bridge Station he was seized with alarming symptoms, and was taken down to Guy's Hospital, where he died within a quarter of an hour from the beginning of the attack. In 1868 a labouring man, aged twenty-three, who had been indisposed for some time, was at his usual work near Guy's Hospital, when about 2 p.m. he began to suffer from dyspnœa; this rapidly got worse, and he was carried to the hospital and died in two hours. In neither case did the autopsy show why death should have occurred at that particular time.

Pneumothorax often brings more or less immediate danger to life. Nevertheless, when the immediate effects are got over, the consequent collapse of the lung appears to be unfavourable to rapid progress of the disease; so that if the other lung is but slightly affected the patient's condition may, at least for a time, improve. Some practitioners, acting on this hint, have even ventured to puncture the pleura and thus produce pneumothorax, with the hope of checking hæmorrhage or other dangerous local complication.

Another accident that may happen to consumptive patients is pulmonary *embolism*, resulting from thrombosis of the femoral veins.

Syncope is sometimes the cause of death; or it may arise from sudden exhaustion of the respiratory centre (p. 9). Indeed, it is not very uncommon for consumptive patients to be found unexpectedly dead in the course of the night.

In other instances phthisis ends fatally by the supervention of tubercular disease elsewhere than in the lungs, by *tubercular meningitis* or peritonitis, or by tubercular disease of the kidney, or by solitary tubercle of the brain or spinal cord.

It is a remarkable fact that *hæmoptysis* is very rarely the immediate cause of death in phthisis. Considering that it is present in more than four fifths of consumptive cases, it is exceptional to find a patient "choked in his own blood," although every practitioner has seen that event.

The main symptom towards the last may be *diarrhœa* resulting from tuberculous ulceration of the intestine, or the dysphagia and the other distressing symptoms produced by a like affection of the larynx.

Some affections which appear to be inflammatory rather than tuberculous are also of sufficient frequency in phthisis to deserve mention. One is suppurative peritonitis from impaction of the cæcal appendix, and another is abscess of the brain.

The coincidence of *fistula in ano* with phthisis is one which requires brief mention. Dr Pollock points out that it occurs far more often in males than in females, and most commonly in persons who are no longer young, the most frequent age for it being from thirty-five to forty-five. The disease of the lungs has very generally advanced to the formation of vomicæ before the fistula appears. Many observers, including Dr Pollock, are of opinion that in such cases no operation should be attempted; for, when it is successful, the phthisis is very apt to assume increased activity two or three months later. But there seems to be no evidence that the cure of a fistula in a person not already consumptive renders him more liable than before to the supervention of pulmonary disease.

Finally, *lardaceous degeneration* plays a very prominent part in bringing to a close many cases of phthisis. If the intestines be involved, an intractable diarrhœa may result, which cannot be distinguished during life from that o

tuberculous ulceration. But it is chiefly by affecting the kidneys that this kind of degeneration acquires its clinical importance. General dropsy sets in and the patient acquires more or less of the appearance usual with those who are affected with Bright's disease. Indeed, tubal nephritis sometimes comes on in phthisis without there being any lardaceous change discoverable in the renal glomeruli or vessels even with the microscope. It is therefore not safe to diagnose a lardaceous affection of the kidneys from the mere fact that the patient has albuminuria. According to observations made by Dr Williams, recorded in a paper read before the Royal Medical and Chirurgical Society in 1882, the occurrence of albuminuria in phthisis has the effect of masking the other symptoms, and especially of making the temperature range lower.

Prognosis.—From what has been stated in the preceding paragraphs it may easily be imagined that to give a correct prognosis in phthisis is no light matter. And in point of fact those physicians who have the largest experience are precisely those who most strictly abstain from attempting to predict the duration of life among their patients.*

Many writers divide the course of phthisis into three stages: the *first* stage they associate with the "formation," deposition, or growth of tubercles, the *second* with their "softening," the *third* with their "elimination" by the process of excavation. But, as already pointed out at p. 135, the moist sounds which are supposed to indicate "softening" are very apt to be fallacious. It is another objection to these so-called stages that at the best they have reference only to the local process in certain parts of the lungs, and not at all to the disease as a whole. For while vomicæ exist in one or both apices, tubercles are commonly being formed lower down. But the strongest objection of all is that to speak of *stages* of phthisis leads almost inevitably to a complete misconception of their significance in prognosis. To every patient, as well as to his friends, it cannot but appear to be a matter of course that in a malady which, like phthisis, is almost inevitably fatal, the third stage must be the worst. And yet it is no paradox to say that the exact contrary would be nearer the truth.

A factor which, more than any other, requires to be taken into account in attempting to determine the probable course of the disease, is its greater or less tendency to advance rapidly in that particular patient. In different cases the differences in this respect are enormous; and it does not appear that any explanation of them can be given, except that, as a rule, the progress is quicker in those who have a strong inherited tendency to consumption than in those who have no such tendency. Now, the formation of a cavity of any size takes a considerable amount of time, especially if its walls are to acquire a smooth lining. Hence, whenever the morbid process spreads with much rapidity through one or both of the lungs, the opportunity for such cavities to develop themselves is wanting. In other words, the fact that a case presents the physical signs of the third stage is proof that its course has been such as generally warrants a comparatively favourable prognosis. And, in reality, patients with large vomicæ often go on year after year with but little change in their condition, and even with enjoyment of life. Dr Walshe speaks of two singers—a distinguished contralto and an excellent soprano—as having within his knowledge continued to perform at the Opera, "while the excavating process advanced in their lungs."

* See on this difficult question the sections on prognosis in the works of Dr Walshe and of Dr Williams, and the monograph of Dr J. E. Pollock.

On the other hand, one is in most cases compelled to speak very guardedly of the probable duration of phthisis, if physical signs indicate that the morbid process is still actively going on in any part of the lungs, whatever may be the stage to which it has reached in the apices. One must not forget, too, that although in the lung first affected its progress may have been slow, it may yet rapidly hurry on to a fatal termination when it passes to the other lung. The degree of severity of the general symptoms is of course very important in regard to prognosis, especially the rate of the pulse and the height of the temperature. But it must be remembered that debility and exhaustion may render the pulse rapid as well as activity of local mischief. And the existence of pyrexia, as has been shown by Dr Theodore Williams, is not incompatible with gain of weight—nor even, we may add, with the subsidence of many of the other symptoms of the disease—provided that the patient eats and digests well.

The majority of cases of acute or "pneumonic" phthisis occur in young subjects, whereas "fibroid" phthisis, which is necessarily a chronic form of the disease, is most frequent in those who are advanced in years. This seems to have led to the idea that the prognosis should be more favourable in proportion as the patient is older. Dr Walshe, however, says that his observations at the Brompton Hospital failed to confirm such an opinion; and Lebert (in vol. xi of the 'Deutsches Archiv') has pointed out that age seems to have little influence on the intensity of the pyrexia, which we have seen to be one of the most important factors in determining the rate of progress of the disease. Indeed, it is obvious that the greater frequency of very acute and of very chronic cases respectively in youth and at an advanced period of life affords no real reason for supposing that cases of what may be called an average degree of severity will run a more rapid course at one age than at another. It can hardly be doubted that in this disease, as in almost every other, the patient's power of resistance and his capacity for repair must alike diminish as he grows older. And as we have seen that the prognosis of phthisis is always to be based mainly on the rate at which it seems to be actually advancing in the particular case under consideration, it seems to follow that the patient's age may be altogether disregarded.

On the whole, good appetite and increasing weight are more important as favourable tokens than absence of well-marked physical signs.

Ætiology.—In discussing the subject of tubercle in general (vol. i, p. 86), we have seen that although the growth and diffusion of a bacillus among the tissues of the body seems to be mainly concerned in determining the spread of tubercular lesions when they have once begun to develop themselves, clinical observation is nevertheless altogether opposed to the idea that *infection from without* is the most essential part of the ætiology of phthisis and of other tubercular diseases. Of the fact that consumption is not ordinarily communicable from one person to another no better illustration could be given than a statement published in 1867 by Mr Vertue Edwards, who had then for seventeen years been resident medical officer at the Brompton Hospital. In that period he remembered personally fifty-nine resident medical assistants, whose duration of office averaged quite six months. Of these he believed all but two to be alive; one had died of aneurysm, one of some cause unknown; three, still living, were said to be consumptive. Very many nurses had been in residence from periods varying from months to eight, twelve, or even twenty-four years. Of the head nurses, who slept each in

a ward of fifty patients, only two were known to have died—one of apoplexy; the other, after an unhappy marriage, of phthisis. No under-nurse, so far as he was aware, had died of phthisis. The matron and her two predecessors, as well as the chaplain and his two predecessors, were all alive. Of the physicians, whether for in-patients or out-patients, all were living except two; one had died of causes unconnected with disease of the lungs, the other from some disease of unknown nature, after twelve years' absence from the hospital. Mr Edwards himself at the end of (now) twenty-five years is still in good health. The circumstance that phthisis does not ordinarily spread from a patient who remains in his home to brothers, or sisters, or other relatives, is the more striking because they must be supposed to have very often inherited a more or less strong predisposition to the disease.

In Dr Weber's cases, recorded in the Clinical Society's 'Transactions' for 1874, the disease seemed to pass immediately from husbands to their wives. The husbands, all of whom were affected before marriage with the pulmonary mischief, were nine in number; but the deaths from phthisis among their wives were as many as eighteen; one lost four wives in succession, one lost three, four lost two each, three lost one each. In seven out of the nine husbands there was a decided family taint; the wives were with one exception free from any such taint, and they were all healthy at the time of marriage. The lung affection ran in all the wives a very rapid course, terminating in several instances within twelve months, and being never prolonged beyond eighteen months. It cannot be supposed to have been caused by anxiety or fatigue in nursing the husbands, for the husbands were all in fair health so far as appearances went, and none of them succumbed to phthisis until long after their wives. All the wives, with one or two exceptions, bore children to their husbands, so that it is perhaps possible that infection took place through the fœtus; but about the state of health of the children nothing is said. Dr Weber seems to have been more disposed to think that the mere absorption of the seminal fluid led to the transference of the disease. But even if we admit that this was probably the case, it does not follow that there must have been an actual conveyance of a specific contagion. May not such cases merely afford another instance of that inexplicable influence of impregnation which stamps on the female organism the characters of the male, so that they can be transmitted long afterwards to offspring by a different male? Cases of this kind among the lower animals are well known to be frequent; and it is said that similar instances occur as the result of sexual intercourse between human beings belonging to different races. The following case, if not merely accidental, was probably an example of the same thing. A candidate for life insurance, whose mother had had two husbands, said that the first husband and several of his children had died of phthisis; the second husband was free from all tubercular tendency, but the eldest of the offspring of this marriage nevertheless became affected with the disease.

Dr Weber was acquainted with only thirty other consumptive husbands whose wives escaped phthisis. But in all likelihood this was an accidentally small number; and among twenty-nine consumptive wives who married healthy husbands, only one lost a husband from phthisis.

On this subject see the facts recorded in the 'Collective Investigation Record,' "Report on the Communicability of Phthisis;" and also a paper by Dr Burney Yeo, in which he skilfully states the case for regarding phthisis as a contagious disease ('Brit. Med. Journ,' April 18th, 1885).

This is no new doctrine. Morton wrote in 1697 : "I have often found by experience that an infected person may poison a bedfellow by a kind of miasm like that of a malignant fever." In Italy consumption was always regarded as an infectious disease. The demonstration of the presence of the bacillus of tubercle in most if not all cases of phthisis affords a ready explanation of their being contagious, if the fact is proved. That it is not readily communicated is certain, but it does seem to be so under certain favourable conditions.

If now we turn to consider what are the ordinary conditions which in clinical practice are concerned in bringing about the development of phthisis, we shall find that they may be roughly arranged in three groups.

i. Those which affect the constitution of the individual before birth.

ii. Those which affect his general health in the course of life,

iii. Those which affect the lungs themselves.

It is not always easy to say to which category a particular cause of phthisis belongs. It is enough for our present purpose if it be admitted that all these groups exist.

i. *Conditions which affect the original constitution of the individual from before birth.*—The first of these to be discussed is :

a. Hereditary transmission.—It is a matter of universal experience that in some families deaths from phthisis occur, generation after generation, with terrible frequency. Parents and their offspring are swept off in turn, so that sometimes there is hardly a survivor to maintain the stock. Actuaries are so impressed with these facts that whenever it can be ascertained, in reference to a candidate for life insurance, that he has lost a parent or more than one brother and sister from consumption, it is held at almost all offices that an addition to the premium is absolutely necessary to cover the increased risk, and if both parents have died of the disease, or more than two other near relatives, the "life" is generally regarded as almost uninsurable, at least on reasonable terms. It might at first sight appear strange that an augmented liability to what is (after all) only one among a great many other possible causes of death should be taken as diminishing to so great an extent the general "expectancy" of the candidate, but the requirements of the offices are in practice found to be fair and equitable.

From a scientific point of view, however, the question of the inheritance of consumption requires far more consideration than has generally been given to it. Among persons actually affected with phthisis, the proportion of cases in which the occurrence of a like disease can be traced in their relatives appears from certain investigations made by Dr Theodore Williams, and recorded in the 'Med.-Chir. Trans.' for 1871, to be 48·4 per cent. The patients were seen in private practice, so that the results are probably as little inaccurate in the way of omission as can be expected in such inquiries, although it would be an advantage, for the purpose of comparison, if we knew to what extent a similar family history exists in the population generally. But it is impossible to accept the figures given by Dr Williams, or any similar figures, as indicating in a scientific sense the extent to which consumption is transmitted by inheritance. The difficulty is often brought out very clearly by proposers for life insurance. A candidate has had perhaps two or three brothers who were consumptive, but one, he says, brought on the disease by dissipation and intemperance; another was in the army, and was stationed first in India and then in Canada at a few months' interval; a third may have got a chill in

bathing; and he winds up by declaring that phthisis has not been a "family complaint" after all. Now, among the cases collected by Dr Williams, 484 in number, in which phthisis was traced among the relatives of patients themselves phthisical, there were 120 in which the disease had existed in one or both of the parents, but 224 in which it affected only brothers or sisters. There can be no doubt that if inquiries had been made as to the existence of definite "exciting causes" of the disease in these cases they would have been found to be very often present. It is in fact impossible to draw a line anywhere between what might be called respectively "hereditary" and "accidental" phthisis. Perhaps there is no family in which the consumptive tendency is so strong that it could not be kept in abeyance by hygienic precautions if they were thoroughly and vigorously carried out, and, on the other hand, there are very few families, if any, in which the disease may not show itself in such members of it as systematically neglect their health, or are exposed year after year to unfavourable circumstances.

It is impossible at present to determine in what proportion of cases the so-called "family predisposition" to consumption implies the actual transmission of a definite tendency to this disease, and in what proportion of cases it is merely the expression of a general delicacy of constitution, or (as the Germans would say) "vulnerability," which renders those who are derived from certain stocks liable to be attacked by consumption in succession, as they happen to come under conditions suitable to its development. From the point of view of the insurance offices the distinction is not material, for in either case the demand for an enhanced premium is equally justifiable. One fact which tells strongly in favour of the opinion that family predisposition is often a mere vulnerability, is that the liability to consumption is believed to be much above the average in those who come from parents already failing in health from any cause, in those begotten by a father advanced in years, in those born of a very young mother, and also in the later offspring of a woman exhausted by frequent and rapid child-bearing. The same opinion is further supported by the circumstance that there is little evidence of a specially strong tendency to phthisis in the children of parents actually consumptive, one or both of them, at the time of procreation.

The experience of insurance offices as well as of private practice is that a phthisical tendency is more frequently transmitted by the mother than by the father.

β. *Diathesis.*—It is a very old suggestion that persons of a particular bodily frame and physiognomy are especially liable to tuberculous diseases, but little value can be attached to the statements of early writers on the subject, because "scrofula," as it was called, was often confounded with rickets or with congenital syphilis. And, according to Sir Thomas Watson, the numerous signs of the "scrofulous diathesis" varied widely with the "temperament" of the individual, whether "nervous," "sanguine," or "bilious." Obviously this was very confusing. It therefore seemed a great step in advance when Sir William Jenner in 1860 proposed to distinguish two separate diathetic states—tuberculosis and scrofulosis.

As leading features of *tuberculosis* he gave the following:—"Nervous system highly developed; mind and body active; figure slim; adipose tissue small in quantity; organisation generally delicate; skin thin; complexion clear; superficial veins distinct; blush ready; eyes bright, pupils

large; eyelashes long; hair silken; face oval, good-looking; ends of long bones small, shafts thin and rigid; limbs straight. Children the subjects of tuberculosis usually cut their teeth, run alone and talk, early."

He described *scrofulosis* as follows:—"Temperament phlegmatic; mind and body lethargic; figure heavy; skin thick and opaque; complexion dull, pasty looking; upper lip and alæ of nose thick; nostrils expanded; face plain; lymphatic glands perceptible to touch; abdomen full; ends of the long bones rather large; shafts thick."

Among the pathological tendencies of the former morbid conditions he mentioned not only "deposits or formations of tubercles," but "fatty degeneration of liver and kidneys, and inflammation of the serous membranes." To the latter he assigned "inflammation of the mucous membranes of a peculiar kind; so-called strumous ophthalmia; inflammation of the tarsi; catarrhal inflammation of the nose, pharynx, bronchi, stomach, and intestines; inflammation and suppuration of the bronchial glands on trifling irritation; obstinate diseases of the skin; caries of bone."

Many physicians still believe that Jenner's descriptions correspond with two great types, the recognition of which is really important in practice. But the idea of their representing different diatheses is quite incompatible with modern views as to the relation between tubercle and caseating affections of lymph-glands; no less incompatible with Buhl's theory of infection than with the more recent opinion that the glandular affections are themselves tuberculous.

There has never yet been any general consensus among observers of experience with regard to the outward signs which indicate a tendency to tuberculous diseases. Although many of those who die of phthisis present the characters, according to Jenner, of scrofula, a much larger proportion are ill-grown with no definite indications of a special diathesis. On the other hand, his description of tuberculosis seems to be made up of those characters which, while not incompatible with rapid and symmetrical growth or with physical beauty, show a want of real vigour and power of resistance to disease in general.

Mr Francis Galton and the late Dr Mahomed recorded in the 'Guy's Hospital Reports' for 1881 the results of "An Inquiry into the Physiognomy of Phthisis by the Method of Composite Portraiture." Their conclusions seem to bear out the view just stated. For although they were able to obtain from the photographs of 442 phthisical patients two types of face —the one of narrow, ovoid shape, the other a broad face with coarse features —yet this was only by the careful selection of a few out of the whole number; and they actually found a larger proportion of narrow, ovoid faces among patients who were not phthisical than among those who were.

The fact is that the words scrofula and struma have been applied in so loose and arbitrary a way, quite independently of chronic swelling of the cervical lymph-glands, or even of evidence of caseous disease in any part of the body, that it is better to avoid their use until we have a collection of facts based on the accurate use of clinical or pathological terms. Moreover, tendencies to scrofula or tubercle which are never carried out can scarcely be the subject of useful discussion, nor can the exploded doctrine of temperaments be revived until we again accept the four Galenical humours and their eucrasia or dyscrasia.

γ. *The phthinoid chest.*—There still remains the question whether an inherited tendency to phthisis is indicated by any particular *configuration of the*

chest. Dr Gee describes two shapes of chest as occurring in "phthinodes," or persons predisposed to consumption.

One of them he terms the "alar," or "pterygoid" chest, following Galen and Aretæus, who used the same name for it centuries before; this, he says, is narrow and shallow, the antero-posterior diameter being especially small, and the angles of the scapulæ projecting like wings; its peculiarities depend upon a drooping or undue obliquity of the ribs, as the result of which the shoulders fall and the length of the thorax from above downwards is increased; the alar appearance is caused by the falling of the shoulders. The pterygoid chest is often accompanied by a prominent throat, due to a long neck, and by the head being carried unduly forwards.

The other is called by Dr Gee the "flat" chest; this, instead of being rounded, is flat in front, the rib cartilages losing their curve and becoming straight. Nay, the sternum may actually be depressed below the level of the costal ends of the cartilages. Now, there can be no doubt that it is very important to be on the look-out for flatness of the chest in judging the configuration of this part of the body. Persons who are extremely flat-chested often have broad shoulders, so that, as one stands facing them, one might fancy them to be by no means ill developed. Traube and other German writers lay great stress on the significance of a flat chest, as indicating a liability to consumption, and Dr Wilks used to insist on it strongly. But the present writer must confess that he has never been able to satisfy himself of the existence of any definite relation of this kind. It seems a mistake to contrast the "flat chest of phthisis" with the "rounded chest of bronchitis," because the latter is merely an acquired condition and the result of that disease, of which it is no doubt an important clinical sign. Neither the alar nor the flat chest is seen among phthisical patients with more frequency than other varieties of ill-shapen chest, which so often result from neglect during childhood, and are consequently so very common among the poorer classes of the population. And it is difficult to say how far a badly-formed chest, in relation to the liability to phthisis, is important as interfering with the play of the lungs, and whether its significance is not merely that it is an indication of a defective development of the body in general.

Again, one cannot dissociate congenital from acquired deformities of the thorax in regard to their possible influence on the subsequent occurrence of tubercular disease of the lungs. Freund, in 1859, maintained that what caused a small and contracted chest was often a premature ossification of the cartilage of the first rib, occurring even in early infancy. It is worthy of notice that Dr Hutchinson, in advocating the use of the spirometer, did not suggest that a defective vital capacity of the lungs indicated a tendency to phthisis, but rather that it was a sign of the actual presence of the disease at an early stage. Again, deformity of the chest from lateral curvature seems certainly not to carry with it any increased liability to consumption. It appears to be very doubtful whether the habit of stooping at a desk, or in the work of a tailor or shoemaker, or weaver, although it cannot but be injurious to the health, specially favours the development of phthisis. Nor is there evidence that the foolish practice of compressing the base of the chest by stays is capable of producing such an effect.

ii. *The conditions which affect the general health of the individual in the course of life,* and act as causes of phthisis, are both numerous and varied.

a. Foremost among them is the *habitual breathing of air rendered impure by overcrowding or by defective ventilation.* It may, indeed, be a question whether this should not be placed under the third head, as affecting the lungs themselves rather than the general health. But on the whole it may fairly be dealt with here, leaving for separate discussion the no less important influence of such impurity of the air as results from the presence of minute particles of mineral or organic substances—fine grit, coal-dust, powdered clay, flax-dust, or cotton-dust.

The organic matter exhaled during respiration appears to be directly poisonous. Dr Parkes cites some experiments made by Gavarret and by Hammond with expired air from which the carbonic acid and water had been removed, so as to leave only the organic matter; a mouse placed in it died in forty-five minutes. Dr Parkes says that he has known instances in which breathing for three or four hours air contaminated by having been previously used in respiration, caused headache and febrile symptoms which lasted one or two days. It is important to remember that such organic substances probably differ from gases like carbonic acid in having far less tendency to rapid diffusion through the atmosphere; they readily adhere to textile fabrics, especially those which are dark coloured, and cling to them obstinately. Every physician is familiar with the peculiar odour belonging to the clothes of the women and children of the poorer classes; one perceives it as soon as they enter the out-patient room, even though they may have just been walking in the open air for a considerable distance. Precisely the same smell is constantly to be perceived in the rooms in which these people live. The organic matters which cause it cannot be removed by merely causing a current of air to blow through a room for a few minutes in the day; still less will they escape through a door towards which there is no active draught. In all probability the only way of getting rid of them is by oxidation; and one may speculate as to the part which ozone is likely to play in accelerating their destruction, and also as to whether they do not disappear more rapidly when they are exposed to full sunlight than in comparative darkness.

Strictly speaking, overcrowding and defective ventilation are not convertible terms; but in practice we scarcely ever meet with one of them apart from the other. It is not, indeed, inconceivable that one person or a few persons occupying a room of good size should so close up all the openings into it as to render the air impure. But, on the other hand, there is never overcrowding without bad ventilation, because when many persons are huddled together in a small space, the needful admission of fresh air always exposes some of them to cold draughts, the fear of which is sure to lead to the shutting up of one aperture after another.

The proof that impure air is a cause of phthisis rests mainly upon the evidence of statistics as to the frequency of the disease among soldiers and certain classes of workmen and among the inmates of prisons. As regards soldiers, a Royal Commission upon the Sanitary Condition of the Army, which reported in 1858, brought to light the fact that the death-rate from consumption in all branches of the service was in excess of that of the civil population of large towns, and (what was most remarkable) that among the Foot Guards it was more than twice as great as that of the civil population. The only explanation that could be offered was that it came from defective barrack accommodation, since neither the clothing of the soldier, nor his food, nor the nature of his occupation, could be supposed to be the cause of it.

There was evidence that in barrack dormitories the cubic space actually given to each man was often not more than one half or two thirds of the amount of 450 feet, which was the minimum allowed by regulation. It was also shown that the air in these rooms became offensive before morning. The accuracy of the conclusion at which the Commission arrived has since been confirmed by the fact that a great fall in the consumptive death-rate, especially among the Foot Guards, has followed the introduction of sanitary improvements.

As to workmen, we have evidence given by Dr Guy before the Commission of Inquiry into the State of Large Towns, of which the Duke of Buccleugh was president, and which reported in 1844. Dr Guy had most elaborately investigated the relative liability to phthisis of different classes of the population of London. He found that the disease was more fatal to artisans than to tradesmen, and more fatal to tradesmen than to professional men and the upper classes. Even hawkers, standing about in the streets and exposed to all inclemencies of weather, had the advantage over men employed in workshops. Among printers he instituted a very close comparison as to the frequency of symptoms of lung disease, arranging the men in classes according to the amount of air-space in the rooms in which they worked. Of 104 men having less than 500 cubic feet of air to breathe, 13 had suffered from blood-spitting and 13 others from catarrh; of 115 men having from 500 to 600 cubic feet of air, 5 had suffered from blood-spitting, 4 from catarrh; of 101 men having more than 600 cubic feet of air, 4 had suffered from blood-spitting and 2 from catarrh.

As for prisoners, there is the contrast between two prisons of Vienna cited by Dr Parkes in his 'Practical Hygiene.' In the Leopoldstadt prison, which was very badly ventilated, there died in the years 1834–47, 378 prisoners out of 4280, or 86 per 1000; of whom no fewer than 220, or 51·4 per 1000, died from phthisis. In the well-ventilated House of Correction in the same city there were from 1850 to 1854, 3037 prisoners, of whom 43 died, or 14 per 1000; and of these 24, or 7·9 per 1000, died of phthisis. Diet and mode of life were, it is believed, the same in both establishments. It is a flaw in the case that the average length of the periods during which the prisoners were detained in each prison is not given; but Dr Parkes thinks that no correction on this ground, even if any correction were needed, could account for the discrepancy in the death-rate. The great prevalence of phthisis among prisoners in general was long ago pointed out by Dr Baly, as the result of an examination of the 'Records of the Millbank Penitentiary.' But he was unable to determine what part in the ætiology of the disease should be assigned to defective ventilation, and what part to cold, poorness of diet, want of active bodily exercise, and a listless or dejected state of mind.

Indeed, it is obvious that only in very exceptional instances can overcrowding or defective ventilation be so isolated from other conditions injurious to health, as to be proved the main cause of phthisis. But when once their power to produce the disease is admitted—and there is a strong probability in its favour—one cannot doubt that they must play a most important part in producing the disease among the poor generally. For example, a very important investigation was made by Dr Greenhow in 1860 and in 1861 as to the origin of the great mortality from phthisis among the workpeople employed in many of the largest industrial occupations of the country, and he found that the inhalation of dust of various kinds is the

main cause of the disease so far as these persons are concerned. But Dr Greenhow himself pointed out that another cause was certainly the "working in ill-ventilated or over-heated factory rooms or workshops, as in those of some of the silk mills of Coventry, in the domestic weaving shops, and in the watchmakers' factories and workshops of the same city, in the button-makers, and various other workshops of Birmingham, in the factory rooms of Blackburn and Nottingham, and in many domestic shops and warehouses." Abundant details in support of this conclusion are to be found in the report which he furnished to Mr Simon.

β. *Food insufficient in quantity and bad in quality* is believed to be among the conditions which tend to produce phthisis, but it would be difficult to adduce any positive proof of this by cases in which it could be shown to be the principal cause of the disease.

γ. *Child-bearing* seems often to play an important part among the causes of phthisis in women. One would certainly think it likely that pregnancy would be apt to bring on the disease in persons naturally delicate or predisposed to it. But it is a well-known and a very remarkable fact that during gestation even the most rapid form of consumption scarcely ever runs on to a fatal issue. The patient almost always survives until labour is over, after which the symptoms become more urgent than ever, and death may follow rapidly. In many cases the disease appears to begin during lactation, or is attributed to the occurrence of profuse puerperal hæmorrhage. According to Dr Pollock it is not merely prolonged suckling that seems to set up phthisis, but the mere fact of suckling at all. Cases associated with child-bearing generally run a particularly acute course.*

δ. *Alcoholic intemperance* is believed to play an important part in the ætiology of phthisis. Clinical experience shows that drunkards, both those who are engaged in the liquor traffic and those who are not, die in large numbers of this disease. No doubt it is often difficult to exclude the operation of other causes, such as defective food, bad ventilation, and exposure to cold and wet. But the author's impression is that the alcohol itself is really concerned in the result.

On this point, however, there is much difference of opinion and some writers have believed that the moderate and even the excessive use of alcohol is rather prophylactic against than productive of phthisis. Observations in the United States have been cited in favour of this view. Dr Walshe and Dr Wilks do not accept intemperance as a cause of consumption, while Dr Williams has no hesitation in doing so. Lately Dr Owen, of St George's Hospital, found that among the phthisical patients attending there 50 per cent. were, on their own showing, excessive drinkers, while only 33·5 per cent. of the other patients confessed to habits of intemperance; or, put in another way, of 100 non-consumptive patients, 41·5 professed to be temperate, of 100 consumptives, only 23.

It must be remembered that drunkenness goes with poverty, exposure, and unhealthy living, which are acknowledged to be predisposing causes of bronchial and alveolar catarrh and so of phthisis. Again, most of the publicans, barmen, and other intemperate classes who consult a specialist will be subjects of the disease for the treatment of which he is esteemed. Is phthisis less

* These considerations are so important in regard to life insurance, that it is only prudent to count all deaths of mothers "in childbed" or "after delivery" as due to phthisis, unless there is explicit evidence of previous good health.

common in Italy than in London, in Constantinople than in Glasgow? Are total abstainers more exempt from consumption than temperate persons? These are questions not readily answered.

It has of late been a favourite idea with some physicians that alcoholic phthisis is usually fibroid rather than tuberculous in character. Huss, of Stockholm, suggested this opinion about 1850, and it has been supposed to derive support from the fact that alcohol causes a growth of interstitial fibrous tissue in the liver, and perhaps also in the kidneys and elsewhere throughout the body. But there is no other instance of the production by alcohol of a local fibroid change spreading slowly through an organ from one particular part. And it is certain that tubercular peritonitis of the most typical kind is often seen in intemperate persons who also have hepatic cirrhosis. We have given reason to believe that "fibroid phthisis" is not essentially distinct from the more purely tubercular form of the disease (p. 203). It is only the extreme type of what almost every case of phthisis shows in some degree, a power of repair by cicatrisation, adhesions and fibroid thickening. In the *post-mortem* room the diagnosis of fibroid phthisis, which during the life of the patient had been partially based upon a history of alcoholic intemperance, has been repeatedly falsified by the disease proving to be most typically tuberculous.

ε. *Diabetes* is a frequent cause of a phthisis which is peculiarly pneumonic in character; its relations to the ordinary tuberculous disease are still doubtful. It may hereafter be shown that the diabetic affection of the lungs is really not distinct from the more acute variety of tuberculous phthisis. This point will be discussed in the chapter on Diabetes.

ζ. *Syphilis* is sometimes followed by the development of a destructive disease of the lung, which on *post-mortem* examination is found to be typically tuberculous. No fewer than thirteen cases of this kind occurred at Guy's Hospital between the years 1863 and 1873. But one must not forget that among syphilitic patients, especially in hospital practice, many are intemperate, destitute, and every way careless of their health. Consequently it is quite possible that the relation between the phthisis and the syphilis may, in many of the instances just referred to, have been one of mere coincidence. On the other hand, we have in the production of lardaceous changes in the viscera an illustration of the fact that syphilis may set up a morbid process which is nevertheless capable of arising from other causes in persons who have not had syphilis. It therefore seems not unlikely that the disease, by depressing the general health, may constitute the starting-point of a tuberculous lung affection. The clinical fact is attested by many observers of the highest authority that the administration of iodide of potassium is often followed by striking improvement in the physical signs and symptoms of what had appeared to be phthisis in a syphilitic subject, or even by their complete subsidence. It is true, as we shall hereafter see, that any treatment which is capable of improving the general health of a consumptive patient may also be followed by an apparent cure of the pulmonary affection. Still it is obvious that a far simpler way of explaining the striking action of antisyphilitic remedies in the cases in question would be to recognise a syphilitic disease of the lungs capable of simulating phthisis, but pathologically distinct.* The characters of such a disease, distinct from true

* My difficulty in adopting this view is that I have never been able to satisfy myself of the existence of such a disease. There is, of course, no reason why gummata should not develop themselves in the lung as well as in any other organ; but the recorded instances

or tubercular phthisis occurring in a syphilitic subject, have been set forth in the chapter on chronic inflammations of the lungs (*supra*, p. 156).

III. *The conditions which affect the lungs directly* and so act as causes of phthisis appear to be mainly the following.

a. Inhalation of dust.—Ramazzini in his work, 'De Morbis Artificum,' published in 1703, seems to have been the first to point out that certain classes of workmen are liable to have their lungs injuriously affected by dust given off by the materials which they employ. In the present century the question has been studied very thoroughly, especially in England and in Germany; and Zenker has proposed to term the pulmonary diseases due to this cause *Pneumonoconiosis* (κόνις = dust). The nature of the mischief set up in the respiratory organs by breathing dust varies widely in different cases. It may be a mere catarrh of the trachea and bronchial tubes, leading to chronic cough and emphysema, and finally destroying life (if at all) by dilatation of the right side of the heart and dropsy. But in many instances it consists in local indurations of the lung substance, which may ultimately lead to the formation of cavities, and spread through the pulmonary tissue from apex to base, as in phthisis. Indeed, it has been usual to speak of "miners'," "weavers'," and "knifegrinders' consumption." Pathologists, however, have almost without exception maintained that the lesions found in such cases are independent of the presence of tubercles. Whether tubercle was regarded as a deposit from the blood in a special dyscrasia, or as a particular kind of new growth, it seemed equally logical to deny the possibility of its being due to a mere local irritation of the tissues. These views were of course still further strengthened by the wide adoption of Niemeyer's doctrines with regard to the non-tubercular nature of phthisis in general. And, still more recently, the theory of the local development of tubercles, as the result of tissue infection from lesions themselves inflammatory, seemed to remove all difficulties, by affording a ready explanation of the fact (which cannot be denied) that in many

in which such lesions are stated to have been found in the *post-mortem* room seem to me to be generally open to more or less doubt. Take, for example, Dr Goodhart's case, of which there is a coloured plate in vol. xxx of the 'Pathological Transactions.' He himself mentions that Dr Reginald Thompson considered the supposed gummata to be relics of pulmonary hæmorrhage (*loc. cit.*, p. 236), and they certainly do not seem to me to differ at all from the encapsuled cheesy masses which are often found in ordinary cases of phthisis. Moreover, one cannot admit in the present state of pathology that the undoubted grey tubercles which also existed in large numbers in Dr Goodhart's case were merely secondary results of the caseation of gummata, causing infection of the pulmonary tissue, although a few years ago such an interpretation of the facts would to most observers have appeared satisfactory. In vol. xxviii of the 'Pathological Transactions' may be found a detailed discussion of the relations between syphilis and phthisis by Dr Goodhart (pp. 313—329); and there are also cases in which the lungs appeared to contain syphilitic lesions, recorded by Dr Pye-Smith and Dr Green (*ibid.*, pp. 331, 334). But I fail to discover, either there or anywhere else, such examples of extensive lung disease, obviously and unmistakably different from tubercular phthisis, as certainly ought now and then to be met with if the views held by these observers were correct. Remembering how for at least a quarter of a century pathologists have vied with one another in the attempt to carve out of tubercular phthisis as many different forms of disease as possible, which they have supposed to be distinct from it, one is surely justified in scrutinising all such statements very closely. Dr Moxon when pathologist at Guy's Hospital was much impressed with the fact that in several syphilitic cases he found in the lungs indurated patches, the centres of which had sloughed away so as to form cavities in the anterior of which shreddy masses were still hanging. And I think that he came to the conclusion that such appearances were almost characteristic of a syphilitic affection. But the difficulty is that they are altogether unlike those which syphilis is known to produce in other organs. For my own part I feel that the relations between this disease and phthisis still require to be investigated afresh by unbiassed observers.—C. H. F.

cases the more recently affected parts of the lungs show tubercles, when none can be discovered in the parts which were earliest diseased. This theory, however, is wrong, as we have attempted to prove in the chapter on tubercle (vol. i, p. 76). According to the conception of the tubercular process as a modification of inflammation, propounded in that chapter, there seems to be no necessity to adopt the views of those who separate all the Pneumonoconioses from phthisis.

It may perhaps be thought that the reports of autopsies are conclusive in the matter, since they generally affirm that the appearances found in the lungs are altogether different from those which are seen in tubercular cases. But several points must be borne in mind. One is that the character of the lesions in the lungs is often greatly obscured by the discolouration from the foreign particles deposited in the pulmonary tissue. Another is that as a rule the destructve diseases of the lungs which are due to dust-inhalation occur at rather a late period of adult life, and advance slowly to a fatal termination; in both respects they resemble that form of ordinary phthisis which is regarded by so many pathologists as distinct from the tubercular, and termed fibroid. The remarks made (*supra*, p. 209) with regard to the morbid anatomy of the so-called fibroid phthisis are indeed strictly applicable to the descriptions which are to be found in modern medical literature of the diseased lungs of miners, potters, or stoneworkers. The use of the word tubercle is often carefully avoided in such descriptions, but mention is almost invariably made of "nodules," from the size of a millet-seed to a pea, scattered in the pulmonary tissue, of an opaque, greyish-yellow colour, and perhaps with a small central cavity. The microscope is called into requisition for the purpose of proving the negative fact that the nodules in question fail to present whatever histological structure may happen to be regarded as characteristic of tubercle at the time when the observation is made. Or possibly it is asserted that they are distinguished by their hardness and resistance to pressure, the fact being overlooked that undoubted tubercles themselves, if they fail to caseate, undergo conversion into precisely similar bodies. We are therefore justified in hesitating to accept the validity of such reports of autopsies until they are confirmed by some pathologist whose mind is open with regard to the general question of the unity of phthisis.

Nor does it appear that the clinical course of the lung diseases due to dust-inhalation differs essentially from that of the more chronic forms of phthisis. Dr Greenhow, who is one of the leading authorities on the subject, has insisted (in the 'Pathological Society's Transactions,' vol. xvi, p. 61, *et passim*) on "the coincidence of a cool skin and a quiet pulse with wheezy asthmatic cough and copious muco-purulent expectoration," as rendering "the diagnosis from tubercular phthisis comparatively easy." And elsewhere he says that "shortness of breath almost invariably precedes by some considerable time the appearance of cough, and the patient is often ailing for many years before being disabled from work." But with regard to the suggestion that the early dyspnœa is distinctive, it must be remembered that bronchitis and emphysema are as marked effects of the inhalation of dust as are pneumonic processes. Moreover, it is possible, as Seltmann suggested in the 'Deutsches Archiv' for 1867, that when foreign matters are deposited in the lungs in large quantities, the respiratory surface may be so much diminished that anæmia and dyspnœa are necessary consequences.

Since the first edition of the present work appeared, the bacilli of tubercle have been recognised in several cases of Pneumoconiosis, so that the justice of the conclusions of the last paragraph may be regarded as established.*

The materials which give off dust that may enter the respiratory organs are of various kinds. They may be classified as follows.

(1) *Carbon* from coal, charcoal, soot, or smoke. The resulting affections of the lungs have been grouped together under the name of *Anthracosis*, a term originally proposed by Stratton in 1837.

As far back as 1813 Pearson, in the 'Philosophical Transactions,' threw out the suggestion that the black discolouration of the lungs and of the bronchial glands, which is found in most adults, but not in children, consists of particles of carbon "introduced with the air in breathing," and originally "derived from the combustion of coal, wood, and other inflammable materials." A similar opinion was expressed by Laennec. In 1831 Dr Gregory recorded the case of a labourer in the coal mines of Dalkeith; both lungs were throughout of a uniform coal-black colour, and yielded, when washed, a dark matter, which was found by Dr Christison to resist the action of concentrated nitric acid and of chlorine, and to yield by distillation products just like those which result from the distillation of coal. The conclusion seemed irresistible that the organs had been discoloured by the penetration of coal-dust from without. "Spurious melanosis," therefore, became generally adopted as the name for this condition of the lungs, which was understood to be only an extreme degree of the normal darkening. But Hasse in 1841, and Virchow in 1847, each refused his assent to the proposition that the black matter was derived from the air inhaled in the act of breathing. It was not until 1860 that a case was recorded by Traube in the 'Deutsche Klinik,' which finally established the real existence of anthracosis. The patient was a man who for about twelve years had been engaged in loading and unloading wood-charcoal, during which occupation he was constantly exposed to the dust of it. He had for a long time been accustomed to expectorate a black substance, and when he died his lungs were found to be almost everywhere of a black colour, yielding to pressure a black frothy fluid which stained the fingers like thin Indian ink. Both in the sputa, and in the pulmonary tissue, there were found numerous pieces of the most irregular forms, with angular pointed processes. That these were fragments of wood-charcoal was evident not only from a comparison of them with particles of the charcoal brought directly from the place where the man had worked, but also from the fact that some of them showed the circular disks characteristic of the woody fibres of coniferous trees. And some had a reddish tint instead of being quite black; this was due to their being imperfectly carbonised. A second similar case came before the same observer six years later. Traube did not, indeed, himself at first conclude from these two cases that the finely granular material which gives a black colour to healthy lungs as life advances is also inhaled carbon, but in the meantime the current of opinion began to set steadily in that direction. It was greatly helped on by Zenker's discovery (to be presently more fully set

* Two cases of grinders' phthisis at Brompton Hospital were kindly communicated to the editor by Dr Theodore Williams, and three cases of potters' phthisis were published by Mr Watson Cheyne in a valuable paper on the "Relation of Micro-organisms to Tuberculosis," in the 'Practitioner' for April, 1883 (p. 294).

forth) that the lungs of those who work with red oxide of iron become full of red particles, occupying precisely the same position as the black. In 1866 Virchow took an opportunity of recanting his former views, and Rindfleisch in his 'Handbook,' ascribes to anthracosis almost every form of black discolouration of the pulmonary tissue. Modern histology has, indeed, removed one of the greatest obstacles in the way of the acceptance of such a doctrine, by showing that leucocytes are capable of taking particles of foreign materials into their substance, and that animal membranes are permeable by such particles in a way that formerly was hardly suspected. In 1858, in describing in the 'Edinburgh Medical Journal' a specimen of "miners' lung" that had been sent to him from Scotland, Virchow had insisted that the seat of the most abundant deposition of black matter accorded ill with the theory of a progressive absorption from the air-passages. Scarcely any of it, he said, was found in the interior of the pulmonary alveoli; and in their walls it lay not beneath the endothelium, but between the elastic fibres and the connective tissue. Moreover, very much larger amounts of it occupied the interlobular and peribronchial fibrous tracts, and accumulated beneath the pulmonary pleura. Nay, it was even present in the costal and diaphragmatic pleura, as well as in the bronchial glands. At Guy's Hospital we once found some of it free in the upper and back part of the pericardial space, separated by the fibrous wall of the sac from an intensely black gland that lay just outside. This question has been studied experimentally by von Ins, whose observations are recorded in the 'Arch. f. exp. Pathologie' for 1876. He injected cinnabar into the air-passages of dogs, and found that the particles were rapidly taken up by cells which he believed to be altered leucocytes, so that five days later scarcely any pigment was left in the pulmonary alveoli; within six hours after the commencement of the experiment some of it reached the bronchial glands, being first deposited in their cortical layer, but ultimately reaching their medullary structure; much, however, remained in the lungs, being accumulated in the connective tissue, between the lobules, round the vessels and the tubes, and beneath the pleura. In other words, von Ins found that its distribution corresponded precisely with what had been described by Virchow in the case of the miner's lung.

The phthisis associated with anthracosis is attended with one special symptom, which may be conveniently mentioned here. This is the "black spit" which is often ejected in considerable quantities and for a long time, even by miners who have entirely ceased to follow their occupation. There is no doubt whatever that it is often due to the gradual disintegration of the blackened and infiltrated parts of the lungs, or comes from vomicæ such as are sometimes found after death to be full of a black liquid. Thus, Dr Greenhow showed to the Pathological Society in 1869 the lungs of a collier who about ten days before his death suddenly spat up a considerable quantity of matter closely resembling black paint, and continued doing so to the amount of four or five ounces daily until he died; in the right lung there was a large irregular cavity, containing a quantity of black pulpy residue. But the former of Traube's two cases shows that the sputa may contain much black matter without there being any lesion of the pulmonary tissue beyond the anthracosis; and he is no doubt right in insisting on the detection of fragments of elastic tissue as the only proof that ulceration is going on.

(2) *Oxide of iron.*—In 1864, in discussing the subject of anthracosis, Friedreich asked the question why, if the black lungs and bronchial glands of coalminers were due to inhaled carbon, the workers in red sandstone quarries should not have the corresponding organs reddened by the dust to which they were exposed? Now, it happened that Zenker had at that very time in his possession the lungs of a woman who had, for seven years before her death, been engaged in making the little paper books in which gold-leaf is laid. The paper has to be coloured red with peroxide of iron, and this is rubbed in by means of a piece of felt. The occupation is a very dusty one; and the woman's lungs were found after death to be throughout of a bright brick-red colour, so that their cut surfaces looked just as if they had been daubed over with red paint. The microscope showed that fine granules of oxide of iron were present in all parts of their texture, beneath the pleura, in the interlobular fibrous septa, in the peribronchial sheaths, in the walls of the alveoli, and even in cells occupying their interior. In the twentieth volume of the 'Pathological Transactions' there is a coloured drawing of a thin section of this lung, taken from a specimen in the possession of Dr Wilson Fox; the tint is, however, far less red and more brown than that represented in Zenker's drawings published in the 'Deutsches Archiv' two years before. Zenker proposed to name the affection Siderosis (σίδηρος = iron). In 1874, Merkel, in 'Ziemssen's Handbuch,' was able to refer to seven other cases, one of which followed the use of red oxide of iron for polishing glass. He had also met with two instances in which the lungs were blackened by the black oxide of iron, and one in which ferric phosphate was present. There was no difficulty in detecting the iron in the sputa by hydrochloric acid and ferro-cyanide of potassium.

(3) *Quartz, sandstone, or clay.*—That workmen whose occupations expose them to the dust of siliceous or argillaceous materials are very prone to die of phthisis has long been known. Very full information with regard to the excessive mortality from this cause in certain districts of England is contained in a paper by Dr Greenhow in Mr Simon's third 'Report to the Privy Council,' published in 1861. Merkel has recently proposed to term the resulting disease of the lungs *Chalicosis* (χάλιξ = gravel). The presence of silica in the pulmonary tissue seems to have been first detected chemically by Dr Peacock and by Dr Greenhow. Kussmaul, however, has recently shown (in vol. ii of the 'Deutsches Archiv') that this substance is present in greater or less quantity in the lungs of all persons (though not in those of a fœtus), having doubtless been derived from the dust of the streets and roads blown up by the wind. In a railway-signal man, stationed in a very sandy district, Meinel found that silica actually formed as much as 18·2 per cent. of the ash of the lungs after incineration; even in a stonemason, who died of phthisis, and whose lungs were analysed by Kussmaul, the amount was not greater than 24·7 per cent. of the ash. Under the microscope the particles of silica may be seen as bright bodies of round or angular shape. Among the classes of workmen who suffer from this cause are, of course, stonemasons and millstone makers. Still more fatal is the grinding and polishing of steel instruments—from scythes to needles—such as is carried on in Sheffield, Birmingham, and elsewhere. The trades, as is well known are subdivided to the greatest possible extent; but whether the exact nature of the work be needle-pointing or fork-grinding, or the sharpening of fish-hooks, the result differs but little; a large number of the men die prematurely, some

between twenty and thirty, and more between thirty and forty; very few survive the age of forty without suffering more or less from pulmonary symptoms. This has been long known at Sheffield as "grinders' rot." A point of some importance is that what is termed "dry-grinding" is much more injurious than "wet-grinding;" the difference is that in wet-grinding the wheel, as it revolves, dips into water and deposits a large part of the dust which would otherwise be carried into the air. But even wet-grinders are greatly exposed to dust in "hacking" their grindstones, which generally has to be done every day or even oftener. Another dangerous occupation is pearl-shell cutting. Potters, again, are exceedingly apt to be attacked with phthisis; "flat-pressers" suffer more than "hollow-pressers;" but the worst off of all are "china-scourers," whose business it is to rub off the loose flint powder from the china with sandpaper, after it has been baked. By wearing respirators, and other preventive means, the terrible mortality in these trades has of late years been happily reduced.

(4) *Cotton and flax.*—Among occupations which expose the persons engaged in them to injury from the inhalation of vegetable matters may be especially mentioned the carding of cotton, and the hackling and carding of flax. Fragments of these materials may not have been detected in the pulmonary tissue after death; but in two men who had worked in the manufacture of tobacco, Zenker found "brown spots in the lungs and in the bronchial glands, evidently due to the deposition of powdered tobacco."

Pathology of these pneumonoconioses.—It is clear, then, that foreign particles of various kinds may find their way into the lungs, may be deposited in the pulmonary tissue, and may either remain there or be ultimately transported to the bronchial glands. But it by no means follows, as a matter of course, that their presence must be injurious. Indeed, since the true nature of the black material found in the lungs, whether in health or in disease, has of late years become established, increasing doubts have been expressed as to whether it can fairly be regarded as the cause of inflammatory or destructive changes. In Traube's first case, referred to above, there was not the slightest trace of any newly-formed connective tissue nor of induration of the substance of the lungs; the patient's symptoms were probably due to chronic pericarditis, accompanied by double pleurisy, which seemed to have begun three months and a half before his admission into the hospital and four months and a half before his death. It is true that this instance does not prove that in other persons irritation of the pulmonary tissue cannot be set up even by wood-charcoal, still less that such a result may not be caused by particles of coal or of lampblack, impregnated as they may be with various chemical products. But the experience of German observers seems to be strongly adverse to such a conclusion. Seltmann, of Zankeroda, near Dresden, asserts positively in the second volume of the 'Deutsches Archiv' that among the coalminers of that district there is generally no overgrowth of connective tissue whatever, even in lungs full of black deposits, and that the formation of cavities is of very rare occurrence. As for pulmonary tuberculosis, he thinks that the inhalation of carbon is actually antagonistic to its development—an opinion which appears to be shared also by Merkel.

If, therefore, English miners are greatly more liable to destructive disease of the lungs, the explanation is in all probability that the galleries in which they work are so often badly ventilated; the real cause of their being

attacked with phthisis is not that the air which they breathe contains coal-dust or smoke, but that it is rendered impure by the products of respiration, exactly as in crowded workshops or sleeping-rooms. Indeed, in his fourth report, Mr Simon drew special attention to the fact that the colliery-miners of Durham and Northumberland differ from all other miners in not suffering any important excess of pulmonary or other disease; and argued that the reason for this is the good ventilation of the mines in which they work; but he was still disposed to think that this operated mainly by removing the coal-dust and powder-smoke which would otherwise be diffused through the air. The great heat to which miners are exposed is probably another factor in the ætiology of the lung diseases to which they are liable.

On the other hand, there seems to be no doubt that destruction of the pulmonary tissue is an almost necessary result of the entrance of some other kinds of foreign particles into the lungs. Merkel says that in his cases of siderosis fibroid changes were never wanting even where the patient had died of some independent disease. And among needle-grinders, potters, and other classes of workmen exposed to the inhalation of flint-dust or finely-powdered clay, the prevalence of phthisis is far too great to be accounted for in any other way. Merkel says that the only opportunity which he ever had of examining a grinder's lung was in the case of a boy, aged sixteen, who was accidentally killed after having been at his occupation four and a half years, and who was said to be in perfectly good health. His lungs already contained small, tough, black nodules of the size of a pin's head, as well as minute particles of sandstone and of iron.

Whatever may be the nature of the irritant which sets up destructive changes in the lungs, the resulting affection appears to have exactly the same characters. This was strongly insisted on by Dr Greenhow when in 1865–70 he successively showed at the Pathological Society's meetings the lungs of a collier, a copper-miner, a razor-grinder, a stoneworker, a potter, a flax-dresser, and a pearl-shell cutter. And it is to be observed that the lungs may be almost if not quite as black in those patients in whom the affection was set up by sand or clay as in the miners themselves. The explanation is doubtless that there has been bronchitis also, and that this, by interfering with the natural ciliary action of the bronchial mucous membrane, causes the particles of carbon in dust and smoke, which in greater or less amount are inhaled by everyone, to become deposited in the pulmonary tissue in excessive quantity instead of being swept back into the trachea. But, further, when any destructive process is set up in a lung, the affected parts are very apt to become more deeply blackened than the rest of the organ. One of the points on which Virchow insisted when he upheld the view that the discolouration was due to a pigment derived from altered hæmatin, was that even in children the development of phthisis was sometimes attended with a blackening of the tissue which at their age could only be regarded as altogether exceptional. The experiments of von Ins, referred to above (p. 242), enable us to understand why the foreign material should accumulate in newly-formed fibroid tissue, and even in fibroid tubercles of a diseased lung, just as it does in the connective-tissue tracts of the healthy organ rather than in its alveoli.

The complete identity of the pulmonary affections to which so many different classes of operatives are liable, is in itself sufficient to show that a

common pathological process is concerned in producing them. And if the phthisis of colliers is but the result of generally unhealthy conditions, the whole question is brought at once under that of chronic or fibroid phthisis in general, which has been fully discussed already. But there is one other argument, which has generally been completely ignored by those who, like Dr Greenhow, have asserted that the lung diseases of those who work in factories or in mines are often merely due to a chronic interstitial pneumonia, and should be sharply distinguished from those in which tuberculosis plays an essential part. It is that since the irritants which excite these several diseases act upon all parts of the lungs alike, the resulting lesions must necessarily be diffused uniformly over all parts of them unless some other factor is involved in their ætiology besides the mere irritation. But as a matter of fact, it is found that in most cases they attack one lung before the other; they almost invariably begin in the upper lobe, and spread gradually downwards through the organ towards its base; and we have seen that such a course is almost constantly taken by tubercular affections of the lungs, indications of it being apparent even in many cases of miliary tuberculosis.

β. *Cold and wet.*—No idea is more firmly rooted in the public mind than that consumption is often the result of accidental causes, such as getting chilled by remaining in wet clothes, or by exposure to a draught when heated in dancing; and generally that there is danger, especially in those who are hereditarily predisposed to the disease, of its supervening upon a cold, or a succession of colds. But the weight of medical opinion has in our day tended strongly towards the rejection of all such notions. The only statistical facts which are in favour of the popular view are those given by Dr Theodore Williams in vol. liv of the 'Med.-Chir. Transactions.' Out of 1000 cases of phthisis he found that no fewer than "149 had originated in, or been closely preceded by, pleurisy and pleuro-pneumonia, and 118 by bronchitis;" but it does not appear probable that this tabulation can have generally rested on other foundation than the unsupported assertions of the patients themselves, who doubtless consulted him or his father, Dr C. J. B. Williams, at variable periods after the commencement of their illness, and often when a considerable time had elapsed.

The considerations which led pathologists to reject the idea that phthisis could arise out of a common catarrh were probably in the main identical with those which induced them to regard as necessarily non-tubercular the cases due to dust-inhalation. And such theoretical opinions were greatly strengthened by the practical observation that many persons, even of delicate aspect, suffer for years from an extreme liability to bronchial attacks without ever becoming consumptive. Indeed, Rokitansky actually declared that pulmonary emphysema and dilatation of the bronchial tubes, if carried far enough to cause venosity of the blood and cyanosis, afford exemption from the liability to pulmonary tuberculosis. But, apart from the crucial instance of congenital disease of the heart, there are striking exceptions to such a rule. In 1864, for example, a girl of seventeen was admitted into Guy's Hospital with extreme dyspnœa and dropsy, and with clubbing of the fingers and toes. The bronchial tubes were found widened out into great sinuous passages, so that the cut surfaces of the lungs showed hollow spaces as extensive as the remains of the pulmonary tissue. Yet there were scattered yellow tubercles, especially in the left lung, spreading from the apex downwards. In 1874 a woman, aged thirty, died, who

had long been more or less subject to cough, which for nine months before had become continuous. There was extreme emphysema of the bases and anterior parts of the lungs, and the tubes contained a large quantity of pus; but both lungs also showed scattered grey tubercles and patches of translucent grey consolidation, with points of caseation breaking down here and there into cavities. There would be no difficulty in citing a good many other similar cases from our *post-mortem* records were it needful.

Another fact which indicates that catarrh is apt to lead to phthisis is the frequency with which, in children, pulmonary tuberculosis follows whooping-cough or measles. There is, of course, the difficulty that catarrh, so far as is known, spreads from the larger tubes into the smaller in all directions, or perhaps towards the bases of the lungs in preference. We certainly have no proof whatever of the occurrence of what is so often spoken of—a catarrh localised at one or both of the apices; and, if so, the adoption of such an expression is meaningless, except for the purpose of covering a doubt on the part of the physician as to whether there is anything the matter with the lung, or for that of soothing the mind of the patient, and inducing him to submit to treatment without being dismayed. But on the view advocated in the first volume with regard to tuberculosis —that it is a modification of the inflammatory process, which is especially apt to arise at the apices of the lungs from a morbid tendency inherent in those parts—there is nothing improbable in the supposition that irritation of the whole of the pulmonary tissue by cold may set up phthisis there, while failing altogether to do any damage elsewhere. More than one instance has come before the writer in which consumption has appeared to be distinctly traceable to residence in a damp, newly-built house, the patient having been quite well up to the time of the change of abode.

Nevertheless, caution is required in accepting the statements of patients themselves with regard to the origin of their illness. In 1869 a young man died of phthisis in Guy's Hospital, who attributed the disease to his having slept with his window open one night five weeks before. He admitted, however, that nine months previously he had been ill for a week with a cough. At the autopsy, besides a very acute phthisis of pneumonic character, there were found at the left apex old clustered grey and black tubercles and cavities. This, therefore, could not be cited as a case caused by exposure to cold.

γ. *Soil.*—It is most probably by the increased liability to attacks of slight bronchial and pulmonary catarrh which must necessarily follow residence in damp situations, that we can account for certain remarkable observations which have recently been made with regard to the influence of *soil* upon the frequency of phthisis. In 1862 Dr Bowditch, of Boston, took occasion, in addressing the Massachusetts Medical Society, to bring forward a mass of evidence which led him to believe that, in that State, consumption, instead of being equally diffused through all parts of it, prevails especially in such places as are situated upon a damp soil, and seldom occurs when the soil is dry. This evidence consisted chiefly of an analysis of the replies of medical men living in 183 townships to inquiries as to the frequency of the disease in their practice, and as to the moisture or dryness of the localities. It also includes some striking instances in which phthisis had carried off in succession a number of persons living in certain houses surrounded

by wet meadows, or placed by the side of a millpond, or shut in by luxuriant trees.

Far more conclusive, because resting upon an accurate statistical basis, is a body of facts which were collected by Dr Buchanan during the years 1865 and 1867 in England, and published in Mr Simon's ninth and tenth reports to the Privy Council. The inquiry began in a tour of inspection made for the purpose of ascertaining the results of sanitary works that had been carried out in twenty-five towns, containing an aggregate population of 606,186 persons. It was found that in several places there had been a great diminution in the general death-rate, and that the prevalence of enteric fever had become much less, especially where a good water supply had been substituted for a bad one, and where drainage-works had displaced cesspools or middens. But in other towns it was by a decrease in the number of cases of phthisis that the good effects of sanitary improvements appeared to be manifested; and the particular change which coincided with this result was found to have been drying the ground by drainage of the subsoil. The following table shows the amount of change in the phthisis death-rate in twenty-four of the towns visited by Dr Buchanan:

Town.	Previous death-rate per 10,000 from phthisis.	Degree of change in death-rate from phthisis.		Influence of sewage-works on subsoil.
		In total population	In females between 15 and 55.	
Salisbury	44⅓	−49 p.c.	?	Much drying.
Ely	32	−47 p.c.	?	Much drying.
Rugby	28½	−43 p.c.	−48 p.c.	Some drying.
Banbury	26⅔	−41 p.c.	−36 p.c.	Much drying.
Worthing	30½	−36 p.c.	−41 p.c.	Some drying.
Macclesfield	51½	−31 p.c.	−22 p.c.	Much drying.
Leicester	43⅓	−32 p.c.	−16 p.c.	Drying.
Newport	37	−32 p.c.	−13 p.c.	Local drying.
Cheltenham	28⅔	−26 p.c.	−25 p.c.	Some drying.
Bristol	33½	−22 p.c.	−18 p.c.	Some drying.
Dover	26½	−20 p.c.	−18 p.c.	Local drying.
Warwick	40	−19 p.c.	−10 p.c.	Some drying.
Croydon	*	−17 p.c.	?	Much drying.
Cardiff	34⅔	−17 p.c.	?	Much drying.
Merthyr	38½	−11 p.c.	−12 p.c.	Some recent drying.
Stratford	26⅔	− 1 p.c.	− 4 p.c.	Some local drying.
Penzance	30⅔	− 5 p.c.	0	No change.
Brynmawr	28⅓	+ 6 p.c.	− 8 p.c.	No notable change.
Morpeth	30½	− 8 p.c.	+12 p.c.	No change.
Chelmsford	32½	0	+11 p.c.	Slight drying.
Penrith	39½	− 5 p.c.	+27 p.c.	No change.
Ashby	25½	+19 p.c.	−10 p.c.	Some drying.
Carlisle	32	+10 p.c.	+11 p.c.	Drying (with local defects).
Alnwick	28⅓	+20 p.c.	+36 p.c.	No drying.

It is perhaps worth while to give some details as to one or two of these towns, since the full significance of the change that has been effected in them by drainage-works can hardly be appreciated otherwise.

In 1851 Mr Rammell had reported of Salisbury as follows: "Numerous

* Phthisis and other lung diseases together were previously 59½ p.c. Reduction of this rate is what is above given for Croydon. (Ninth Report, 1866, p. 48.)

streams of water, supplied by the Avon, run through most of the streets. . . The soil is a porous gravel, containing everywhere a great deal of water, which rises to within a short distance of the surface. There have been several instances of the cathedral being flooded by the water of the subsoil. The foundations of the houses are almost without exception damp." . . "The drainage system of the principal part of the town is formed by the open channels which run along the footpath at the side of the street, and the house-drains open into them from both sides. The fall of these drains is necessarily very small, they are frequently in their whole length below the level of the water in the open channel, and consequently, so far from being able to eject their contents into the common channel, they are in such cases generally filled with water from the channel." The water supply is from wells "dug about eight or ten feet deep, the water rising to within three or four feet of the surface." In 1853 sanitary works were commenced in Salisbury, and they were completed in 1855. The drainage consists of brick mains, and of glazed earthenware pipes opening into them. The mains have floors of hollow stoneware pieces laid without cement, and at the sides of the pipes are placed drain-tiles. These arrangements are for the purpose of carrying off the subsoil water, of which there is constantly a rapid flow through the mains. In 1865 Dr Buchanan writes as follows: "The subsoil is now dry, and cellars of considerable depth can now be made in different parts of the town which do not become flooded at any time. On an average the subsoil water has been lowered four or five feet all over the city. The cathedral has never been flooded since the drainage-works. As is shown in the table, the annual death-rate from phthisis fell in Salisbury from $44\frac{1}{3}$ per 10,000 in 1844–52, to $22\frac{2}{3}$ per 10,000 in 1857–64."

Of another town, Banbury, Mr Rammell had reported in 1850 in the following terms:—"The drains are not all at a sufficient depth to drain the cellars of the houses. In the principal streets of the town water is raised from the cellars into the drain by buckets, and creates a nuisance." Sanitary operations were begun there in 1854. The sewerage was done on a uniform system. Most of the sewers have a good fall, and are laid seven to ten feet below the surface. "At present," says Dr Buchanan, writing in 1865, "the sewers and drains all act efficiently. . . Many of the wells of the town have been dried by the sewers. . . The sewage discharged into the river after 10 p.m. is little more than water from the springs." As appears from the table, the phthisis death-rate for 10,000 has declined from $26\frac{2}{3}$ in 1845–53 to $15\frac{3}{5}$ in 1857–64.

It must of course be understood that drying of the subsoil is not the only improvement that has been made in these towns. Excreta have at the same time been carried off from the houses, a good water supply has often been provided, and overcrowding has been diminished. Now, as regards removal of filth it does not seem that this has acted in reducing the death-rate from phthisis. Dr Buchanan placed the several towns in another list, according to amount of decrease in the mortality from enteric fever, and the order in which they stand in the phthisis-list is by no means the same as that in the fever-list, which appears to be more affected by removal of filth than by anything else. Many of the towns lower down the phthisis-list, such as Alnwick and Brynmawr, had made very good arrangements for carrying away excreta. And, on the other hand, Worthing and Rugby, both of which stand well in the phthisis-list, are very low in the fever-list. The cases in which "sanitary works" have failed to reduce the death-rate

from consumption are chiefly those in which the soil previously contained little water (as at Penzance and Brynmawr), and those in which the deep drainage was effected by impervious pipes laid down in compact channels (as at Penrith and Alnwick) so that no extensive soil-drainage could occur either through or beside them.

The importance of these observations appeared to be so great that, in 1867, Dr Buchanan was directed by the Privy Council to make a special investigation in the three south-eastern counties, Surrey, Kent, and Sussex, for the purpose of determining whether any relation could be traced between the prevalence of consumption and the state of the soil as regards moisture. These three counties were chosen because in them only had the Geological Survey then minutely mapped out the surface; but no part of England could have been better adapted to the purpose, on account of the great varieties of soil found there and the comparative absence of other differences between the several districts.

The first point was to ascertain the extent to which phthisis prevailed in different parts of the three counties. The basis for this part of the inquiry was afforded by the Registrar-General's Returns. Of course they cannot pretend to exact pathological accuracy. But seeing that in each registration district the certificates are furnished by several medical men, it is not likely that any serious error can result from their being used for the purpose of comparing the death-rate from so common a disease as consumption in one district with that in another. Moreover, if cases of phthisis are wrongly returned under any other head, it must be generally under that of bronchitis or of some other lung disease. Dr Buchanan, therefore, took pains to consider the mortality from lung diseases in general, as well as that from phthisis, before he drew his conclusions.

Corrections had to be made in the returns for certain districts on account of the presence of camps or dockyards, causing the population to have a large excess of males in the prime of life; in others the returns were vitiated by their containing hospitals or asylums; in others, again, by their being resorts for invalid visitors; eight districts were set aside as being subject to one or other disturbing influence, which rendered their true consumptive death-rate uncertain. There were left fifty other districts which it was believed could be fairly compared with one another.

In instituting this comparison, Dr Buchanan first classified the several districts as having mainly soils *permeable* by moisture, or soils of such a character that water is unable to escape from them, so that they might be called *retentive*. He then massed together the fifty districts into five groups of ten each, according to the greater or less prevalence of phthisis in them, and in this way he obtained the following table.

Groups of districts.	Proportion of population (per 1000) residing on	
	Permeable soils.	Retentive soils.
A. With least phthisis	909	91
B. With next least phthisis . . .	877	123
C. Middle as to phthisis. . . .	795	205
D. With more phthisis	792	208
E. With most phthisis	642	358

He pointed out that this tabulation, apart from arithmetical objections,

must be corrected by certain geological considerations. Where the soil is pervious, its being moist or dry must depend entirely upon whether the water which reaches and sinks into it can escape through or from beneath it. It is no advantage for a place to be situated on gravel if the subsoil water cannot get away. Roughly, one may say that such a district will be *dry* in proportion as it *lies high* in relation to the places round it, *damp* in proportion as it *lies low*. On the other hand, among impervious soils, the question of dryness or moisture is almost entirely one of the *inclination of the surface*. Even among clays there is a great difference as regards dampness, according to the flat or sloping character of the ground.

A more exact comparison between retentive and pervious soils in regard to the prevalence of phthisis is afforded by a limited area, the Wealden, which in part is formed by the Weald clay, in part by the Hastings beds of alternate sands and clays. There are, indeed no districts wholly of sand to contrast with others wholly of clay; but there are great differences in the proportion of the two soils in different districts. How closely these differences correspond with differences in the consumptive death-rate appears from the following table.

District (in order of phthisis death-rate).	Percentage of population resident on						Total on	
	Higher beds, mostly lower greensand.		Weald clays.		Hastings beds.		Sands + half gravels over Weald clay.	Clays + half gravels over Weald clay.
	Sands.	Clays.	With gravel.	Without gravel.	Sands.	Clays.		
Hastings	...	...	...	...	95	5	95	5
Cranbrook	...	...	1	6	84	9	84	16
East Grinstead	...	...	...	12	82	6	82	18
Tunbridge	...	1	24	7	64	4	76	24
Hambledon	49	...	20	31	...	...	59	41
Battle	...	...	...	...	80	20	80	20
Rye	...	4	...	...	79	17	79	21
Maidstone	43	1	45	11	...	...	66	24
Cuckfield	21	1	...	25	48	5	69	31
Uckfield	...	...	...	1	82	17	82	18
Hailsham	...	...	...	34	61	4	61	38
Ticehurst	...	...	...	...	67	33	67	33
Tenterden	...	...	...	29	42	29	42	58
Horsham	...	...	...	56	44	...	44	56
Petworth	30	...	...	70	...	...	30	70

The districts are arranged in order of the death-rate from phthisis, those being placed highest in which it is least. Where there are gravels over the Weald clay the figure is divided between the last two columns, it being presumed that they occupy an intermediate position.

Still more striking perhaps are certain comparisons which can be made between particular sets of districts which differ in the manner suggested in the last paragraph but one; if *pervious*, in being *high-lying* or *low-lying* respectively; if impervious, in being *sloping* or *flat*.

1. As between *high-lying* and *low-lying pervious* soils, a contrast is afforded by districts formed by the chalk. No soil is drier than chalk when it has a good elevation; at its higher parts there are no streams, water cannot be reached by ordinary wells, and the people can only obtain a water supply

from less elevated ground. In many districts, however, the bulk of the population who live on chalk occupy valleys with the water-line in the chalk not very far below their houses; and in the south of Sussex a still greater degree of wetness is reached, for a large part of this area reckoned as chalk is a flat plain on the sea level, covered by gravel, with the dip of the chalk here and there inland. Accordingly, we find the phthisis death-rate for North Aylesford and Dover (both of which lie high) to be 289 and 296 respectively, while those for Worthing, Lewes, and Westbourne (all of which lie low) are 419, 426, and 498. In general, Dr Buchanan adds, the connection between a low death-rate from phthisis and elevation of the chalk area is unmistakeable.

So, again, with regard to the population living mainly on the Lower Greensand, there is a great contrast between a southern tract of this formation which lies low and the hills which are made up of it elsewhere. And a corresponding difference exists between the phthisis death-rates of Thakeham, Midhurst, and West Ashford (454, 455, and 421 respectively) and those of Reigate and Godstone (337 and 282).

2. As between *sloping* and *flat* impervious soils, a capital contrast is presented by two widely distributed tracts of clay, the London clay and the Weald clay. The former in its main extent throughout the three counties is disposed in long slopes or hills, the latter constitutes sometimes gently undulating, but more often flat and level ground. The former is covered over large areas by gravel reaching to many feet in thickness; the latter has only very level gravels, occupying its undulations, and these are seldom thick. The former generally has the direction of drainage from other beds away from it; the latter is always bounded immediately to the north and to the south by higher ground so that other beds drain more or less into it. All these considerations show that the London clay is commonly much less wet than the Weald clay. The difference between the two formations in respect of their phthisis death-rate is, Dr Buchanan says, unmistakeable. All districts that have even a third of their population on Weald clay have a high mortality from consumption, whereas there may be in a district a notable proportion of uncovered London clay without any like result.

It is right to mention that Dr Kelly, the Medical Officer of Health for East Sussex, has expressed doubts of there being any intimate relation between dampness of soil and phthisis. He finds that in the years 1861–70 the order in which the several districts have to be placed in regard to their death-rates from phthisis is different from that given by Dr Buchanan for 1851–60. He points out that most of the impervious beds are to the north of the South Downs, and that consumption seems most common in places which are bleak and exposed as well as damp. He insists on the fact that in West Sussex (as indeed throughout England and Wales) there has of late years been a great decrease in the mortality from consumption, although there has been no change in the drainage of Sussex. Dr Kelly is inclined to attribute it mainly to the progress which has taken place in the social state of the rural population.

It is much to be desired that by a Royal Commission, or any better means, an extensive and searching official investigation over long periods of time should be undertaken, which might at least settle the facts upon which must rest the significance of damp soil and the conditions of its operation in this most destructive malady.

δ. *Direct injury.*—Whether this can be enumerated among the causes of phthisis is doubtful. In 1880 there died at Guy's Hospital a man, aged thirty-eight, who had been a patient of Dr Moxon's. He had been a cab-driver, but was said to have been very moderate in his habits; there was no history of consumption in his family. Four weeks before his admission his cab had been upset and turned over upon him, the step coming upon his chest. Afterwards he had coughed and spat a little blood. Three days later he had brought up half a pint of blood. His illness was regarded as the direct result of the accident, but the physical signs were exactly like those of phthisis affecting the left lung, and spreading through it from above downwards. When he had been in the hospital two months and a half he died. The left lung was found to be destroyed by a phthisical affection of "pneumonic" character. The upper lobe and the upper part of the lower lobe were hollowed out into a number of cavities; the rest of the lower lobe was consolidated by a pinkish-grey infiltration, scattered in which were many yellow tubercles and caseating patches with sinuous edges. But the right lung was affected with a more chronic form of the disease; in the upper lobe was scattered much pigmented grey tubercle; there were also some caseous tubercles, and on each side of the lobar septum there were one or two small vomicæ. The tubercular nature of the disease was confirmed by the fact that the small intestine contained ulcers which, although circular, had thickened edges and showed yellow submucous tubercles in their floors. In the larynx, too, there was a deep ulcer over the left arytænoid cartilage. Evidently the relation of the accident to the phthisis can only have been that it started with fresh activity a morbid process in the lungs which was already in existence before; and one may fairly question whether this was not rather the result of the general shock than of the local injuries which the man received.

In four other cases phthisis followed an accident; in one there was fracture of ribs, in another fracture of the collar-bone, in the other two injuries of an undetermined kind from a fall into the hold of a ship and from a railway accident respectively. But in none of these cases is there any proof that the lungs were previously healthy; nor, indeed, is it certain that there was any closer connection between the accidents and the pulmonary disease than mere coincidence.

Analogy with some other tubercular affections, as those of the kidney and the joints, would lead one to admit that an injury to the chest might set up phthisis. But analogy is an uncertain guide, and considering how common are injuries to the chest and how common a disease is phthisis, instances of a connection between them ought to be more frequent if it existed.

Opposing pathological conditions.—To complete our survey of the ætiological relations of phthisis, it is necessary that brief allusion should be made to certain conditions which have been, or still are, supposed to be antagonistic to its development.

One of these is habitual exposure to *malaria.* This opinion is now generally discredited, but Walshe brings considerable evidence in its favour.

The simultaneous occurrence of *carcinoma* and of tuberculosis is rare, but probably only due to the facts that each of these diseases is usually fatal, and that one of them chiefly attacks persons who are old, the other those who are comparatively young. Nine or ten cases occurred at Guy's Hospital between 1855 and 1874 in which more or less active phthisis has

been present in patients who have also had cancer of the stomach, or womb, or œsophagus, or some other organ. One was a woman, aged twenty-two, another a man, aged twenty-four, a third a man, aged thirty, the rest were older, one having reached the age of sixty-seven.

Antagonism between phthisis and *gout* has also been generally accepted, and probably not without reason. The period between twenty-five and forty-five in men is very liable to both diseases, yet cases of their concurrence in the same patient are rare. It would be interesting to know how often deposits of lithate of soda in the great toe joint are found in bodies which also show evidence of obsolete or recent disease of the apices of the lungs. That gout and active phthisis may coexist is proved by three cases observed during life and recorded in the 'Guy's Hospital Reports' for 1873 (vol. xix, p. 338), while in a fourth patient with gouty deposits in the joints signs of old phthisis and some recent clusters of tubercles were discovered in both lungs.

On the other hand, it appears to be indisputable that at least one kind of valvular *disease of the heart* is an almost complete bar to the development of phthisis. Mitral stenosis is exceedingly common in young persons, and it often fails for several years to affect the general health to any marked extent. That this lesion should be scarcely ever found in those who die of consumption is therefore a very remarkable fact. Traube could not remember to have met with an instance. In our records of post-mortem examinations at Guy's Hospital, from 1854 onwards, only four examples occur. One was in a man, aged forty-two, whose mitral orifice was so far narrowed, as the result of rheumatism, that it would only admit two fingers. On the other hand, Traube speaks of having seen several cases in which persons with regurgitant disease of the aortic valves became affected with phthisis.

Whatever may be the explanation of the rarity of consumption in those who have mitral stenosis, it can hardly depend upon the venosity of their blood, as Rokitansky thought. For it is now well known that those who have congenital narrowing of the pulmonary orifice are exceedingly apt to die of tubercular disease of the lungs. Traube says that he has seen two examples of this; and two at least have occurred at Guy's Hospital. Traube's view is that mitral stenosis, by causing liquor sanguinis to exude into the pulmonary tissue, prevents the occurrence of caseation; and Dr Hamilton, in the 'Practitioner' for 1880, throws out a similar suggestion. But such an interpretation of the facts involves the assumption that phthisis begins as a modification of catarrhal pneumonia.

Age and sex.—Phthisis may occur at all periods of life. The idea that it is a disease chiefly confined to young adults is inaccurate. At Guy's Hospital there has been little if any diminution in the number of fatal cases for each quinquennial period up to the age of forty-five. Several cases have occurred in persons between sixty and seventy; and two at the age of seventy-two. Statistics of Dr C. J. B. Williams's patients appear to show that, other things being equal, the *duration* of phthisis increases with age, and it is a matter of general observation that the disease in children runs an acute course, while senile phthisis is usually chronic. This would bring the average age at which it begins earlier than the following statement of ages at death would indicate.

The period at which appeared the first decided symptoms of what afterwards developed into phthisis was found by Dr Williams to be

distributed as follows among 1000 private patients:—195 cases at an age below twenty (of these only 13 under ten), 667 between twenty and forty (and of these 418 below thirty), 94 between forty and fifty, 30 between fifty and sixty, and 14 cases began at an age above sixty. The average period was later in men than in women.

More men than women appear to die of consumption in the hospitals of London, although the Registrar-General puts the rates of males to females in the population generally as 3·77 to 4·13. Of Dr Williams's 1000 cases 625 were males and 375 females. Among the out-patients at the Brompton Hospital Dr Pollock found 60·7 per cent. male and 39·3 per cent female. It must not be assumed that sex or age constitute predisposing causes of the disease, in a strict sense of that term. The question may be of the more frequent operation, at different periods of life and in one sex rather than the other, of the various causes that have already been enumerated, particularly exposure and catarrh.

In women consumption shows itself, as a rule, earlier than in men, and more frequently runs a rapid course.

Geographical distribution.—Phthisis is, on the whole, more prevalent in temperate regions than in those which are very hot or very cold. It is endemic throughout Europe, but is most abundant in large cities, particularly in Paris and Vienna. It is probably as common in the United States, in China, and in Australia; but is less so in the East Indies and within the tropics generally, although imported cases from Europe are said to be very unfavourably influenced by a hot climate. It is extremely rare in Iceland, in the highlands of the Andes, in New Zealand and in some oceanic islands, in Syria, and in Egypt. It is rare in swampy and malarious regions, as was first pointed out in 1812 by Dr Wells (the author of the essay on 'Dew'), and since confirmed by Boudin and many other observers. No race of men is known to be exempt from consumption.*

Prophylaxis.—It would of course be far simpler, if we could attribute phthisis to one single cause, instead of looking upon it as the result of the joint operation of several. But if, as seems almost certain, the latter view is the true one, it opens to the physician a wide field of usefulness, in preventing the development of this terrible disease. Even the hereditary transmission of consumption is not altogether beyond his scope as regards prophylaxis. He can often do much to prevent the intermarriage of cousins belonging to a phthisical family, and he can exert his influence to induce the members of such a family if they marry to select partners from a healthy stock. To abstain from marriage altogether is seldom, if ever, incumbent on those who are not themselves consumptive, provided that they are able to bring up their offspring under favourable conditions.

If a mother is known to be already phthisical, or even disposed thereto, it is a grave question whether she should be allowed to suckle her children.

Care should be taken that the residence of those who have tubercular tendencies should be on a dry soil. The rooms in which they live or sleep or work should be airy, well ventilated, and so situated as to be exposed to

* Phthisis is well known among the lower animals, particularly the quadrumana and the ruminants (see Mr J. B. Sutton's records of "Diseases of Monkeys," 'Path. Trans.,' and Dr Creighton's monograph on "Bovine Tuberculosis").

sunlight. Their food should be nutritious and fattening. They should have plenty of exercise in the open air, and they should be accustomed to exposure to the weather, but within reasonable limits, and only so that they do not become chilled. Cold bathing is advisable, provided that there is always a good reaction after the bath.

Special care should be taken during convalescence from whooping-cough and measles; and the recurrence of attacks of bronchial catarrh should be sedulously avoided. Outdoor sports are probably beneficial, so long as they are not carried to excess. Study, whether in preparing for examinations or in the pursuit of professional eminence, should be kept within due bounds. Indeed, as adult life is approached, the necessity for moderation in all things should be impressed on everyone who would avoid the risk of phthisis. Temptations to intemperance and to dissipation must be strenuously resisted. If an occupation is to be chosen, it should be such as is favourable to health. The son of a phthisical miner, or potter, or weaver, should, even more than others, avoid such dangerous kinds of work. If a youth is to be a clerk, he should, if possible, be put where the hours are comparatively short, and where the duties need not be performed under severe pressure.

Treatment.—This naturally falls under three heads:

(1) The early symptoms of phthisis have to be checked, and the disease brought into a state of quiescence.

(2) A strenuous effort must, if possible, be made to arrest its further progress or to prevent a relapse.

(3) If this effort fails, one must aim at retarding the fatal issue to the farthest attainable limit.

(1) We have seen that many cases begin with alarming hæmoptysis. The patient must then be kept strictly in the recumbent position for two or three weeks at least. He should not be allowed to talk. His diet should be limited almost entirely to milk. He should have ice to suck, and everything that is given to him should be cold. Of styptics it is difficult to say which is the best; ergot, gallic acid, acetate of lead, opium, have each their advocates, and it sometimes seems necessary to try one after another. A large ice-bag may also be placed over the chest. When the hæmorrhage has ceased, the patient is very cautiously allowed to get up and to move about, and the amount of food is gradually increased, while the pulse and temperature are being carefully watched from day to day.

But whenever phthisis sets in with well-marked early symptoms, even though hæmoptysis may be absent, similar regimen should be adopted. The patient should be put to bed and kept absolutely at rest. He should be limited for a time to a very light diet, consisting mainly of milk, without wine or other stimulant. Whether hæmorrhage has occurred or not, a very good prescription, if there is much pyrexia, is Niemeyer's pill of quinine (gr. j), digitalis (gr. $\frac{1}{2}$), and opium (gr. $\frac{1}{4}$), to be taken every four or six hours. Tincture of iodine should be applied to the affected part of the chest, or even a blister, or the croton-oil liniment. It is almost an advantage to the patient, if he is to have phthisis at all, for it to set in with hæmoptysis; because then the real gravity of his condition is appreciated, and there is no hesitation in carrying out the measures which are necessary. "Catarrhal phthisis," in which one apex becomes quickly consolidated, has been held distinct from the "tubercular" form of the disease, because it yields so readily to treatment; and probably cases in which rapid consolidation occurs may run a more favour-

able course than others; but this may be because they are taken in hand more carefully and more energetically.

When the acute symptoms have passed off, the patient may go to the seaside for a few weeks, or to some dry and healthy place inland, such as Tunbridge Wells or Malvern, or Ben Rhydding if the season be suitable. On the Continent a favourite plan is to send him to Lippspringe, near Paderborn, to drink the water of the lime-spring there, or to Soden and other health resorts in the Taunus.

(2) It is impossible to insist too strongly on the importance of not letting slip the opportunity, which occurs in phthisis only at an early stage, of arresting its further progress, and of preventing a relapse. The measures by which this—the virtual *cure* of the disease—can alone be effected, all involve, as a rule, a prolonged change of climate. They are as follows:

a. A long sea-voyage, generally either to the Cape of Good Hope (or the highlands of Natal), or else to Australia by the Cape of Good Hope. About twenty-three days are taken in going to the Cape, twenty-eight days to Natal; the voyage to Australia varies greatly in length, according as it is made in a steamship or in a sailing-ship, being in the one case about forty days, in the other about three months. With regard to a host of details, a knowledge of which is absolutely necessary to the invalid for whom a long voyage is recommended, information must be sought either in a little book by Dr Wilson, 'The Ocean as a Health Resort,' or in a series of papers by Dr Faber, published in the 'Practitioner' during 1876–77. On the whole, sailing ships appear to be preferable to steamers, one among other advantages being the greater length of the voyage, which renders the changes of climate less sudden and trying to the health; but the greater cleanliness and quiet, together with the easier motion, are also strong recommendations. There is of course considerable heat in crossing the equator, and, on the other hand, during the latter half of the journey to Australia the weather is often cold and stormy. As large a part of the day as possible should be passed in the open air, and exercise on deck should be systematically taken. It seems to be important that the excessive appetite which generally arises should not be too freely indulged. A stay in Australia of from six weeks to three months should be made, at the end of which time the voyage home should, if possible, be made by the Cape, this taking generally three and a half or even four months. Dr Faber insists that no patient should be sent to the Antipodes who is not quite free from pyrexia in the evening; the climate of the tropics is very apt to cause a great increase of fever, and to render it continuous, from having been hectic in type. It is also apt to bring on hæmorrhage, so that a marked disposition to hæmoptysis, or the presence of rigid degenerated systemic arteries in an old person, constitutes another strong objection to a long sea voyage. The extent to which an individual is likely to suffer from sea-sickness cannot be foretold, unless it has already been proved by former experience; the result of a short trip across the Channel decides nothing.

b. Residence in an elevated mountain region for a length of time, or at least in a dry bracing climate. It is impossible, in the limits of this work, to enter into full details with regard to climatic treatment of phthisis. The reader must consult other works, especially the fourth edition of Dr Walshe's 'Diseases of the Lungs,' Dr Weber's translation of Braun's 'Baths and Waters,' and a little book by Mr R. H. Otter, entitled 'Winters Abroad;' also 'The Influence of Climate in the Prevention and Treatment of Pulmonary

Consumption,' by Dr Theodore Williams; and Dr Weber's interesting Croonian Lectures on "The Hygienic and Climatic Treatment of Consumption," 'Lancet,' March, 1885.

Within the last few years it has become a common practice to send consumptive patients during the winter to Davos, a village situated in the Grisons, at an elevation of about 5200 feet above the sea. Other places, perhaps, might be found which would yield equally good results; and St Moritz and Samaden, in the Engadine, had in fact been tried before Davos, at least by English invalids. The Maloja Pass has more recently come into favour. The great peculiarity of the weather in these elevated stations is the stillness and the dryness of the air. In the shade the cold is extreme, but as the sun is very powerful, and as the sky is generally perfectly clear, patients are able to take exercise nearly every day—walking, skating, sledging, or "toboggining." When sitting in the verandahs of the hotels the sunshine is hot. At night the double windows in the bedrooms are left slightly open; yet so motionless is the air that the temperature within scarcely falls below 50° Fahr., even when it is from 2° to 16° Fahr. outside. Many persons who are very liable to take cold elsewhere are free from the tendency at Davos. The proper time for a patient to arrive there is about the first or second week in October, or even earlier. It is generally supposed to be undesirable for him to remain after the beginning of April, when the snow melts. Unfortunately, there is no little difficulty in saying where he should then go. He should on no account return to England before the first week in June; and during the interval the choice seems to lie between Baden-Baden, Wiesbaden, Montreux, on the Lake of Geneva, and Monte Generoso above Lago Lugano. The fact that hæmoptysis has been one of the symptoms does not appear to be an objection to sending a consumptive patient to Davos; but the actual presence of pyrexia is an objection, and still more so irritability of the larynx or trachea.

In America there are mountain climates in which phthisical patients derive great benefit without being exposed to extreme cold. This was pointed out long ago by Dr Archibald Smith, who practised for many years in Peru; but indeed, it seems to have been familiar knowledge to the Peruvians themselves, who regard the valleys of the Andes, from 8000 to 10,000 feet above the sea-level, as almost omnipotent in the prevention and cure of consumption. As a general rule, it may be said that the nearer the equator the greater the elevation which is necessary to render a mountain region salutary in such cases. The chief resorts in the Cordilleras appear to be Huanuco and Jauja. Dr Walshe recommends, as more accessible, the plateau of Santa Fé de Bogotà in New Granada. A great peculiarity of this place is the equality of its climate at different seasons; the mean temperature of each quarter of the year is within a degree or two of 86° Fahr. Other mountain regions to which phthisical patients may be sent are San Paulo, near Santos, the tablelands of Guatemala and of Mexico, and Manitoba or Colorado.* One can hardly doubt that in the Himalayas also there must be valuable resorts: Dr Weber is inclined to think that the present military sanitaria there may not be at a sufficient elevation for the climate.

c. A prolonged stay in the Southern Hemisphere, during what would be the winter of Europe, but is of course summer there. Certain parts of Australia are very serviceable to phthisical patients. It must not, however,

* See a paper on the "Climatic Treatment of Phthisis in Colorado," by Dr Charteris, of Glasgow ('Lancet,' Nov. 26th, 1887).

be imagined that a residence in the large towns is advisable. Melbourne, in particular, is apt to be intensely hot and dusty, with very rapid changes of temperature and cold winds. The best health resorts appear to be certain places in the interior of New South Wales, especially Bathurst, Goulburn, Boural (3000 feet above the sea), and Currajong, but above all, the Darling Downs, in the South of Queensland, where the weather is cool, dry, and bracing. During a large part of the year, the Riverina also has a magnificent climate, but in the hot season it is advisable to go elsewhere. Both Tasmania and New Zealand are suitable for consumptive cases; Hobart Town and Wellington or Auckland especially are well spoken of.

Certain parts of South Africa have climates which appear to be very favourable to phthisical patients, but it is essential that no long stay should be made at the sea-coast; Mr Otter says not within 100 miles of it, nor at a less elevation than 1500 feet. The easiest way of reaching the interior is to land at Port Elizabeth, and to go on by Grahamstown to Cradock or to Bloemfontein, the capital of the Orange Free State. This, however, cannot be done safely by an invalid, except at great expense, on account of the badness of the roads, and the accommodation becomes rougher and rougher the greater the distance from the sea. Bloemfontein has an exceedingly dry climate; the daily range of temperature is great, but this is said not to act prejudicially. The best plan for those who can afford it is said to be to buy an ox-waggon and to "trek" through the Free State and the Transvaal for three or four months, sleeping generally in the waggon.

The working of change of climate, as a curative agent in phthisis, is still uncertain. In many instances the beneficial influence on patients who come from a distance finds its parallel in the fact that natives of the same district are very seldom, if ever, attacked. This appears to be the case, for example, in the high Alps, as well as in the valleys of the Andes, and until recently it was so in Australia, although the disease is now rife among the inhabitants of Melbourne and of other large towns there. But Dr Walshe and others have rightly insisted that there is no necessary connection between the two things, and there is no difficulty in finding countries, such as Iceland, to which one would not think of sending a consumptive patient, notwithstanding that the natives escape the disease. To the author it appears evident that the "aseptic" character of the air of a place cannot be the direct reason why phthisis should cease to advance in those who inhale it. Observations showing that meat remains fresh there longer than elsewhere are altogether inapplicable; the only case that could be in point would be if putrefaction once started should fail to become complete. It is very probable that ozone has a therapeutic action in disease, but we cannot at present say what the nature of such action may be. On the whole, it seems likely that the good effects of change of climate depend partly upon its improving the general health and increasing the resistance of the organism to the further progress of the disease, partly upon its protecting the patient from fresh attacks of bronchial catarrh. The number of bacilli found in the sputum is not diminished in Davos, according to Dr Theodore Williams.

(3) When from any cause the complete arrest of phthisis is no longer attainable, a great deal may still be done to prolong the patient's life and to give him relief from suffering. The climates which Dr Walshe terms *sedative* seem to have their chief use under such circumstances; for example, Madeira, Pau, San Remo, and some other places on the Riviera, Torquay, and Penzance.

The climates which Dr Walshe classifies as *stimulant* include St Leonards and Hastings, various health resorts of the Riviera, Algiers and even Egypt. Of the Upper Nile he speaks in terms of the highest praise; and probably a voyage up the Nile is the best thing for patients who dislike cold, and who habitually feel stronger and better the hotter they are. Whether special benefit can be anticipated from the air being impregnated with resinous emanations from pine forests—as at Arcachon and Bournemouth—or from places where great fires are burnt, is easier to assume than to explain or to prove.

It must of course be remembered that phthisis sometimes becomes quiescent without any change of climate whatever. On the other hand, although it is a heavy responsibility to advise, or allow, a patient with advanced lung disease to take a long sea-voyage, or to spend a winter in Australia, in South Africa, among the Andes, or even at Davos, there is no question that, if he chooses to run the risks inseparable from such undertakings, he has at least a chance of unexpected benefit.

Physic.—The treatment of phthisis by drugs, though decidedly secondary in importance to hygienic measures, must not be neglected. Foremost in value is *cod-liver oil*, in doses of from a drachm to half an ounce two or three times a day. The remedy was introduced for cases of chronic "rheumatism," but was first advocated as peculiarly useful in consumption by the late Dr Hughes Bennett. Now that it is taken by almost all consumptive patients, some of whom do not even wait for it to be formally prescribed, one is apt to underrate its real importance as a means of preventing emaciation and keeping up the strength. It is said to be less useful in proportion as the age of the individual is more advanced. If it gives rise to nausea and vomiting, cream may sometimes be substituted for it, or glycerine, but they are not as good. With perseverance *small* doses of cod-liver oil can almost always be taken, at least during cold weather. It is best taken after meals; alone, or in orange or ginger wine, or as an emulsion. Chewing a bit of orange peel is the best way to prevent the after-taste. In children cod-liver oil is often rubbed into the skin, but the smell is extremely unpleasant, and probably olive oil is just as good.

The *cough* of phthisis has to be combated by the usual remedies. Most prescriptions contain a small dose of opium or morphia, together with tolu, aniseed, benzoic acid, or some other of the so-called expectorants.

The only other symptom that needs special mention is the *night sweating*. This may sometimes be checked by sponging the chest and the arms at bedtime with vinegar and water. Sometimes it ceases if a subcutaneous injection of atropine ($\frac{1}{200}$th to $\frac{1}{100}$th of a grain) is given at bedtime, or a dose of belladonna, oxide or sulphate of zinc, gallic acid, or strychnia. But in too many cases it persists in spite of all treatment.

The first object in the early stages of phthisis, or of threatened phthisis which has not yet begun, is to improve the patient's *appetite and digestion*. This is one result of change of climate. If he gains weight he generally is doing well. Usually small doses of alkalies, with the milder and less astringent bitters and gentle laxatives, act best in this direction. The combination of carbonate of soda with rhubarb and calumba, long famous at Victoria Park and other hospitals, is excellent for the purpose. In the later stages laxatives must be administered with great caution, for nothing is then more injurious than diarrhœa. Sometimes strychnia and gentian or chiretta are borne well and help the appetite.

In diet, bacon, milk, butter, cream, dripping, are indicated. Wine with meals is as a rule desirable; but in early cases, before there is much cough, malt liquors, and particularly porter, are more useful. Brandy is as a rule best adapted for advanced cases, and even then should be given in moderate doses; but rum and milk, either before breakfast or between breakfast and lunch, is a well-known and useful way of giving a nourishing stimulant.

In most cases of phthisis there is early and marked *anæmia*, indicating the use of steel. This may be given as the sulphate, with small doses of Epsom salts, or in one of the alkaline preparations, or (if borne well) in the most efficient form, the muriated tincture, with glycerine and quassia or calumba. Arsenic seems sometimes to succeed when preparations of iron fail, and now and then has a remarkable effect in restoring appetite, and adding fulness as well as colour to the cheeks. Lately it has been given with salicylates.

No drug has any claim to be considered a "specific" remedy in consumption, *i. e.* a remedy which does good apart from its immediate effect upon indigestion, or anorexia, or anæmia. Cod-liver oil itself probably acts only as the most easily assimilated fatty food. The alkaline hypophosphates are given by respectable authorities with belief in their usefulness, but not with the least expectation of any specific or remarkable effect.*

Treatment by compressed air,† by oxygen, and by inhalation of other gases, is either useless or of very limited benefit. A mode of treatment, lately introduced by Dr Bergeon, of Lyons, gaseous enemata of sulphuretted hydrogen, has no theoretical probability to recommend it; it is unpleasant in its action, and after being tried (perhaps with more patience than was due to it) has been shown to be useless.

Of late, however, those who believe that the bacilli of tubercle are the efficient cause of phthisis have naturally attempted to destroy them by antiseptic methods. Iodoform and thymol have been given by the mouth with this object and more recently sodic sulpho-carbolate and phenyl-propionic acid. Creasote, thymol, and eucalyptol have been inhaled by means of ori-nasal respirators (of which Sir William Roberts, Dr Burney Yeo and others have devised ingenious forms); steam inhalers have been supplied with such antiseptic agents as creasote or carbolic acid, and similar solutions have been administered by Siegle's spray apparatus. Sometimes apparent benefit to the local and general symptoms has resulted, and the number of bacilli in the sputum has diminished. But experiments in the laboratory show that Koch's bacilli are extremely difficult to kill, and survive prolonged immersion n germicide solutions. Iodine seems to have the most power in destroying them. Perhaps the most ingenious method of "bacillicide" treatment is that introduced by Dr Cantani, of Naples, who introduces the common *Bacterium termo* of putrefaction into the lungs in a spray, with the hope of its destroying the specific bacillus. Unfortunately the latter is the more powerful of the two; and Dr Theodore Acland reports, after a visit to Naples, that the results of this treatment are negative.‡

* It is interesting to note the list of drugs which Louis in 1843 thought most noticeable. They are iodide of iron, chloride of sodium, carbonate of potash, chloride of ammonium, chloride of calcium, chlorine gas, digitalis, hydrocyanic acid, creasote and iodine.

† See some recent lectures by Dr T. Williams ('Brit. Med. Journ.'), 1885, vol. i, p. 769.

‡ For further information on this point, see the last chapter of Dr Williams's work.

MILIARY TUBERCULOSIS OF THE LUNGS

Distinction from phthisis—Morbid anatomy—Physical signs—Clinical symptoms and course—Diagnosis by concomitant tuberculosis, especially of the choroid—Ætiology—Prognosis—Asserted cases of recovery and possible treatment.

Both from a clinical and from a pathological point of view it is necessary to distinguish from cases of phthisis—in which tubercles spread through the lung from the apex downwards and produce softening and excavation—those of miliary tuberculosis, in which each tubercle appears to be the result of the deposition in the pulmonary tissue of a particle of virus (probably a bacillus, or the spore of a bacillus) brought to the organ from elsewhere by the blood-current. True, the distinction is not absolute. For, on the one hand, in many instances of phthisis, dissemination by the blood-current sooner or later occurs, and often constitutes the immediate cause of death. And, on the other hand, it is probable that in some instances of miliary tuberculosis the tubercles, originally not very numerous, at length form the starting-points of a process of local infection, extending from each of them as a centre, until an affection exactly like phthisis is developed. The cases which most demand attention in this chapter are those in which the lungs, previously healthy or but slightly affected with phthisis, become suddenly the seat of such immense numbers of tubercles that acute symptoms arise and life can no longer be maintained.

It must, however, be remembered that other organs are almost always attacked at the same time. If tubercles appear in the membranes of the brain, they generally (but not invariably) give to the disease its main clinical features. If the peritoneum is much involved, the abdomen alone may appear to suffer. So that it is often almost an accident whether a case is regarded during life as one of tubercular meningitis, or tubercular peritonitis, or of miliary tuberculosis of the lungs; while there are cases which run their entire course without definite clinical localisation. One cannot even say absolutely that the degree of severity of the pulmonary symptoms is in every instance directly proportioned to the number of miliary tubercles in the lungs. In cases which are classified as examples of tubercular meningitis the lungs are often intensely affected.

Anatomy.—With regard to the morbid anatomy of miliary tuberculosis of the lungs there is little, if anything, to add to what was stated in the chapter upon Tubercle in general. As was there remarked (p. 93), many cases occur in which, from the greater abundance of the tubercles in the upper lobes, and from their more advanced state there than towards the bases, it is clear that the proclivities of the pulmonary tissue in different regions produce their effect on this disease, as well as in phthisis. It has also sometimes been noticed that when a general outbreak of miliary tubercles occurs throughout the body of a patient who previously had the apex of one lung affected with phthisis, there are found in that lung more numerous (or

perhaps much larger) tubercles than in the opposite one, from which fact we may infer that it had a greater inherent tendency to the occurrence of tubercles in it, and that this manifested itself when their development was acute no less conspicuously than when it was chronic. The characters of the tubercles themselves vary widely in different cases. Sometimes they are mainly lymphoid in structure, sometimes they are almost entirely catarrhal. Sometimes they are grey, and tend not to caseation, but to fibrous change or to horny induration (p. 80). Sometimes they become cheesy almost as soon as they are formed. In some exceptional instances, and only towards the apices, they are found to have already softened in their centres with minute vomicæ.

Signs.—Clinically the recognition of this, as of all other pulmonary diseases, depends partly upon physical signs, partly upon symptoms.

The physical signs of miliary tuberculosis of the lungs are in most cases vague and doubtful. Jürgensen, indeed, described in the 'Berliner klin. Wochenschrift' for 1872, a case in which during five days he heard over a large part of both sides of the chest a peculiar soft, rubbing sound, perceptible also to the touch; when death occurred, two days later, the only cause to which this sound could be attributed was the presence of a number of miliary tubercles situated on the right side beneath the pulmonary pleura, which was free, and on the left side in the substance of adhesions which completely closed the cavity. From the very first day, the soft quality of the sound led him to conclude that it was produced by miliary tuberculosis, and not by pleurisy. The patient complained of no pain, and could draw a deep breath without embarrassment. Jürgensen thinks that in future cases a positive diagnosis may safely be based upon this sign. Burkart has since maintained (in vol. xii of the 'Deutsches Archiv') that he has twice detected with the hand a friction-sound, due to the presence of obsolete tubercles, which was not soft, but rough.

In all probability miliary tubercles are never set sufficiently close together, even in the apex of a lung, to impair the percussion-resonance of the corresponding part of the chest. Sometimes it appears doubtful whether the sound is not slightly dull beneath one or both of the clavicles; but if this is so, the dulness is most likely due, not to the tubercles themselves, but to the collapse of the surrounding pulmonary tissue. Dr Eustace Smith remarks that in children this interpretation is borne out by the fact that variations may be observed from day to day, the resonance becoming good where it had been deficient; and the case of an adult patient will be presently mentioned in which the same thing seemed to occur. Again, it is not uncommon for the presence of pulmonary emphysema to render the percussion-sound over-resonant; and the progressive emaciation of the patient tends to modify it in the same direction as the case goes on.

With the stethoscope one may be able to detect absolutely nothing abnormal, even where tubercles exist in enormous numbers. But in some cases, especially towards the apex, the vesicular murmur has a harsh quality, the cause of which is not very apparent. More frequently the auscultatory signs of bronchitis are present, sometimes to an extreme degree. Not only may sibilus and rhonchus be audible more or less extensively, but there may be also abundant moist sounds, from fine and crepitant up to much larger and coarser râles. The expiration, too, may be prolonged and wheezing. On *post-mortem* examination bronchitis is found in such cases, the

smaller tubes being reddened and filled with muco-pus. But sometimes the moist sounds are so bright and clear—so consonating in quality—over the upper lobes, that it is difficult to believe that there is not diffused infiltration, with "breaking up" of the pulmonary tissue. In one such case which occurred at Guy's Hospital in 1874, there were in fact a large number of small cavities, especially in the left apex. These were evidently of older date than the general eruption of miliary grey tubercles, which filled every organ in the body, and it appeared from the history that the patient had had a cough for three mouths, whereas his more acute illness began only ten days before death. But in another case, in 1868, it is reported that there were "mucous râles" at the left apex, "gurgling" at the left base, and "pneumonic crepitation" over the right upper lobe; and yet the tubercles were nowhere seen softening, the only source of the moist sounds being pus in the smaller tubes. In other instances *post-mortem* examinations have shown that the lesions were much less advanced than had been thought during life. One such case occurred in 1882 in a woman, aged twenty-five, in Guy's Hospital. When she was admitted, on July 19th, the only physical sign was a slight crackling sound heard at the right apex after she coughed. However, on the 28th there was a marked crepitant râle in both upper lobes, and especially along the anterior edges of the lungs; and during the next three or four days its character became so "consonating" that we were almost disposed to look upon the disease as acute phthisis rather than as miliary tuberculosis. But at the autopsy, made on August 4th, the lungs, though bulky and œdematous, everywhere contained air; the tubercles were discrete, nowhere softening, and caseous only in the upper lobes; the tubes yielded a frothy fluid.

Clinical symptoms.—These fall under two heads. On the one hand there are cough, dyspnœa, and other indications of embarrassment of the breathing; on the other hand there is pyrexia.

There is always more or less troublesome *cough;* it is generally short and hacking, and often dry.

When expectoration is present it sometimes consists of clear mucus; sometimes it is muco-purulent. There are not infrequently streaks of blood in it, and it may be quite "rusty" in appearance, like the sputa in acute pneumonia, or even plum-coloured. Actual *hæmoptysis* in any considerable quantity is not common. But in 1869 there was brought into Guy's Hospital the dead body of a child, aged five, who was said to have been well on the previous evening, and to have eaten some herring for supper. In the course of the night it was found to have brought up blood, and to be in an alarming state, and it died on its way to the hospital. An autopsy showed that there was an acute general tuberculosis; and some of the tubercles in the lungs were already caseating, especially in the upper lobes. No definite source for the bleeding could be discovered; the pulmonary tissue was mottled with blood drawn into it by inhalation. In all probability the cause of hæmoptysis in such cases is the extremely congested state of the vessels immediately outside the tubercles, which often gives them the appearance of being surrounded by a reddish-brown border after death; in fact, obvious points of capillary hæmorrhage may sometimes be seen, not only in the lungs, but in other organs.

Far more significant is *dyspnœa.* At first the breathing is only hurried, the number of inspirations in the minute gradually increasing until it

reaches fifty to sixty, or in children eighty to ninety. In the woman aged twenty-five, whose case was mentioned above, it was counted at fifty-six on the very day of her admission. After a time the patient becomes conscious of shortness of breath; there is orthopnœa, the movements of the thoracic muscles are forced, and the nostrils work. The cheeks, the lips, the fingers, and the nails are of a lilac or purple colour. This symptom is one which more than any other suggests the idea of pulmonary tuberculosis to the experienced physician, if there is no long-standing emphysema or heart disease to account for it. I have notes of only one case in which it is said to have been absent. Sometimes albumen appears in the urine. There is not infrequently slight œdema of the lower limbs, and the face becomes puffy and swollen. In the case of the young woman already referred to, the urine contained sugar during the first few days after her admission into the hospital, the proportion being on one occasion 0·4 grain, and on another occasion 0·265 grain in the ounce. Senator's experiments seem to show that this cannot be attributed to deficiency of oxygen in the blood; and, indeed, as the case went on and marked cyanosis developed itself, the glycosuria ceased.

Pyrexia seems to be invariably present, but it varies greatly in its degree and in its course. Sometimes the temperature ranges up to 104° or 105°, but more often it remains at a lower level, perhaps not at any time exceeding 102°. Its progress is irregular. For two or three days there may be scarcely any differences in the thermometric readings at different periods of the twenty-four hours; and then the usual diurnal variations may appear in an exaggerated form, or what is termed the *typus inversus* may show itself, the morning temperature being higher than that of the evening. Brunniche is said to have observed this in fifteen cases out of seventeen, so that it would appear to be decidedly more frequent than in other febrile diseases. Jürgensen makes it a point that the pyrexia does not yield to tepid baths or to antipyretic remedies like quinine so readily as in the specific fevers. This opinion, however, seems to have been partly theoretical, and based upon the idea that in tuberculosis the high temperature of the body generally is the result of the local morbid process, just as when there is inflammation. But since the discovery of the bacillus, there is surely a probability that it is the presence of this organism in the blood which is the chief cause of the rise of the thermometer; and, if so, the distinction from a specific fever can no longer be maintained.

The onset of the pyrexia is usually gradual, and the patient does not take to his bed until he has been ailing for some days. But Rühle, in 'Ziemssen's Handbuch,' speaks of an initial rigor as not infrequent. There are the ordinary early febrile symptoms of headache, malaise, depression, intense thirst and loss of appetite. The skin is often wet with perspiration. Epistaxis occurs in some cases, and herpes may appear about the mouth. The pulse is generally very rapid—often out of all proportion to the height of the temperature. There may be a flush on the cheeks, but the face is more usually pale before it becomes livid. At one time it was taught that in miliary tuberculosis enlargement of the spleen is exceptional, and that if it occurs at all it is only slight. But all observers seem now to be agreed that some degree of swelling of the organ is almost constantly to be detected by careful percussion, at least when the disease is at an advanced stage. Rühle says that if tubercles are developed in the spleen it may become as large as in enteric fever, and may be tender on pressure.

Towards the end a typhoid state may develop itself, with sordes, a dry brown tongue, subsultus, delirium, and coma. Death is sometimes preceded by a rise of temperature, sometimes by a fall and by collapse.

Diagnosis.—In many instances one is much assisted in the recognition of miliary tuberculosis of the lungs by indications of a like affection of some other organ. Thus the case may at any period of its course become complicated with symptoms of tubercular *meningitis* in a more or less marked form. Sometimes, though very rarely, there is more or less *jaundice*, due to the presence of very numerous tubercles in the liver. In other exceptional cases the occurrence of a tubercular inflammation in one or more of the *joints* may perhaps aid in clearing up a doubt as to the nature of the disease. A striking example of this was recorded by Laveran in the 'Progrès Médical' for 1877. A man, aged twenty-two, was attacked with articular pains, especially in the knees. Effusion occurred into the right knee-joint, and when admitted into hospital he was supposed to be suffering from subacute rheumatism. However, at the end of a week, great dyspnœa set in and high fever, the temperature ranging from 102° to 104°. A fortnight later he died, the cause of death being acute tuberculosis. The synovial membrane of the right knee was found to be injected and covered with a large number of greyish granulations the size of pins' heads, which could be felt. Where they had been in contact with a surface of articular cartilage their summits were flattened. A few granulations were present also in the left knee. In 1867 a woman died in Guy's Hospital of tubercular meningitis, whose right knee had become swollen and painful in the course of her illness. At the autopsy all that was noted was that the synovial membrane was very vascular and œdematous, and that the cartilage over the external condyle of the femur was slightly eroded. But it is not at all unlikely that tubercles were present. In 1880, in a patient who died of cancer of the stomach, and who had suffered from a painful affection of the knee attributed to an injury eight months back, the soft tissues of the joint were found full of the most conspicuous tubercles, many yellow and caseating; but this is hardly a case in point. Cornil and Ranvier have shown that in cases of acute tuberculosis miliary granulations may often be found in the cancellous tissue of the bones, especially in the vertebræ, the sternum, and the ribs. It seems quite possible that their presence may sometimes give rise to pains vaguely referred to different parts of the limbs and body, but probably these could not be distinguished from the similar pains which so commonly accompany various forms of pyrexia.

From a clinical point of view, the most important disease to distinguish from acute tuberculosis is enteric fever. Both diseases occur in patients of the same age. Both are attended with pyrexia, both run an acute course, and in both the lungs are constantly, the intestines and peritoneum sometimes, the seat of disturbance. The difficulties of diagnosis have already been described in the first volume (p. 176). Here we may remark that the temperature in tuberculosis is less regular and interrupted by subnormal readings; that the ratio of respiration to pulse and temperature is greater, and that cyanosis comes on earlier. Moreover, some chronic disease of the pulmonary apices is often to be discovered. The rash and characteristic stools are of course absent, but both may be so in enteric fever.

As a help to diagnosis, the most important of all the seats of miliary tubercles is one where they can actually be seen during life, although

it seldom if ever happens that there are symptoms which draw attention to their presence. In 1857 a German observer, Manz, discovered tubercles in the *choroid* of each eyeball, in the body of a girl who had died of acute tuberculosis. Subsequently he and Busch recorded other instances of a similar kind. And in 1867-68 Cohnheim, investigating this point carefully in all the cases of miliary tuberculosis—eighteen in number—that came under his notice in the Pathological Institute at Berlin during a period of fourteen months, found that in every instance one or both of the eyes—almost invariably both of them—showed choroidal tubercles. In April, 1867, the ophthalmoscope was for the first time used, apparently by v. Graefe himself, for the discovery of these tubercles during life in a patient of Griesinger's In November of the same year Mr Soelberg Wells exhibited to the Pathological Society of London a specimen of choroidal tuberculosis, which he had detected in a little girl under the care of Dr Garrod, five days before her death. Since then many other observers have recorded similar facts. But further experience seems to have shown that the eyeballs are very far from being so constantly involved in cases of miliary tuberculosis as would be supposed from Cohnheim's statement. It is to be noted that the development of tubercles in the choroid appears to bear no specially near relation to their occurrence in the pia mater of the brain ; all that can be said is that the more numerous the organs which are the seats of tubercle in a given case, the more likely is it that the eyeballs will be affected. One of the most remarkable cases was recorded by Fränkel in 1872 in the 'Berliner klin. Wochenschrift.' A delicate girl of six was attacked in May of 1871 with slight shiverings, and her temperature rose occasionally to 100·4°. Then partial ptosis appeared and afterwards paralysis of some of the ocular muscles. On May 22nd the ophthalmoscope showed a white patch to the upper and inner side of the disc in the left eye ; it was as large as the disc itself, and had a rounded form, except that in one direction it was drawn out into a point. By the 1st of June it had increased in size by one half. On account of its characters being so different from those generally described as belonging to tubercles in the choroid, Fränkel hesitated to diagnose it as tubercular. The child now went into the country with her parents and remained there until August, when she came back apparently in perfect health. The patch in the fundus of the eyeball, however, was more prominent, though not larger than before. On August 21st she became ill with gastric symptoms and pyrexia, and died on October 1st. On September 10th five fresh miliary tubercles had been detected in the choroid, and afterwards a sixth made its appearance ; as the case went on they gradually increased in size. Vision remained unimpaired until death.*

In acute cases the number aud the size of the tubercles may increase from day to day, as was noticed in a child examined by Fränkel and by Leber. If, however, nothing should at first be detected by the ophthalmoscope in a suspected case of general miliary tuberculosis, the instrument ought to be used again and again as the disease advances. The tubercles vary much in size. The largest seen by Cohnheim was 2·5 mm. in diameter ; but Ponfick met with one that measured 5 mm. On the other hand, Cohnheim seems often to have detected them (in the dead body) where they could only be seen after carefully removing the choroidal pigment, and even where they

* As a rare instance in which there was any defect of sight, may be quoted that of a girl in whom, besides from forty to fifty tubercles in each choroid, Cohnheim found also hæmorrhages into the retinæ.

were too small to be visible by the naked eye. It is possible that this may account for the discrepancy between his statements and those of other observers as to the constancy of their occurrence in cases of general miliary tuberculosis. Where there are but few of them they seem to be developed in the neighbourhood of the disc or of the yellow spot more often than towards the equator of the eyeball. When very numerous, some of them may run together into irregular masses. They almost always become caseous as soon as they reach about 1 mm. in diameter. With regard to the ophthalmological diagnosis between tubercles and the white spots that are seen in disseminated choroiditis, von Graefe and Leber insisted on the rounded form generally presented by tubercles, their projecting above the level of the choroid, the gradual thinning away of the choroidal pigment from their periphery inwards to their centres, which appear white, and the absence of any accumulation of pigment outside them, except perhaps when they are very large.

Ætiology.—With regard to the causes of miliary tuberculosis of the lungs there is very little to be noted. Whatever may be said theoretically as to its relation to caseating tubercular glands, it is very seldom directly traceable to any such affection which had been recognisable during life in the neck or elsewhere. Many of those who are attacked, whether children or adults, are robust and healthy looking, altogether devoid of the supposed indications of a scrofulous tendency. On the other hand, one must always be prepared for the supervention of miliary tuberculosis in cases of phthisis, for it brings the disease to an end far more quickly than might have been anticipated.

Acute tuberculosis occurs at all ages. Among forty cases observed at Guy's Hospital between the years 1857 and 1873, one was in an infant aged nine months, three were in patients between one and ten years, seven between eleven and twenty, thirteen between twenty-one and thirty, six between thirty-one and forty, four between forty-one and fifty, six between fifty-one and sixty. But it is to be observed that this list does not include the cases in which the clinical symptoms were those of tubercular meningitis ; if they were taken in, the proportion of children would of course be far higher. Burkart gives very similar figures. Among the above cases there were almost exactly twice as many males as females ; among Burkart's the proportion was as sixteen to two.

It is worthy of notice that in two of the Guy's cases the patients were admitted into the hospital for what appeared to be acute pleuro-pneumonia. One, a young man, aged twenty-two, at first complained of pain round the lower part of the right side of the chest, which seemed to be the seat of his disease. He appeared to be doing well, and was allowed to go out to the hospital grounds, when he was seized with an epileptic fit. He was then carefully examined again, and was found to have ascites. The fits continued, and he passed into a state of stupor. Crepitation became audible throughout both lungs, and at length he died, ten weeks after his admission. At the autopsy, the pleura was found to be thickened over the base of the right lung, and there was fluid. The right lower lobe was completely airless ; it contained only a few scattered miliary tubercles, forming a marked contrast with all other parts of the lungs, which were full of them. The pericardium was adherent and both layers of it were enormously thickened ; nothing is said about its containing tubercles. The liver and the kidneys all contained

many tubercles, the liver also was cirrhotic. There appeared to be only a few tubercles in the pia mater. The other patient, a youth of eighteen, came in three months and a half before his death with an attack of pleuro-pneumonia, which (in his case also) was on the right side. Afterwards it appeared, according to the clinical report, that phthisis developed itself. On *post-mortem* examination the lungs were found to be studded with miliary tubercles, especially the right one. There were no cavities anywhere, nor was the pulmonary tissue solidified round the tubercles. The substance of the right lung was unusually firm, a condition which was thought to be the result of the primary pneumonia. The liver and the kidneys contained miliary tubercles. The small intestine showed characteristic tubercular ulcers running transversely; the mesenteric glands and those near the cæcum were caseous. It is doubtful whether the relation of the miliary tuberculosis to the pneumonia in these cases was more than one of coincidence. In children the disease often follows measles, scarlet fever, and smallpox.

Prognosis.—The duration of the disease is commonly three to four weeks, reckoning from the first commencement of marked symptoms up to the time of the patient's death. But, on the other hand, there may be a protracted illness, lasting three or four, or even eight months. Clinically, acute pulmonary tuberculosis generally resembles severe bronchitis more than any other affection, and is not seldom mistaken for it until an autopsy reveals the real nature of the case. Indeed, as Burkart has pointed out, it is common for miliary tuberculosis to develop itself in lungs which are already emphysematous from old bronchitis. Twelve of his eighteen cases were examples of this, and their course was often very prolonged. In no fewer than six of these, in fact, the tubercles which were found *post mortem* were already calcified or fibrous, with a lustre like that of mother-of-pearl, so that they might fairly be considered obsolete; and the causes of death were various, sometimes pleural effusion, sometimes dilatation of the right heart, sometimes Bright's disease; the presence of the tubercles seemed to be little more than an accident. A similar obsolescence of miliary tubercles scattered through the lungs has been observed by the writer, who was not, however, sufficiently careful in determining what part they took in bringing the patient's illness to a fatal termination. It is worthy of notice that ten of Burkart's cases occurred in men exposed to the dust of stone or coal in their work, for this fact bears out the belief as to the tubercular character of "pneumonoconiosis" expressed above (p. 239); unless, indeed, it could be urged that the bodies found in the lungs by Burkart were not really tubercles at all but fibrous nodules formed round embedded particles of silica or carbon.

This question of the possible obsolescence of miliary tubercles in the lungs is of great importance, in reference to the diagnosis from enteric fever of those cases which run a rapid course, and which are attended with pyrexia as a principal symptom. A case of Senator's is quoted in the first volume (p. 186) which shows that it is sometimes impossible, in the present state of our knowledge, to distinguish the two affections. In this country, it has been usual to regard the fact that a patient recovers as conclusive proof that he was not suffering from miliary tuberculosis; although Dr Bristowe says that the progress of the disease "may be occasionally arrested, but with more or less permanent damage to the tissue of the lung." Now, in cases of fatal tubercular meningitis, we often have opportunities of observing that the number of miliary tubercles scattered through the lungs

may be very small. And it is certainly difficult to understand why, if the brain-affection happened in such a case to be absent, recovery should not take place. All that would be necessary would be that the tubercles should fail to become the starting-points of fresh tissue-infection. It may, of course, be a question whether such a scanty development of miliary tubercles would be attended with pyrexia or other clinical symptoms. But there is reason to think that the high temperature in this disease is in great part independent of the local lesions, and due to infection of the blood. Wunderlich, as far back as 1860, recorded in the 'Archiv für Heilkunde' cases which he regarded as examples of cured miliary tuberculosis. But he based his diagnosis solely on the fact that the temperature-chart failed to correspond with what he regarded as the necessary course of enteric fever. Few observers will be found at the present time to endorse his opinions in this respect. The points of distinction between the thermometric readings in the one and in the other disease will appear from the description given above (p. 265) compared with that in vol. i, p. 171. Wunderlich attached great importance to the occurrence of a prolonged "amphibolic stage," with alternate exacerbations and remissions (sometimes amounting to complete apyrexia) as conclusive against the presence of enteric fever in his supposed cases of recovery from acute tuberculosis. But perhaps the only way in which the occurrence of recovery from an attack of miliary tuberculosis could be proved by clinical evidence alone would be by the discovery of tubercles in the choroid of the eye. Or the patient, after getting well, might die from some other cause, and the tubercles be found in a state of obsolescence. In regard to this, Dr Bristowe speaks of the lung, after the arrest of the discrete tubercles, as becoming "seamed throughout with minute patches of cicatricial tissue, the fibres of which have something of a stellate arrangement, and within the limits of which the lung tissue presents, from the presence of concurrent emphysema, a coarsely spongy character; occasionally in the centres of the scars minute fibroid knots or concretions may be recognised."

Burkart records the case of a woman, aged twenty-eight, who died after a fortnight's illness, and in whom the lungs, the peritoneum, and the kidneys showed recent miliary tubercles in abundance, while in the intestine there were the typical lesions of enteric fever, some of the ulcers having sloughs still adherent, or partially detached. He also cites eight cases recorded by Birch-Hirschfeld, in which acute tuberculosis is said to have developed itself immediately after enteric fever.

With regard to the *treatment* of miliary tuberculosis of the lungs, all that can be said is that the strength of the patient should be maintained, that symptoms should be checked by appropriate drugs, and that perhaps bichloride of mercury or phenylpropionic acid should be given in the hope of favouring the obsolescence of the tubercles.

FUNCTIONAL DISORDERS OF THE RESPIRATORY TRACT

CORYZA OR NASAL CATARRH.—*Its symptoms, predisposing causes and treatment —Feverish colds.*
INFLUENZA OR EPIDEMIC CATARRH.—*History—Symptoms—Events—Ætiology —Diagnosis—Treatment.*
OZÆNA.—*Origin in atrophy of the nasal tissues—Minor forms—Treatment.*
EPISTAXIS.—*Ætiology—Course—Treatment.*
HAY FEVER OR SUMMER CATARRH.—*Onset and Course—Ætiology—Distribution among persons and places—Treatment.*
WHOOPING-COUGH.—*Nomenclature—Symptoms and course—Events—Prognosis —Pathology—Treatment.*
ASTHMA.—*The term—Description of an attack—Diagnosis and relation to structural lesions of the lung—Ætiology—Nature and physiology of the disease—Treatment and prognosis.*

IN the present chapter will be described certain affections of the air-passages and lungs which cannot be classed with structural lesions. Indeed, but for practical convenience, they might find their places in the chapters on specific fevers, on spasmodic disorders, and on affections of the blood-vessels.

NASAL CATARRH.—*Coryza.—Cold in the head.*—Almost everyone, at least in climates like that of England, is liable from time to time to a "cold in the head." This affection commonly begins with sneezing, repeated again and again. The nose becomes dry and "stuffy," its mucous membrane may be seen to be swollen and injected, there is difficulty in breathing through it, and the nasal consonants *n*, *m*, *ng*, cannot be properly pronounced. Often from the very commencement there may be a profuse flow of a thin, saltish, watery fluid from the nostrils, so that the handkerchief has to be used almost uninterruptedly. Smell and taste for the time are completely abolished. If the frontal sinuses are involved, there is dull pain in the forehead, or even severe headache. Extension to the lachrymal passages causes the tears to flow over the cheeks, and the conjunctiva becomes injected. Extension to the fauces and Eustachian tube may give rise to deafness. The skin near the nostrils often becomes inflamed and excoriated, a result which is attributed to the "acrid" character of the discharge. In this fluid leucocytes appear to be always present, though sometimes only in small numbers; and, according to Hüter, micrococci are also abundant.

Sometimes an attack of nasal catarrh passes off as suddenly as it began. In two or three hours the flow may cease entirely; or, the person having been tormented with a "running cold" up to the very moment of going to bed, may forthwith fall asleep, and on waking in the morning may find himself entirely free. According to Trousseau, such a transitory coryza is

sometimes an irregular manifestation of asthma, either leading directly to a genuine attack of that disease, or at least occurring in individuals subject to it. In cases of this kind there is little or no constitutional disturbance. But the more common kinds of nasal catarrh often set in with marked malaise and chilliness, and in children they may be attended with considerable pyrexia. The usual duration of such an affection is from two days to a week, but sometimes it lasts considerably longer, or (it would perhaps be more correct to say) a series of attacks occur in succession, a fresh one setting in each time as the other is passing off. Whenever coryza runs on for more than a day or two, the discharge from the nose alters in character, becoming viscid, opaque, and muco-purulent. Sometimes, as the nasal affection subsides, the larynx and the trachea are in their turn attacked with a like catarrhal inflammation. Lastly, a severe cold is in certain cases followed by a long-continued impairment or loss of the sense of smell.

In regard to the *causes* of coryza, it is in the first place to be noted that some individuals are far more liable to it than others, and that it is also especially apt to occur in certain families. There are persons who are sure to take cold if they get their feet wet; but such an over-susceptibility to catarrh may often be controlled by cold sponging in the morning, by the regular use of a shower-bath, by keeping the bedroom supplied with fresh air during the night, or by other like measures. Among the public it is almost universally believed that nasal catarrhs are contagious; they are supposed to be easily conveyed from one person to another by handkerchiefs, or by the act of kissing. A suggestive observation tending to support this notion is the statement made by Fränkel (in 'Ziemssen's Handbuch') that he has repeatedly seen a newly-married man, who had never before had a cold, attacked without any other obvious cause shortly after the wife, she having before been subject to the affection. On the other hand Friedreich is said to have inoculated his nasal mucous membrane with discharges from persons suffering from coryza without any result. It is as yet uncertain whether the very great prevalence of coryza at certain seasons of the year is to be regarded as constituting a real epidemic, or merely as a consequence of the widespread distribution of the common exciting causes of the affection.

As a special variety of nasal catarrh must be mentioned that which arises in certain individuals when they are taking iodide of potassium medicinally; this is supposed by Fränkel to be due to direct irritation of the lining of the nose by iodide dissolved in the mucous secretion.

It must not be forgotten that coryza is a prominent early symptom of measles, and sometimes of typhus.

Again, an inflammation of the nasal mucous membrane that may be mistaken for an ordinary catarrh, is sometimes dependent upon an infection of the nose with leucorrhœal or gonorrhœal discharge. In such cases, however, the exudation is from the first yellow and purulent, or mixed with blood, and the duration of the affection is much more protracted, lasting several weeks. Dr Hermann Weber, in vol. xliii of the 'Med.-Chir. Transactions,' seems to have first drawn attention to the possibility of the nasal cavities of an infant becoming infected during birth when the mother has leucorrhœa. Fränkel thinks that such an occurrence is by no means infrequent. To obviate it one might in suitable cases employ an injection of some antiseptic fluid just at the commencement of the second stage of labour. In the 'Lancet' for 1857 Mr A. M. Edwards relates a case of nasal gonorrhœa in a

woman, who was ultimately shown to have caught it by using a handkerchief that had been employed as a suspensory bandage by her son.

Treatment.—Coryza is not a disease about the treatment of which one is often consulted, for most people manage their attacks themselves. It is best to keep indoors, or even in bed. At the commencement, ten grains of Dover's powder may be taken at bedtime with a basin of hot gruel, and next morning five grains of quinine. A warm bath is also useful; free perspiration seems to favour subsidence of the affection. Locally, marked relief often follows the insufflation of bismuth mixed with a little morphia, as suggested by Dr Ferrier ('Lancet,' 1876, vol. i, p. 523). Dr C. J. B. Williams recommended abstinence from all liquids. If there is no fever and the patient is allowed lozenges or orange-peel to suck, or a few teaspoonfuls of tea at intervals, the restriction is tolerable and prevents the profuse coryza. It applies best to those who are obliged to go about their work.

When nasal catarrh, instead of subsiding, persists for weeks together, change of air is often the best remedy; but the internal administration of arsenic is sometimes very useful under such circumstances, and striking results have been obtained from the same medicine when loss of taste and smell as the result of coryza had continued for many months; in one case, however, it entirely failed.

Feverish colds.—Sporadic influenza.—The usually trivial disorder just described has considerable pathological interest. It is generally assumed to be the result of a chill, and few who are subject to it but can recall instances in which it follows directly and unmistakeably as the result of exposure to a cold wind, or to rain, or to a draught of air. The first effect is a sudden feeling of chilliness, an involuntary shudder (*i. e.* a slight rigor), and a fit of sneezing. The reflex character of this spasmodic neurosis is shown by its being caused by a bright light irritating the eyes, as well as by cold air in the face or snuff in the nostrils, and also by its being prevented (inhibited) by pressure on the upper lip or bridge of the nose, and completely stopped by covering the face.*

But the same question arises which met us in the case of pneumonia. What seems a local inflammation determined by a chill to the surface, like bronchitis or pleurisy, assumes from another point of view the aspect of a specific fever, like measles. It sometimes comes on with no unusual exposure to cold; it "runs through the house" like a contagious disease; it is accompanied by malaise and pyrexia; it lasts for a limited time, and then subsides. In one point it differs from other specific fevers, in that it does not protect against itself; or rather, perhaps, it protects for only a short time, for while relapses from an ordinary cold in the head are very frequent, a severe feverish cold rarely occurs except at considerable intervals.

The connecting link between the ordinary local coryza, described in the previous section, and the epidemic influenza, to be next considered, is furnished by what is somewhat loosely named a "feverish cold."

This, like the less severe and more local form, is usually the result of exposure to cold—particularly to an east wind blowing on the face—but it is sometimes undoubtedly caught from another case of the same kind, and sometimes it is idiopathic, *i. e.* of unknown origin.

The symptoms begin with shivering or rigor, then there is usually pain

* The action of the domestic remedy of smearing the nose with tallow or cold cream is to prevent sneezing by protecting the skin from cold.

in the throat felt on swallowing, together with headache and severe aching pains in the loins, the thighs, or the back and limbs generally. To this stage succeeds coryza and a short, dry, painful cough. The temperature rises, sometimes to 103° in an adult and often to 102°, the urine becomes febrile, the pulse quick, and the skin dry. After a day of distress, the skin begins to act, and the symptoms are somewhat relieved, but the coryza becomes more severe, a little mucus is expectorated, the bowels are constipated, and headache and pains in the limbs continue. After three, or at the latest four days, the feverish stage is almost always over, but there remains great prostration of strength for so short an illness, and considerable bronchial as well as nasal catarrh. Finally, there remains some cough and expectoration for a week or ten days more. Occasionally a sharp attack of diarrhœa seems to take the place of the nasal catarrh.

The *treatment* is first to avoid fresh chills, and secondly to keep warm. A hot bath or a Turkish bath is excellent, and for the time the patient feels well, but they do not cure; within an hour he is as ill as ever. He must go to bed, drink warm diluents, and get the skin to act. Dover's powder is certainly useful with most people. Others find antimonial wine more effectual in producing perspiration and relieving the pains and oppression. Low diet, salines, and laxatives are indicated, with constant warmth to the feet, the face, and the hands. Thus the patient gets comfortably through his malady. During convalescence quinine is the best tonic.

As prophylactics, covering the face and hands, or wearing cotton wool in the ears and nostrils, are the most effectual.

INFLUENZA.*—There is yet another form of catarrh of the nose, throat, and air-passages which, from the manner of its distribution as an epidemic, is evidently a specific disease. This is the *influenza*, as it is termed both in England and in Germany, the name being of Italian origin and having come into use in 1741: in France it is known as *la grippe*, a word which is said to be derived from the Polish Crypka or Grypka (= *raucedo*). Its history is supposed to date from remote antiquity; but, as might be expected, doubts exist as to the real nature of many of the older epidemics.

During the last four centuries the periods during which influenza has prevailed have been carefully recorded; the late Dr Parkes (in 'Reynolds' System') says that there were eleven in the sixteenth century, sixteen in the seventeenth, and eighteen in the eighteenth. Between 1800 and 1850 there were no fewer than ten, of which three were most important; one, in its spread over different countries, occupied the years 1830 to 1833, another occurred in 1837, and the third in 1847–48. It seems to be doubtful whether there has been any reappearance of the disease, at least as an epidemic, during the second half of this century.

Course.—Most of the symptoms of influenza are those of a common feverish cold; but it is attended with greater pyrexia, and with more severe depression of strength. The patient perhaps suddenly becomes chilly, and shivers, and for some hours he may feel exceedingly ill before any definite local affection appears. Sir Thomas Watson says that when he was first called to two cases on April 3rd, 1833, the symptoms were just those which frequently mark the commencement of an attack of

* *Synonyms.*—Tussis epidemica (Sydenham)—Catarrhus à contagio (Cullen)—Epidemic catarrh (R. C. P. nomenclature),—"the Chinese catarrhal fever."—*Fr.* La grippe.—*Germ.* Grippe.—*Ital.* Influenza.

continued fever, and that he did not then know what was about to happen; but in the course of that and the following day all London was smitten with the disease. Since the introduction of the thermometer into clinical practice no well-marked epidemic has occurred; but it is believed that the fever presents nocturnal exacerbations, and that it is sometimes very high. The pulse is rapid, remarkably weak and small, and sometimes intermittent. After a short interval, or even from the first, the patient begins to sneeze, and a thin acrid fluid runs from his nose; his eyes grow red and watery, and his fauces are reddened. He grows more and more prostrate, so that he cannot keep up, and is altogether unable to perform his customary duties. He may suffer from giddiness or faintness; in some epidemics drowsiness is a very common symptom, in others many patients become delirious. Pains in the limbs or cramps in the calves may be complained of. There is always severe headache, and often an intense pain at the root of the nose, which is attributed to extension of the catarrh to the frontal sinuses. At the same time, or a little later, the patient is attacked with an irritating cough, dry, or attended with scanty expectoration of mucus without froth. Sometimes there is great dyspnœa, with a sensation of distress in the præcordial region, and liability to severe suffocative attacks; the lips and face may even become livid. Loss of appetite is a marked symptom; the tongue is thickly furred; there may be nausea, or vomiting; sometimes diarrhœa is present; the skin may have an icteric tinge. The urine is scanty and high coloured. In pregnant women abortion may occur, as the result (it is said) of the violent cough; or, if the menstrual functions have been suppressed from other causes, they are sometimes re-established. Dr Walshe has observed facial herpes.

At the end of from three to five days the attack passes off. If it lasts much longer, the usual reason is either that pneumonia has developed itself, or that the catarrh of the air-passages has passed into ordinary bronchitis. The subsidence is sometimes gradual, sometimes by a critical sweating or diarrhœa; sometimes it seems to set in with epistaxis. The patient's convalescence is always slow, and he is long in regaining his strength. Relapses, according to some authors, are not infrequent, but Dr Robert Williams observed that few persons suffer more than one attack in the same season. On the other hand, to have had influenza in the course of an epidemic seems to confer no immunity against its recurrence on a subsequent occasion.

In some cases the disease assumes a rudimentary form, the patient merely complaining of a slight coryza, perhaps accompanied with severe sorethroat and cough, and attended by a little malaise, headache, and disinclination for work.

Sir George Baker, in his 'Opuscula Medica' (Second Edition, London, 1871), gives the following account of the epidemic of influenza in the year 1762. It was characterised by alternate heats and chills, by a constant cough, sometimes dry, occasionally accompanied by a little thin mucous expectoration. There was depression of strength, a sense of weight, and severe pain in the forehead and temples; inflamed, swollen, and watery eyes, with photophobia; frequent sneezing and altered voice. There was painful rawness felt in the windpipe and chest, with, in some cases, a feeling of choking, and wandering pains in the arms, legs, and sides. The fever was chiefly nocturnal, but even then so slight that it rarely interfered either with sleep or with food. There was more or less perspiration, and when it was profuse the disorder was relieved or cured. The tongue was white and thickly furred; the urine was dark, and threw down a furfuraceous or

lateritious deposit. In all cases there was more depression of spirits and loss of strength than the character of the disease seemed to account for, and convalescence was often tedious and imperfect. Many cases of abortion or premature birth were observed in London from this disease. In a few of the more severe cases there was a miliary eruption.

Mortality.—It may appear paradoxical to say that influenza is seldom fatal, and yet that it always causes a great increase above the normal death-rate of the town in which it breaks out. But the explanation is that almost all the mortality is brought about indirectly, and that the number of those who fall ill with influenza is greater, beyond all comparison, than in the case of any other disease. In London in 1847 not less than 5000 persons are said to have died of it in six weeks; but then it was computed that 250,000 persons were attacked. In Paris, above one fourth of the population suffered; in Geneva, not less than one third. Those who die are chiefly old and debilitated subjects, who have previously laboured under emphysema of the lungs, or who have feeble and dilated hearts. It is sometimes dangerous to very young children.* Parkes states that patients with lesions of the valves of the heart, and some at least of those who have phthisis, pass through influenza without being the worse for it; but other writers have remarked that after its subsidence phthisis often takes a very rapid course. Dr Farr pointed out, in 1847, that the mortality was much greater in those districts of England in which the death-rate was generally high than it was in healthier places. In February, 1762, the weekly death-rate at Warsaw ran from 30 or 40 to 150. In 1837 the mortality in 2347 cases was 2·3 per cent., in 1847 about 3 per cent. (Peacock).

No special morbid changes are seen in the bodies of those who have succumbed during an attack. The lungs and air-passages are congested, sometimes to an extreme degree, and there may be great œdema of the pulmonary tissue. Pneumonia is not infrequent, but this is regarded as a complication; it may be either catarrhal or fibrinous. Plastic exudation into the bronchial tubes has now and then been found.

Recovery from influenza is sometimes followed by parotitis; and it is also stated that when the patient had previously suffered from neuralgia this affection is apt to return after convalescence from the epidemic disease.

Ætiology.—As to the cause of influenza there have been many speculations, but as yet no positive conclusion has been arrived at. Certain observations which have been made as to the way in which it spreads are, however, of great importance, by suggesting some possible solution of the difficult question of its origin or by negativing others. No other disease diffuses itself equally widely over the earth's surface. Not only is it capable of existing in all inhabited regions, so far as is known, but in some epidemics it has ranged over every quarter of the globe, and has established itself in places presenting all kinds of soil and every variety of climatic conditions. It therefore cannot be attributed to any "telluric emanation" or miasm. The suggestion has sometimes been made that there may be a relation between it and ague, but it is not found to be especially severe in countries like Holland, which are infected with malaria; indeed, Holland is said to have escaped some epidemics which have traversed Europe.

* But Sir George Baker writes of the epidemic in 1762: "*Leviter plectebantur infantes et liberabantur facillime.*" He found it most serious in advanced age, and particularly in asthmatic old men. The elder Dr Babington died of the influenza in 1833. Graves found the epidemic of 1837 very fatal among the aged, yet saw Judge Day recover at ninety-three.

Again, the progress of influenza from district to district occupies time. Many observers have thought that it commonly observes a definite direction, namely, from the East or the North-East towards the West or South-West. Thus, the epidemic which raged in London in 1833, is supposed by Hirsch to have been related to one which occurred in 1830 in China, and which reached Moscow later on in that year. In 1831 it spread over Russia, Poland, Germany, France, Sweden, Italy ; it next appeared in the Isle of Man, and lastly in New Jersey, on the other side of the Atlantic. In 1832 it occurred chiefly in Spain and in some of the United States. In 1833 it broke out again in the north of Europe, and after extending over Russia and Germany, and passing to Denmark, it reached London, as above mentioned, in April. It was also observed at different dates of this year in France, Switzerland, the Tyrol, Italy, and Egypt.

The epidemic of 1762 appeared at Warsaw in February, reached Vienna at the end of March, and Magdeburg in April ; at which time it also invaded Hamburg and London. It was much more severe at Venice and at Warsaw than in England and Germany, and did not visit Paris at all. In June it was epidemic throughout Alsace,* and in July attacked the British fleet in the Mediterranean. After that month no cases were known to occur in Europe. Beside London, the towns of Manchester, Lincoln, Leicester, Exeter, and Norwich were seats of the disease, apparently by conveyance from the capital. Similar epidemics of catarrh or influenza had appeared at Norwich in 1733 and 1743. It was less common in country places—an argument in favour of its being infectious.

It is particularly worthy of notice that both in England and France epidemic influenza seems to occur only as the result of extension from other countries. Some writers have doubted whether it can arise in any part of Europe, and have imagined that it has its home in some remote part of Asia, such as Chinese Tartary, and is always thence derived. Hence the Russian name of the Chinese catarrh.

Influenza often suddenly breaks out at the same time in places far distant from one another, and at once attacks a large proportion of their inhabitants ; and it has been said to appear on exactly corresponding dates on board ships which had been long at sea, and which had sailed from ports where influenza was not prevailing. Thus, in 1782, Admiral Kempenfeldt's squadron sailed from Spithead on May 2nd to cruise between Brest and the Lizard. On the 29th, there having been no communication with the shore, the men who formed the crew of one of the ships were attacked with influenza, and soon afterwards so many of the sailors on the other ships that by the second week in June the whole squadron had to return to port. In the meantime another fleet, under Lord Howe, had sailed, all in perfect health, for the Dutch coast. Towards the end of the month of May the disease appeared in several of his vessels also, although there had been no intercourse with the land. So, again, on April 3rd, 1833, the day on which Sir Thomas Watson saw his first two cases of influenza in London, a vessel called the "Stag" was coming up the Channel and arrived at two o'clock off Berry Head on the Devonshire coast, all on board being well. The breeze was blowing from the land, and in half an hour forty men were down with influenza ; by six o'clock the number was increased to sixty, and by two o'clock on the following day to 120. The very same evening a regiment at Portsmouth was in a perfectly healthy state, but by the next morning so many of the soldiers were affected by the

* These accounts were received by Sir Geo. Baker from Drs Jackwitz, Mertens, Kothen, Pringle, and Reimarus.

disease that the garrison duty could not be performed. Parkes, indeed, expresses some doubts as to whether these instances can be entirely relied upon. But, as he says, if they are not altogether without foundation they effectually disprove all chemical theories as to the cause of influenza. Neither a vapour nor any kind of molecular matter wafted in the air could travel such distances without undergoing dispersion and destruction. Nor is it possible that the morbific agency can be any substance, such as ozone, which, naturally present in the air in small quantities, might conceivably become enormously increased in amount at the same time over a wide area. For the disease, instead of affecting the whole of a town or city, sometimes confines itself to certain districts, or even to particular streets; and it may leave adjacent villages free.

Thus, by a process of exclusion, we are brought to the conviction that, unless the cause of influenza is something of the nature of which we have no conception, it must be a living thing, which is capable of reproducing and multiplying itself when once it has been introduced into a particular district or country. It would seem, however, that there is one very important objection to such a view in the fact that the distribution of the disease has hitherto appeared in no way to be dependent upon any climatic or meteorological conditions. It may prevail at any season, in every climate, and during all kinds of weather. If it has sometimes broken out after a sudden thaw, or just when there has been a heavy fog, such occurrences are to be regarded as mere accidental coincidences; and it has often been epidemic in countries, such as Egypt, where the air is extremely dry as well as hot.

The difficulty involved in the foregoing statements is all the greater because it cannot be supposed that the organisms which give rise to influenza, if organisms there be, undergo multiplication and development anywhere except in the air itself. It is true that, according to Watson, there have been numerous instances in which the complaint has first broken out in those particular houses of a town at which travellers had recently arrived from infected places. And there have also been examples of its having spared the inmates of prisons or convents, as though their isolation had served to protect them. But all that such cases can prove is that the morbific agency is capable of adhering to the human body, or to clothes, or luggage, so as to be conveyed from one place to another; its subsequent growth and development is doubtless altogether independent of this kind of assistance. Cullen, indeed, defined influenza as *catarrhus à contagio*, but if he meant that it is contagious in the sense in which we now use that term, it is tolerably certain that such a view is incorrect. The disease seems to occur as frequently among persons who are confined indoors as among those who go about; it often attacks bedridden people; it does not spread from one patient to another, nor to relatives or nurses of the sick. It is decidedly more common in women than in men; children suffer less than adults, and in some epidemics they seem very generally to escape.

Another point which negatives the idea that an attack of influenza is due to an organism which multiplies within the human body is the absence of a stage of incubation. Parkes cites several instances in which persons who have come from a distance to places infected with influenza have fallen ill on the following day or two days afterwards. Such cases, indeed, are not in themselves conclusive, for they could be matched by exceptional cases of scarlet fever, in which disease the incubation usually lasts several days. But they go very far towards proving the point in question, when they are

taken in conjunction with the fact that at the commencement of an epidemic of influenza an immense number of persons fall ill simultaneously, or nearly so. If the disease took several days to hatch, one may be quite sure that the preliminary morbid process would not thus come to an end in every patient at the same time. Parkes, indeed, alludes to cases "in which the incubation period must have been two or three weeks," but on looking up the reference which he gives to Dr Robert Williams's work on morbid poisons, we find that the only instances given there are those already alluded to, in which the disease appeared on board ship when there had been no communication with the land.

Another circumstance bearing on this question is the fact that epidemics of influenza have almost always a definite duration of from four to six weeks, and that their subsidence is scarcely less sudden than their commencement. It is mentioned, as a solitary exception, that, in 1831, the disease prevailed in Paris for nine or ten months at a stretch. One cannot but see how easily this may be explained on the hypothesis that the pabulum which is required for the maintenance of the organisms rapidly becomes exhausted if such are supposed to exist.

The way to remove all doubt about the matter would of course be to inoculate upon a healthy person the blood of a patient suffering from influenza. In the horse this experiment has already been tried by Hertwig, but has failed. It is, however, a fair question whether the human disease is identical with the influenza to which horses are liable, and which, for example, raged in the United States in 1872, when it is said to have attacked about 16,000 horses in New York alone. During epidemics of influenza among human beings, horses, dogs, cats, and even birds are said to suffer. But when the equine epizootic prevails men seem entirely to escape.

Definition.—During the prevalence of an epidemic of influenza the only point to be mentioned in regard to its diagnosis is the risk of misinterpreting the early stages of other febrile complaints, such as enteric fever or the exanthemata. But is it possible for the disease to occur sporadically? and if so, how can it be distinguished from a non-specific catarrh? These questions are by no means easy to answer. Sir Thomas Watson, indeed, says that "in the years immediately succeeding an epidemic it generally shows itself again, but in a milder and less general form." And in further explanation of his meaning he goes on to say that "many of the colds and bronchial disorders of the seasons which follow a period of genuine influenza are attended with much more languor, debility, muscular aching, and distress, than belong to an ordinary attack of catarrh." Now, in the case of cholera (a disease which from a certain point of view is analogous to influenza) something precisely similar to this seems unquestionably to occur. But the statements just quoted are exceedingly difficult of proof in regard to a complaint of which the symptoms are so little characteristic as are those of influenza. Parkes says that sporadic cases are not met with.

We ought to protest against the practice, so common among the higher classes in this country, of designating as influenza any catarrhal attack that happens to be unusually severe. They think that in this way they give a sort of dignity to what would otherwise be a common complaint hardly worthy of attention; in reality they are debasing a word of which it is important to retain the special significance.* A fair criterion may generally be found

* Yet, as Niemeyer remarks, "Diese Unsitte hat gewissermassen ihr Gutes. An einem Katarralfieber will Niemand erlieblich erkrankt sein und längere Zeit im Bette liegen; hat er aber die Grippe so ist er zufrieden wenn er nur in acht bis zehn Tagen genest."

in the fact that these false influenzas only occur during the damp and cold seasons of the year, whereas, as we have seen, the genuine disease prevails in all weathers.

A further question still remains as to the nature of certain localised forms of catarrh, affecting a greater or less part of the population of particular towns or districts. Thus in 1864 influenza is said to have existed in Switzerland, during the spring of 1867 in Paris, and so recently as 1874 in Cape Breton. As Parkes observes, strict proof that the disease in such "local epidemics" is really influenza ought to be required before they are admitted, but it is difficult to say how the proof is to be obtained.

Treatment.—From a historical point of view it will always be interesting to know that bleeding and the administration of antimony were recognised by universal experience to be injurious in this disease at a time when they were regarded as almost essential to the cure of pneumonia and other inflammations. In one or two of the later epidemics quinine was found useful even from the commencement of the attack, but the most usual practice is, after having given one purgative dose, to prescribe salines during the first day or two, and afterwards ammonia with senega or serpentary. Parkes gives many practical details as to the management of the disease, evidently based upon his own experience; and these would doubtless be found useful if one should hereafter be called to deal with a new epidemic. One point on which he insists is that the common custom of feeding the patient with hot beef-tea is a bad one; it invariably, he says, increases the headache and the languor. As there is complete loss of appetite, and as the attack may be expected to come to an end in a few days, it may be sufficient, if the patient is young and healthy, to let him have such beverages as iced milk and soda-water, barley-water with lemon-juice, or very weak cold white-wine whey. To such persons stimulants should not be given during the early period of the disease. As it begins to pass off, a good supply of food should be allowed, and at this stage tonics are very necessary.

Ozæna.*—A very intractable and distressing form of chronic nasal catarrh is one in which the discharge from the nostrils, and even the air expired through the nose, has constantly a peculiar and disgusting fœtor.

With regard to many points about it there is still much uncertainty. Formerly it was thought to be generally dependent, either upon a scrofulous diathesis, or upon a syphilitic taint, congenital or acquired. There is of course no doubt that disease of bone in the interior of the nose, whether due to syphilis or to any other cause, may produce great fœtor. But the better opinion seems to be that the special odour that characterises ozæna is not generally traceable to such lesions, nor even to ulceration of the mucous membrane.

All recent observers who have made autopsies in cases of ozæna appear to be agreed that in this affection there is an *atrophic* condition of the tissues within the nose, including even the turbinated bones. At one time it was supposed that this was the ultimate stage of a chronic "rhinitis," attended at an earlier period with thickening and swelling of the mucous and submucous structures. There is, however, no evidence of the occurrence of such an antecedent condition. Nor is it clear how the atrophy, when it has developed itself, is related to the fœtor. Some have thought that the

* *Synonyms.*—Chronic atrophic fœtid coryza. Ὄζαινα (from ὄζω, I smell) is applied by Greek writers in the second century to a fœtid polypus in the nose.

current of air through the widened nasal passages, being unduly slow, fails to clear away the mucus which is constantly being secreted, and which therefore dries up and undergoes decomposition. Others hold that the wasted condition of the glands of the mucous membrane leads to an insufficient formation of mucus. In either case it appears probable that the process of desiccation is essential to the production of the offensive odour.

But there can be no doubt that mucus may and often does form dry crusts in the interior of the nose, without any fœtor resulting. The explanation seems to be that for the production of ozæna a special ferment or microbe is required, the presence of which induces a particular kind of putrescence.

So penetrating is the smell in the worst cases of this distressing malady, that it pervades the air for some distance round the patient, and renders him unfit for society. Yet he himself is often quite unconscious of it.

It must not be supposed, however, that the affection is always so severe as this. Fränkel remarks that about some persons, when they first wake in the morning, very faint indications of the ozæna-odour can be plainly recognised, which are altogether absent at other times. And, again, he says that in some cases in which mucus leaves the nose free from smell, it acquires more or less of the characteristic fœtor in drying on the handkerchief. This statement is confirmed by others.

Treatment.—The usual method of treating ozæna has been by systematic irrigation of the nose with water, or with saline solutions, or by injections of antiseptic agents, such as boracic acid or salicylate of soda. Of late, however, a considerable advance seems to have been made by Gottstein, of Breslau, who simply introduces a plug of cotton wool into one nostril, leaving it there for twelve or twenty-four hours, and then withdrawing it, and plugging the other nostril in its turn for a like period. The effect is often successful, so far as concerns the temporary removal of the fœtor. But almost as soon as the treatment is discontinued the case becomes as bad as ever. Ozæna is, in fact, at present incurable.

So serious is the discomfort entailed and so unsatisfactory have milder remedies proved, that more than once surgeons have undertaken the severe operation of opening the nostril and extirpating the turbinated bones. (See on this subject a paper by Mr Warrington Haward in the 'Lancet' for 1877, vol. i, p. 784.)

Epistaxis.*—*Bleeding at the nose.*—Epistaxis occurs as a complication of many maladies. It is apt to accompany all hæmorrhagic diseases, and in splenic leuchæmia it is often an early symptom. It is more frequent in aortic than in mitral affections of the heart. Persons who have granular kidneys with hypertrophy of the heart are exceedingly liable to it, and in them it may often be taken as a warning of the probable supervention of uræmic seizures or of cerebral hæmorrhage. It is also frequent in persons who have cirrhosis of the liver, with more or less jaundice and with dilatation of the small veins of the cheeks. "Bleeding at the nose," wrote the elder Heberden, "is a usual attendant upon the diseases of the liver in hard drinkers." It may accompany some of the acute infective diseases, especially variola and enteric fever. In cases of ague not only is it sometimes associated with the ordinary paroxysms of the disease, but it is said to have occurred periodically as the sole symptom and effect of malarial poisoning, until stopped by the administration of quinine.

* This form seems to be a corruption of ἐπίστασις sc. αἵματος, a staunching of blood.

But in the majority of cases, bleeding at the nose is due to none of these causes. Boys towards the age of puberty are very subject to it spontaneously from time to time; but at any moment, a slight blow upon the face, touching the nasal mucous membrane, or forcibly blowing the nose, may bring it on. In the 'Lancet' for 1865 Dr Guy Babington gave the history of a family in which marked liability to habitual and violent epistaxis was traced through five generations, and in thirteen out of twenty or more individuals. Some of those who suffer from recurrent or "habitual" epistaxis are pallid, with apparently little blood to spare. But others are plethoric, with flushed cheeks and injected conjunctivæ; and they may experience from time to time sensations of oppression and giddiness, noises in the ears, throbbing in the head, fulness and heat in the nose, which they recognise as indications of the approach of an attack of hæmorrhage, and which are at once relieved when it occurs.

In some young women, epistaxis has been observed to be distinctly vicarious of the catamenia. A striking instance of this is one recorded by Obermeier in vol. liv of 'Virchow's Archiv.' A girl of fifteen, after once menstruating in the ordinary way, began to suffer at regular intervals of a month from bleeding at the nose; this occurred two or three times a day for three successive days, and it was attended with malaise and other symptoms like those which had accompanied the natural flux. She became pregnant and the hæmorrhage then ceased, to return six weeks after her delivery. No doubt caution is required in accepting the statements of patients as to any form of vicarious menstruation; but this case appears to be beyond question.

In epistaxis the blood almost always comes from one side of the nose only; but sometimes part of it passes round behind the nasal septum and ultimately escapes from the opposite nostril, so that both sides may appear to be bleeding simultaneously. It may either flow in drops or in a more or less continuous stream; the quantity lost is in some cases very great. It is commonly of a bright red colour, this being perhaps due to exposure to the air after it has escaped from the vessels. It forms a solid coagulum. One can very seldom discover, whether by rhinoscopy or by inspection through the nostril, the exact point from which the oozing of blood takes place; nor have pathological anatomists as yet ascertained whether in cases of habitual epistaxis the veins in the submucous tissue of the nose (which are normally very wide and numerous) are in a varicose condition or affected with any degenerative change. In many cases a part of the blood escapes through the posterior nares into the pharynx and is swallowed. Indeed, if the patient should happen to be asleep or recumbent from any cause, the whole of it may take this course; and when a large quantity of blood is subsequently ejected from the stomach by vomiting, the real seat of the hæmorrhage may be altogether overlooked unless a careful examination of the nose is made, which will almost always reveal the presence of clots in one or the other of the nostrils or in the pharynx.

The ordinary course of epistaxis is to cease spontaneously sooner or later. But it sometimes continues for many hours, or even for days, without intermission. In such cases the patient may rapidly pass into a condition of extreme anæmia, and may possibly die of syncope; and in other cases the frequent repetition of attacks of bleeding at the nose brings about a chronic state of bloodlessness that itself tends to favour the occurrence of further hæmorrhage. The process by which the natural arrest of epistaxis is

effected appears to be by the formation of coagula, which adhere firmly to the mucous membrane and close up the vessels. Hence, after the cessation of an attack any disturbance of the parts may at once cause a return of bleeding. Fränkel, in 'Ziemssen's Handbuch,' remarks that obstruction of the free part of the nose by clots, with apparent cessation of the epistaxis, affords no guarantee that oozing into the pharynx may not still continue, the patient swallowing the blood without knowing it; in a case of enteric fever, for example, great danger may arise in this way.

Epistaxis should not always be actively treated; as already observed, it may give relief to other symptoms of which the patient had been complaining, and it then generally ceases of its own accord. On the other hand, when anæmia is beginning to result, one must be careful not to delay too long in carrying out whatever measures may be requisite. Sometimes compression of the side of the nose against the septum by the finger placed just below the nasal bone is sufficient to arrest the flow of blood, at least for the time. It is important to notice whether this is the case: for, if so, one can be sure that the bleeding spot is in the fore part of the nasal cavity, and that one can stop all further hæmorrhage by the "anterior tamponnade," that is by systematic plugging with a long strip of lint introduced through the nostril. But before adopting this method of treatment it is as well to make trial of astringents, such as gallic or tannic acid, which may be sniffed up in the form of a powder. To inject cold water or a solution of alum or any other astringent liquid is less advisable, because it tends to disturb any clots that may have formed. Cold may be applied to the outside of the nose as well as to the whole of the patient's neck and chest. At the same time he may have his feet placed in a hot mustard bath. He should sit upright with his head slightly bent forward, so as to prevent the blood passing backwards towards the pharynx, and should keep the hands raised above the head.

But if the hæmorrhage should continue, and if the source of it should appear to be from the back part of the nasal cavity, one must not wait long before having recourse to the radical method of the "posterior tamponnade," or "plugging the posterior nares." This is generally effected by means of an instrument which is described in every surgical work and which is known as a "Belloc's cannula;" the plug being a piece of folded lint of suitable size and shape. It has, however, its disadvantages; for if the lint is not left undisturbed for some days, epistaxis is apt to begin again as soon as it is interfered with; while, on the other hand, if it be allowed to remain long *in situ*, there is often great difficulty in loosening it, and the mucous membrane may be torn. In any case it should certainly never be left to become fœtid by putrefaction of the blood and other fluids which soak into it. A better method is to fill up the nasal cavity with an elastic pouch, which having been introduced in a collapsed state, is afterwards inflated with air until it exerts considerable pressure in every direction.

HAY FEVER.*—Certain persons are liable every summer to a very troublesome affection, which sometimes assumes the characters of catarrh, sometimes those of asthma, and therefore may find its place provisionally

* *Synonyms.*—Hay asthma—Periodic vaso-motor coryza—Specific or nervous coryza—Catarrhus æstivus—Catarrhus ex fœnisicio—Rose-cold and autumnal catarrh (U.S.A.).—*Fr.* Fièvre asthme ou catarrhe, de foine, asthme d'été.—*Germ.* Heu-asthma, Heufieber, Bostock'sher Katarrh.

between coryza, influenza, and spasmodic asthma. Attention was first directed to it by Dr Bostock in a paper read before the Royal Medical and Chirurgical Society in 1819.

Symptoms.—The *asthmatic* form will be considered separately (p. 296). The *catarrhal* form sets in with a feeling of irritation in the nose, throat, and eyes. Then the patient begins to sneeze—perhaps twenty or thirty times in succession. A thin watery secretion pours from his nostrils. The nasal submucous tissue rapidly swells, until in a little while no air can be drawn through the nose. If, however, he lies down and turns on his side, the nostril which is now uppermost becomes in a short time free (apparently as the result of gravitation of the œdematous fluid), while the other one becomes more occluded than before. The swelling affects the lachrymal passages also, so that the tears run down over the cheeks. The eyes become inflamed, and there may be œdema of the eyelids. So distressing are these symptoms that it is almost impossible for the patient to avoid giving way to them and suspending his usual occupations. From day to day they vary in severity, but they commonly last three or four weeks, or even longer. Paroxysms of sneezing continue to recur from time to time. The nasal discharge presently becomes thicker and more purulent, or it may be stained with blood. Ultimately the affection passes off, leaving more or less weakness and prostration behind it.

Exciting cause.—One of the first points observed about this "summer catarrh" was that its onset often corresponded closely with the beginning of the hay season, and that persons were attacked immediately after being in or close to a hayfield. But it was not until 1873 that Dr Blackley, of Manchester, observed by careful observations and experiments upon himself that the true cause of the affection is the diffusion in the air of the pollen of certain plants, and especially of grasses, which settles upon the mucous membrane of the nose and eyes, and acts as a local irritant. Up to that time some medical writers had attributed hay fever to coumarin,* others to ozone, others to "common dust," others to the mere heat of early summer. Dr Blackley found that by introducing a small quantity of pollen into the nostrils he could bring on all the symptoms of the disease almost instantaneously. During the summers of 1866 and 1867 he made daily observations upon the amount of pollen which was deposited upon glass slides moistened with glycerine and exposed to the air; and he found that there was in general a close relation between the quantities collected and the severity of the symptoms of hay fever under which he laboured from day to day. Dr Blackley appears to have accounted satisfactorily for certain observations which had seemed to Dr Bostock and others to prove that the supposed connection between summer catarrh and the emanations from flowering plants was a mistake. At any rate, he showed that when one might imagine that there can be no pollen in the air, it really may be present in abundance. Once he was suddenly seized while on the shore, with a sea breeze blowing; between him and the sea there was but a narrow belt of land, but upon this he found a field of wheat in full bloom. Another time an attack was brought on by the dust of an unfrequented country lane; it was summer time, and on examining the superficial layer of dust with the microscope he discovered that it was full of the pollen grains of grasses.

Predisposing causes.—Hay fever most commonly appears for the first time

* Or coumaric anhydride ($C_9H_6O_2$), an aromatic compound belonging to the Benzene group. It gives its scent to the grass *Anthozantum odoratum*, and to *Asperula odorata*.

about the age of puberty, but sometimes it is observed in children four or five years old. Persons who have reached the age of forty without being affected with it are probably never attacked afterwards. It is more common in males than in females.

One very curious circumstance about this disease is that those who suffer from it appear always to belong to the educated classes. It is never seen among gardeners or farm labourers, who are of course more exposed to the influence of pollen than any other set of men. This fact has led to the suggestion that the individual predisposition to the disease, which plays so important a part in its ætiology, is perhaps a result of an indoors life, especially in towns or cities. Should that be the case, one can understand how it is that hay fever appears to have become so much more common of late years than in the earlier part of the present century. In addition to the personal peculiarity, which we may call an idiosyncrasy or neurotic disposition or diathesis, Sir Andrew Clark believes that there is a local peculiarity in the nasal mucous membrane of those subject to this curious malady, which leads it readily to swell and become vascular.

It is supposed to prevail in England much more than on the Continent, but it is also very common in the United States, where it has been carefully investigated by Dr Mackenzie, of Baltimore. In most cases the susceptibility to the disease appears to increase with each successive year. At first the patient may be attacked only when he is actually in a meadow where the grass is in full bloom ; ultimately he suffers as soon as he attempts to go into the country during the hay season. Sometimes hay fever begins to assume the asthmatic form only after it has for several years recurred as a catarrh.

The *treatment* of hay fever is extremely unsatisfactory. Neither quinine, nor arsenic, nor any other medicine appears to have the power of enabling those who are liable to the disease to bear exposure to its exciting cause without being at once attacked by it. The local application of quinine in solution was first used in his own case by Professor Helmholtz, but it has not proved so successful with meaner sufferers. Sir Andrew Clark (after trying and discarding every kind of internal remedy, including aconite and belladonna), finds that a solution of cucaine (5-15 per cent.) applied locally with a brush, or as a spray or a bougie, is sometimes successful, though it sometimes fails. As a more radical plan, and one which he estimates to have been effectual in about a third of the cases treated, the same physician recommends the thorough local application of a solution of carbolic acid to the interior of the nostril (see the formula and method described in the 'Brit. Med. Journ.,' June 11th, 1887, p. 1256).

The most thoroughgoing treatment of all is to destroy the secretive parts of the Schneiderian membrane by caustics or galvano-cautery. This severe treatment has been carried out in the United States, and it is said with complete and permanent success.

For those who suffer severely, the only course (apart from local treatment) is to remain in a large town through the whole of the summer months, or else to go to the seaside, choosing some narrow peninsula or island, or to take a sea voyage. Staying indoors during the middle of the day, even in a country house, often does much to mitigate the symptoms. The continuance of hay fever during several weeks—generally from some time in May until the middle of July—is dependent upon the continued exposure of the patient to the exciting cause of the disease. If he can get away to a place where there is no pollen in the air, the attack quickly passes off.

When the symptoms are first coming on considerable relief is afforded by the use of a smelling-bottle containing ammonia, iodine, and carbolic acid, made into a paste with wood-charcoal and compound tincture of benzoin.

WHOOPING-COUGH.*—Like so many other epidemic diseases, whooping-cough can be certainly dated back only to a comparatively recent period—the earliest notice of it is said to have been by Schenck in the year 1600. It is a very frequent and widespread disease, and, next to scarlatina, more fatal than any other in childhood; indeed, for infants under one year it is probably the most fatal.

Clinical course.—The *incubation* of whooping-cough after infection is perhaps variable—probably a fortnight as a rule, but often less.

During the early period of the disease no symptoms whatever are present by which its nature can be suspected, unless, indeed, other cases have recently occurred in the same family or in the same neighbourhood. This *prodromal stage*, as it is sometimes called, is characterised by the occurrence of an ordinary bronchial catarrh, with a more or less troublesome cough and some pyrexia. Sometimes there is also nasal catarrh, with running at the nose and sneezing. The child (for whooping-cough chiefly affects children) is pale, out of sorts, and restless. A point on which Trousseau lays stress is that the cough is sometimes remarkably frequent, recurring fifteen, twenty, or thirty times in the minute; in such cases there is generally high fever. He speaks of having been able to diagnose the real nature of the disease at this period by the incessant repetition of the cough. The duration of the prodromal stage is very uncertain. Sometimes in young children it lasts only a day or two, or it may perhaps be entirely absent. Sometimes it runs on for two, four, or even five weeks. In some cases it is believed to constitute the whole of the disease, the patient recovering without having any more distinctive symptoms; this possibility was suggested by Cullen, and it has been recently supported by Dr R. J. Lee and Dr Eustace Smith.

In all but these somewhat doubtful exceptions, a second stage succeeds, characterised by the development of the "whoop" with the cough. The change in question may occur either suddenly or very gradually, so that the boundary-line between the two stages is often not to be fixed with any certainty. The cough may on some one or two occasions be attended with a sound which raises the suspicion of an experienced nurse or visitor, but several days may afterwards pass before any confirmation of their suspicion is to be obtained. Or, on the other hand, in a case which is not really one of whooping-cough, doubts may arise; for some healthy children, especially if they happen to cough while crying, make a kind of crowing or whooping noise that is not very different.

But when whooping-cough is well marked, there can be no mistake about it. Pyrexia is now absent. There may be a good appetite, and except for the cough the child may appear to be well. The cough itself comes on in paroxysms, of which there may be only a few in the twenty-four hours, or as many as sixty or eighty, or even more. They are usually more frequently repeated in the night than during the day. Each begins with a series of

* *Synonyms.*—Chin-cough—Tussis clangosa (Glisson)—Pertussis infantum (Sydenham, 'Proc. Integr.,' cap. xliv, 1695)—Tussis puerorum convulsiva (Sydenham, 'Obs. Med.,' iv, 5, § 8, 1685; also Heberden and Cullen). *Fr.* Coqueluche, Touxquihoupe (whence "hooping" cough).—*Germ.* Keuchhusten.

short explosions succeeding one another rapidly, and of course consisting of as many expiratory efforts, but with no noticeable inspiratory movements between them; then, after the air in the lungs has been reduced to a very small amount, there occurs a long-drawn inspiration, attended with a loud whooping or crowing sound that gives the name to the disease. It, in its turn, may be followed by a repetition of the short explosions, and they by the whoop, and such a succession of attacks may continue over a period of several minutes. They end generally by the expectoration of a viscid mucoid fluid, and sometimes by the ejection of the contents of the stomach.

It may well be imagined that the paroxysms of whooping-cough, when severe, cause great distress. The child, when it feels one coming on, runs to its nurse or to its mother for support, or it clings to a chair or to a table so as to diminish as much as possible the shock which affects its whole frame. Patients old enough to take notice speak of experiencing a tickling sensation in the larynx, as if there were something there; or a feeling of compression about the throat, as though the air could not pass freely. Those who are younger betray the approach of an attack by restlessness and by anxiety of face.

As the paroxysms continue, the child may become intensely cyanotic. The eyes protrude, the face and the neck become swollen, and a cold sweat breaks out. Steffen (in 'Ziemssen's Handbuch') says that albumen sometimes appears in the urine. *Hæmorrhages* are by no means infrequent; the expectoration may be stained bright red by blood (which probably comes from the fauces or from the larynx), the nose may bleed, one or both of the conjunctivæ may become ecchymosed, the tears even may be mixed with blood, or the tympanic membrane may be ruptured, with the escape of blood from the ear on one side or on both. Steffen says that a momentary stoppage of the heart is not uncommon, and cites a case, in a girl six years old, in which there was a temporary loss of sight during each attack. He also speaks of a boy, nine years of age, in whom, when severe paroxysms occurred, he noticed an internal squint of the right eye, whereas the left eye looked straight forward, being held fixed with tonic spasm. Sometimes spasmodic movements of the muscles of the face occur. General *convulsions* are not of very infrequent occurrence, and they often prove fatal.

After the subsidence of the paroxysm, the child may be out of breath for a time and may be glad to lie down; sometimes it complains of headache, which may continue all day long; it may be dull and apathetic or fretful. But in many cases it almost at once begins to play again and seems as gay and lively as though nothing had happened. When there has been vomiting it often asks for food and eats it greedily.

Sequelæ.—Among the more remote effects of the violence of the cough is the formation of one or more shallow whitish ulcers on the under surface of the tongue by the side of the frænum; they appear to be caused by the lower teeth, against which the tongue is forced outwards during the paroxysm. In this country Dr Thomas Morton first drew attention to these ulcers in a paper read before the Harveian Society in 1876; but on the Continent they had been previously described by Bouchard and others. Dr Morton detected them in about 40 per cent. of his cases, generally between the third and the fifth week. He once saw an ulcer in an infant who had no teeth, when the tongue may possibly have been injured by the edge of the gums. The recognition of these sublingual ulcers may sometimes be useful in diagnosis.

Ascending paralysis has been once or twice observed after whooping-cough.

But bronchitis and bronchopneumonia are the most frequent and most important sequelæ. They are usually combined with lobular collapse.

Another result of whooping-cough is the production of pulmonary emphysema, and even the extravasation of air into the interlobular and subpleural areolar tissue, whence in some rare cases it reaches the mediastinum and may ultimately diffuse itself into the subcutaneous connective tissue of the chest, throat, and limbs. Or a pulmonary alveolus may rupture so that pneumothorax results. Either of these affections may cause the disease to end fatally.

Prognosis.—It very seldom happens that a paroxysm of whooping-cough directly destroys life. A very young child, however, may die as the result of a complete closure of the glottis, or perhaps from syncope, or from the rupture of an intracranial blood-vessel. When the attacks are very violent and follow one another with extreme frequency, they sometimes give rise to a condition of apathy and stupor which is attributed to the effusion of serum upon the brain and its membranes and which may terminate in death.

Much more often fatal are the pulmonary complications of the disease. Of these the chief are bronchitis and broncho-pneumonia; it is said that they kill half or two thirds of all the children who are attacked by them. As may easily be imagined, patients who before were weakly and delicate are much more likely to succumb to whooping-cough than the strong and healthy. It is infinitely more dangerous among the poor than among the rich. There are, however, differences in different epidemics as regards its severity. It is far more serious in its consequences during the cold seasons of the year than in the summer. When it affects adult patients it is very distressing, but it is not dangerous nor is its duration generally very long.

As a rule, even in children, the tendency of whooping-cough is, after a variable period, to subside and to terminate in recovery. The expectoration which ends the paroxysms becomes looser, more abundant, and more puriform. The violence of the cough lessens and the characteristic whoop disappears. Perhaps the length of time during which it continues to be heard may be altogether six weeks or two or three months. In a case related by Trousseau its duration was altogether only three days; the patient was a child three years old, an inmate of the Necker Hospital, where the disease was epidemic at the time. After the paroxysms have ceased, symptoms of ordinary bronchial catarrh may remain for a time. The child may be a long while in regaining its appetite and strength, especially if the season happens to be winter, so that there is a difficulty in getting it out of doors and into the fresh air. Sometimes, when the whoop has apparently passed off, a fresh attack of catarrh brings it back again for a few days in as marked a form as ever. Even after the lapse of a year it may be noticed that the cough arising out of a simple cold is attended with a somewhat similar sound.

In some cases, even when the paroxysmal stage of whooping-cough has completely passed off, the child nevertheless fails to recover its strength, and ultimately dies of marasmus. Not infrequently pulmonary phthisis, with caseous bronchial glands, develops itself as a kind of sequela. Permanent deafness and otorrhœa are said to be occasional results of the injury sustained by the tympanic membrane during the paroxysms.

Pathology.—There is still considerable uncertainty with regard to the nature of whooping-cough. That its proper place is among the infective diseases is suggested not only by its marked contagiousness, but also by the fact that those persons who have once had it are protected against future

attacks. Indeed, Steffen says that its occurrence for the second time in the same individual, although not absolutely impossible, is infinitely more rare than that of scarlet fever, smallpox, or any other exanthem.

It is said to be one peculiarity of the contagion of whooping-cough that it is far less apt than most other contagia to be transmitted to a distance in an active state. At the Evelina Hospital for Children the whooping-cough ward is allowed to be separated by a short passage only from other wards on the same floor, whereas the wards for cases of measles and scarlet fever are isolated in a different building. Steffen remarks that he has never met with an instance of the conveyance of the contagion of whooping-cough by persons not themselves affected with the disease. However, one can hardly doubt that it might be spread by the use of handkerchiefs or towels contaminated by dried secretions from the air-passages of patients; and a case is recorded in which linen sent to be washed on some island from a ship, on board which there were children affected with whooping-cough, conveyed it to the inhabitants of the island. In vol. xi of the Clinical Society's 'Transactions' there is recorded an observation, made by Dr Bristowe, of a case in which a lady appeared clearly to have conveyed the contagion of the disease from Sydenham to London upon her dress. During a visit to the former place a boy affected with whooping-cough was climbing upon her knee and coughing and sneezing over her; she returned home the same evening, and early next morning one of her children was found playing over her dress, which had been laid upon an ottoman. This girl took the disease and afterwards gave it to two other children. A further point of interest is that the boy himself had only begun to have a constant troublesome cough on the very day on which the lady visited him; in fact, he was staying away from home in the hope that he might escape the disease, which was prevailing among his brothers and sisters. The case is also important as tending to show that the period of incubation in whooping-cough is about a fortnight; for the girl fell ill exactly thirteen days after she was exposed to the contagion, and the two other children sickened after about the same interval.

Climate does not appear to have much influence upon the prevalence of the disease, except that perhaps cold and damp countries are more favourable to it. And Hirsch has shown that it is not more apt to be epidemic at one season of the year than another. There are doubtless great individual differences as regards susceptibility to the contagion.

Female children are decidedly more liable to be attacked than males. The age at which whooping-cough is most common is between the first year and the eighth. Of Dr Goodhart's 314 cases, 62 were under a year old, 212 were between one and four, 65 between four and six, and 13 between six and ten. Barthez and Rilliet recorded the case of an infant whose mother had had the disease for three weeks before its birth, and in whom severe paroxysms occurred on the second day. Sir Thomas Watson relates in his lectures how the grandchild of his bedmaker at Cambridge whooped on the very day of birth, there having been another child affected with the disease in the same house for three weeks before. Dr Eustace Smith found more than a fourth of a series of cases occurred in children under one year old. On the other hand, whooping-cough is sometimes observed in adults up to forty or fifty or even a still greater age.* Heberden met with one case in a woman aged seventy and another in a man aged eighty.

* An eminent London physician suffered severely from an attack of whooping-cough when more than sixty-five years of age.

An association is often traceable between epidemics of measles and those of whooping-cough, children falling ill with the latter disease soon after having passed through the former. Such cases are peculiarly apt to be accompanied with severe broncho-pneumonia and to have a fatal termination, the exanthem having often already brought the lungs into a morbid condition. Sometimes, however, the relation between the two diseases is reversed, measles breaking out after whooping-cough has existed for some time. It is then sometimes noticed that the paroxysms become much less frequent and much less severe than before, and that they remain so as long as fever persists; but, indeed, the same thing may happen when pyrexia develops itself as a mere result of pulmonary or bronchial inflammation in cases of whooping-cough not otherwise complicated.

As already remarked, whooping-cough differs in one very important feature from infective diseases in general, namely, in not being attended with pyrexia at the time when its more characteristic symptoms are manifested. There is, however, a parallel instance in hydrophobia, and it is not impossible that Pasteur's discovery of the actual presence of the virus of hydrophobia in the nervous centres, and of its multiplication there, may hereafter be found to supply the key to the pathology of whooping-cough. One can easily imagine that the poison of this disease, having originally entered the air-passages from without, and having set up a catarrh there, is during the prodromal stage conveyed to some part of the central nervous system and there sets up the peculiar spasmodic cough.

None of the theories propounded hitherto really throw light upon this disease. Some have regarded it as a pure neurosis; some have attributed it to pressure upon the vagus nerve by swollen tracheal or bronchial glands; some have maintained that it is a mere catarrh of the respiratory mucous membrane. Beare, on the ground that the paroxysms of whooping-cough resemble those that follow the entrance of a foreign body into the larynx, would have it that the fundamental lesion is an inflammation of the tract immediately above the vocal cords. Observers differ as to whether or not reddening of the laryngeal mucous membrane can be seen with the laryngoscope during life; according to Rossbach ('Berl. klin. Woch.,' 1880) no morbid change is discoverable, either in the larynx or in the upper part of the trachea; according to Meyer-Hüni ('Ztsch. f. klin. Med.,' 1880) there is marked reddening, and even slight swelling, of most parts of the larynx (but not of the cords), as well as of the trachea.

Recent observations render it probable that the contagious principle of whooping-cough is an organism analogous to those which produce so many other infective diseases, and that it has already been seen with the microscope. As far back as 1870 Letzerich described and figured in 'Virchow's Archiv' a whooping-cough fungus, consisting of thallus-filaments as well as of spores. This is found abundantly in the sputa of patients affected with the disease, and he asserted that he had succeeded in cultivating it, and also in producing a like malady in rabbits by inoculating the trachea of these animals with the product of his cultivation experiments. In the 'Jahrbuch der Kinderheilkunde' for 1876 Tschamer supported Letzerich's views, and also maintained that an identical fungus, commonly found adhering to the surfaces of oranges or apples, is capable of giving rise to whooping-cough when inhaled into the human air-passages. More recently, however, Burger, in the 'Berliner klin. Wochenschrift' for 1883, has cast doubt on the

accuracy of Letzerich's observations, while at the same time he has asserted that the sputum in whooping-cough always contains large quantities of bacteria, which appear as rods of oval form, sometimes constricted in the centre, which are generally scattered quite irregularly, but occasionally arranged in chains. They are said to be easily brought into view by staining with fuchsin or methyl violet.

Treatment.—We have no specific or effectual treatment of whooping-cough. Sydenham depended on venæsection; Fothergill advocated nauseating doses of antimony, and many less probable modes of treatment have been from time to time introduced, to be in turn forgotten.

It is of great importance that the patient should be kept in a spacious room, warm and equable in its temperature; for exposure to cold air has a marked tendency to bring on the paroxysms, and according to Hauke the presence of an excess of carbonic acid in the air has a like effect. In genial weather it is often right to allow a walk out of doors in the middle of the day. When the disease lingers in its course, nothing is so likely to bring it to an end as change of air, especially to the seaside. The food should be nourishing but simple, and should be given directly after each paroxysm. Talking, crying, and excitement of every kind, should as much as possible be avoided.

As regards drugs, *belladonna*, hydrocyanic acid, chloral, bromide of potassium or ammonium, hemlock, appear each of them to diminish the frequency and the severity of the paroxysms in some cases, and even to shorten the duration of the disease. But not one of these medicines can be depended on to produce a definite effect in all, or even in almost all, cases; they often fail altogether. Dr Eustace Smith strongly recommends sulphate of zinc (gr. $\frac{1}{6}$th) and solution of atropine (B.P.) ♏ss, gradually increased in amount and frequency. Alum was introduced by the late Dr Golding Bird as an astringent in the later stages of the disease, and has often proved useful. Quinine also is sometimes very serviceable; there are doubts as to whether it acts by diminishing the excitability of the nervous centres, or by checking the growth of the specific organisms on which the disease is believed to depend. The latter view has led to its administration by insufflation into the air-passages.

In fact, it has lately become a common practice to treat whooping-cough by inhalations. The earliest attempts of this kind consisted in placing patients in the purifying chambers of gasworks, where the air is laden with tarry products, as well as with sulphuretted hydrogen and ammonia. According to Roger ('Bull. de l'Acad.,' 1880) the evidence of the value of this mode of treatment is by no means conclusive.

A plan more recently suggested is that of impregnating the air of the patient's chamber with turpentine, or with petroleum, or with carbolic acid. A solution of carbolic acid, for example, is diffused through the room by means of a spray apparatus, or by simply heating a vessel containing it. Children who are old enough may be made to inhale a weak carbolic spray for fifteen or twenty minutes two or three times a day. Thorner, in the 'Deutsches Archiv' for 1878, reports very favourably of this practice; for about a week there was little or no change; but at the end of that time the symptoms of the disease began rapidly to subside. Successful results by similar means are reported in this country. Inhalations of the vapour from a boiling solution of salicylic acid (of the strength of 2 per cent.) have also been recommended. Dr Goodhart has found both carbolic and salicylic acids disappointing.

Dr Marshall Hall's suggestion of protecting the infant at night from draughts by a mosquito curtain has been tried with success.

ASTHMA.*—Until the present century the word asthma was used to mean what we now call dyspnœa; hence the phrase "cardiac asthma" was often used, and even at the present day it is common to hear persons spoken of as "asthmatic," who are suffering merely from bronchitis and emphysema. Indeed, soon after the discovery of auscultation Rostan and some other French physicians were strongly disposed to deny the existence of any disease deserving to be distinguished by this name, and occurring in persons with healthy lungs. But that there is such a disease there can be no doubt; and now that it has been separated from other affections with which it used to be confounded, its characters are found to be exceedingly definite and well marked. As for its nature, it is commonly believed to depend upon spasm of the smallest air-tubes. We shall have to consider presently whether this is an entirely satisfactory theory with regard to it.

Symptoms of an attack.—Asthma is, in the first place, a paroxysmal affection. It sets in generally with remarkable suddenness; most frequently in the middle of the night, between 2 and 4 a.m., but in some cases at other times, between 6 and 8 a.m., or in the afternoon. The forenoon is almost always the period in the day when the patient is freest from it. In the same case it commonly begins at about the same hour. When this is, as usual, between two and four in the morning, the patient, who may have gone to sleep in perfect health, wakes up with a sense of oppression of the chest which soon passes on to the most extreme distress of breathing. But sometimes the seizure is preceded by symptoms which previous experience enables him to recognise as premonitory; among them are a peculiar drowsiness, flatulence, a slight degree of sneezing, a troublesome itching under the chin, the passing of a quantity of pale limpid urine like that which is secreted in hysteria and other nervous diseases. However this may be, the urgent dyspnœa which now attacks him compels him to sit up, and perhaps drives him to the window, which he throws wide open, in the hope of getting air more freely. Or he may be obliged to sit with his elbows planted upon a table, or to stand with his hands grasping the mantelpiece or some article of furniture above his head; such attitudes being adopted for the purpose of fixing the shoulders and so assisting the muscles of forced respiration in their action. His face becomes livid or purple, his eyeballs start from their sockets, his hands and feet are cold, his skin is covered with a profuse sweat, and his expression indicates extreme anxiety. In fact, he may appear to be at the point of death. There is sometimes a preliminary sensation, paræsthesia of various kinds, which Romberg happily called an asthmatic aura.

Examination of the chest shows that the physical conditions are as follows:—The breathing is not accelerated but of normal frequency, or even slower than natural. Its rhythm is perverted, the inspiration being short, whereas the expiration is greatly prolonged. With the inspiration there may be some wheezing, but this is nothing in comparison with that which accompanies the expiration, and which is audible all over the room. The shape of the chest is such as corresponds with a very deep inspiration; the upper ribs are raised to the fullest possible extent, and the diaphragm has

* Ἄσθμα (ἀσθμαίνω), *panting*, is a Homeric as well as a Hippocratic word.

descended towards the abdomen, so that the area of pulmonary resonance extends considerably lower than natural. During inspiration the sterno-mastoidei and the scaleni are brought into action, but there is scarcely any advance in the degree of expansion; during expiration there is but little recession, although the rigid abdominal muscles can be seen and felt to be doing their utmost to expel air from the lungs. Percussion shows much less than the natural amount of difference in the relation of the edges of the lungs to the heart and liver during inspiration and expiration. The percussion-note over the chest is over-resonant. On auscultation, the inspiratory vesicular murmur is found to be almost or quite inaudible; more frequently sibilus is heard in its stead, or sonorous rhonchi. With expiration there is heard through the stethoscope the same loud wheezing sound which has been already mentioned as being audible at a distance.

So entirely occupied is the patient with the mere act of breathing that he can scarcely utter a word, or turn his head to one side, or even stop to cough; but after a time a slight cough comes on, leading to the expectoration of a few greyish-white pellets of mucus about as large as peas. Not infrequently the mucus is stained with blood, and sometimes there is considerable hæmoptysis. The occurrence of expectoration generally indicates that the symptoms are about to subside. The duration of a paroxysm of asthma is very variable; usually it lasts from one to three hours, sometimes only a quarter of an hour, and occasionally with but slight remissions for a whole night or a day. As it passes off, the patient falls asleep, and when he wakes in the morning his breathing may be quite easy and unattended with any discomfort. The temperature is not raised. The urine after a fit is, according to Ringer, deficient in both urea and salines. But sometimes the disease continues for several days in succession with scarcely any abatement, except that there is almost always some increase in its severity at night, and some lessening during the early part of the day. In such cases the patient's condition causes extreme alarm, although a fatal termination scarcely ever occurs. One instance in which the breathing actually ceased and life was maintained only by artificial respiration was mentioned in the first volume (p. 10).

When asthma passes off in the usual way it is apt to return during the following night. The paroxysms may, in fact, recur for several successive nights, and may then cease, leaving the patient entirely free for weeks or months together, but there are other cases in which the disease shows itself night after night for years. A friend of the author's who was liable to asthma, for the last twenty-five or thirty years of his life, was never able to lie down to sleep; when night came on he dressed himself in a flannel suit and seated himself in a large chair, and here he remained till the morning.

Diagnosis.—From the above description of asthma it will be apparent that the disease can rarely be mistaken for any other by a careful observer; certainly it ought never to be confounded with those tracheal or laryngeal affections (such as bilateral paralysis of the abductors of the cords) in which the dyspnœa is mainly inspiratory.

But it is often only by the history that one can tell whether a patient is suffering from asthma or from bronchitis and emphysema. And, according to Trousseau, it sometimes happens that a child (or even an adult) is seized with what appears to be an acute and dangerous attack of broncho-pneumonia, with abundant moist sounds over the chest; and yet the rapid subsidence of the symptoms in the course of a day or two, and the recurrence of like

attacks on future occasions, ultimately justify the conclusion that the affection is really asthma. The relations of bronchitis and emphysema to asthma are somewhat complicated. On the one hand, it is not uncommon for patients who have chronic bronchitis to suffer from time to time from paroxysms of dyspnœa, which cannot be accounted for by any increase of the bronchial inflammation, and which seem referable only to a concomitant spasm of the air-tubes, and if such spasm is regarded as constituting the essential condition in asthma, it may be fairly said that in these cases *secondary* asthma is really present as a complication of the bronchitis. A "bronchitic asthma" was, in fact, formerly recognised by Salter and other writers; one peculiarity of it is that it is constantly worse in the winter than in the summer, which is not generally the case when asthma occurs as a primary affection.

On the other hand, if a person with perfectly healthy thoracic viscera becomes subject to frequently recurring attacks of asthma, his lungs always become sooner or later emphysematous. We have seen that during the paroxysm of asthma the ribs are raised and that the diaphragm is placed at a lower level than natural. In other words the lungs are in a state of over-distension. Now, when the symptoms quickly pass off, as is usually the case, the chest walls return in a few hours to their normal position and the lungs to their normal size. But if similar attacks recur again and again at short intervals, the inevitable result is that the elasticity of the pulmonary tissue becomes impaired, and that the alveoli become permanently over-stretched and emphysematous; ultimately the right side of the heart undergoes dilatation, dropsy sets in, and then follow the other mechanical results of general venous congestion, which have been described as the last stage of chronic bronchitis and of valvular disease of the heart.

Patients with confirmed asthma gradually acquire a peculiar configuration, which is very characteristic and has been well described by Salter. They are round-backed, high-shouldered, and stooping; the chest is obviously rigid and without pliancy, and from it the arms hang suspended, but inclined rather backwards and bent at the elbows. They are thin almost to emaciation, with prominent veins, cold thin hands, and a dusky complexion. The cheeks are hollow, the eyeballs turgid and watery, the mouth generally open, and the jaw rather hanging. The voice is feeble and somewhat hoarse and rough.

Ætiology.—With respect to the causes of asthma two questions have to be asked: (1) what are the *predisposing conditions* which render certain persons susceptible of the disease, whereas other persons seem to be incapable of being affected by it? (2) what are the various *exciting causes* which, in such individuals, are found to bring on the paroxysms?

The answer to the first of these questions must at present be incomplete, while of the *causa efficiens*, the invariable antecedent of asthma, either regarded as a paroxysm or as a diathesis, we are quite ignorant.

Inheritance plays a certain part in the "asthmatic tendency," as Salter calls it. This writer gives many striking instances of the transmission of asthma from generation to generation; and also mentions cases in which several brothers and sisters in a family were asthmatic without the parents being so. Dr Walshe likewise gives instances of three or four brothers or sisters being asthmatic without the disease having appeared in the family before for at least the preceding generation. This kind of connection, which is not hereditary so much as consanguineous, we have already noticed in the case

of certain undoubtedly nervous diseases, *e. g.* Thomsen's form of motor neurosis (vol. i, p. 736). It is a point of great interest, and requires much more investigation than it has yet received.

In early life a good many cases appear to be directly traceable either to *measles*, to *whooping-cough*, or less certainly to an attack of ordinary bronchitis; at the time the child seems to recover perfectly, but it becomes for the future liable to asthma, from which it had previously been entirely free. This fact certainly looks as though the fundamental defect were at least sometimes seated in the texture of the lungs or of the bronchial tubes. On the other hand, there are points which suggest that the disease is really a *paroxysmal neurosis*. Thus Salter relates the case of an epileptic patient whose fits, after having set in with their usual premonitory symptoms, were on several occasions replaced by asthmatic paroxysms. This is, however, so exceptional that the argument lies the other way; nor is it pretended that there is any connection between asthma and hysteria. Another of Salter's cases is that of a gentleman in whom a violent attack of asthma was twice suddenly excited by fear. Dr Walshe also tells how an asthmatic patient who had forgotten to take his cigarettes of stramonium and belladonna out with him, was so alarmed on discovering his omission that the dreaded attack at once came on.

Then, again, asthma is said to be sometimes closely related to gout. And it has been observed to alternate with cutaneous eruptions, becoming worse when the skin has got better, and *vice versâ*. Sir Andrew Clark has particularly called attention to the frequent coexistence or alternation of asthma and *urticaria*, and has founded thereon the hypothesis that the immediate cause of a fit may be urticarial swelling of the bronchial mucous membrane, both the cutaneous and the mucous wheals being the result of sudden vaso-motor disturbance.*

The *age* at which patients first become affected with asthma is very variable. It sets in during childhood much more often than used to be supposed. Salter found that in a fourth of his cases it had begun before the tenth year, and he saw two cases in infants of fourteen and twenty-eight days. More *males* than females are affected with it in the proportion of two to one.

The *exciting causes* of the asthmatic paroxysm vary widely in different cases. Indeed, hardly any two patients agree in their statements as to the precise conditions which bring on their attacks.

Particular kinds of weather, certain winds, cold air, the confined air of crowded rooms or railway carriages, act as exciting causes in some cases. Or the disease may be especially apt to follow the inhalation of dust, fluff, or smoke, even the smoke of an extinguished candle or of a lucifer match.

Some patients are sure to be attacked if they come near to, or in contact with, certain kinds of animals, cats, rabbits, dogs, horses, guinea-pigs, or the wild beasts of a menagerie. Salter relates many remarkable cases of this kind, and what is especially noteworthy is that years have often passed before the patient has discovered to what simple cause all his sufferings are really due. One man, a livery stable keeper, was continually asthmatic until he retired from business, and then became almost entirely free; but whenever he went back among the horses, the disease returned, and so at last he found out what was for him the special exciting cause. The writer

* See also a case in point related by Dr T. D. Pryce in the 'Lancet' for May, 1886.

once knew a lady who was attacked with asthma whenever she was in the same room with a cat; the animal could not be hidden anywhere near her without her discovering it by the painful sense of constriction in the air-passages which she quickly began to experience.

The asthma produced by hay, or rather by the pollen of grasses, is one form of the disease known as hay fever. Some patients never have asthma unless they are exposed to the influence of the pollen; others are habitually asthmatic, this being only one of many causes capable of exciting the disease in them. Many persons are attacked if they inhale the powder of ipecacuanha diffused in the air, and odours of various kinds act as exciting causes in particular cases.*

Diet plays an important part in setting up the paroxysms in almost all asthmatic patients. Heavy suppers and late dinners are very injurious; many asthmatic persons are unable to eat any solid food for several hours before bedtime. Special articles of food, among which are cheese, nuts, coffee, bottled stout, and wine are apt to provoke the disease.

Another occasional cause of asthma is the presence of polypi in the nose. This was first pointed out by Voltolini; it has since been confirmed by Haenisch. Removal of the nasal growths frees the patient from the liability to the recurrence of the bronchial affection.

Salter mentions one case in which a paroxysm was sure to occur if the rectum was allowed to remain loaded. Sometimes again the attacks are clearly traceable to uterine irritation, as when they return with each catamenial period or come on only during pregnancy or parturition.

But of all the exciting causes of asthma the most important in its influence is *locality*. And here, again, there are the strangest differences between different cases, so that it almost seems as if the disease were regulated only by caprice. In certain places the patient is sure to be attacked; in other places he is as sure to escape. As a rule, the places which are most favourable in their effects upon asthmatic subjects are large, crowded, smoky towns, like London, Glasgow, and Manchester. The most extraordinary stories are related by Salter of the effects of London air upon the disease. Persons whose lives had been rendered miserable for years have become entirely free from the disease on taking up their residence in the metropolis. He thought that three fourths or seven eighths of all cases of asthma might be cured in this way. A patient of Dr Walshe found that he could only live in perfect freedom from his asthma in the Seven Dials. It is to be noted that the influence of locality extends to neutralising the ill-effects of some other exciting causes of the paroxysms; the patient may be able in London to eat what he pleases and at whatever hours; whereas in the country the strictest dieting may be required to keep off the disease. On the other hand, there are a few cases in which the air of the seaside or of a bracing hilly district is found to be the best.

Pathology. — Various theories have been framed to account for the paroxysms of asthma, but even now the pathology of the disease cannot be said to be fully established. The *expiratory* character of the dyspnœa, exactly like that which accompanies capillary bronchitis, seems to clearly show that it must depend upon a morbid affection of the very small bronchial tubes within the lungs. During the act of expiration these tubes are as much exposed to pressure as the pulmonary alveoli themselves, and

* The narrations of such cases of asthma given by Watson and by Nousseau are the most graphic and entertaining possible.

it is not difficult to suppose that, when from any cause they are partially obstructed, they may admit air into the lungs in inspiration, and yet refuse to allow it to pass out in expiration. The question is, What is the nature of the obstruction in asthma? Now, the most obvious suggestion certainly is that it results from spasm of the muscular fibres in the walls of the tubes. That these fibres are capable of contracting, so as to narrow to some extent the calibre of the tubes, has been established by physiologists. The suddenness of the onset of the asthmatic paroxysm, the equally sudden way in which it sometimes subsides under the influence of a violent mental shock or emotion, the marked effect of such remedies as chloral, belladonna, and stramonium in bringing it to an end, the close relation which appears to exist between asthma and certain neuroses, may all be mentioned as tending to confirm the view that it is essentially spasmodic in its nature. On the other hand, it is by no means clear that spasm can account for such considerable narrowing of the tubes as must be present in asthma; nor that it is possible for spasm to be kept up for so great a length of time as that which is sometimes occupied by a prolonged paroxysm of the disease. The alternative hypothesis is that the mucous membrane of the tubes becomes very rapidly swollen by what German writers term a "fluxionary hyperæmia," or (as Weber has put it) by "a dilatation of its blood-vessels through the influence of the vaso-motor nerves." The fact that the catarrhal form of hay fever is attended with an obvious swelling of the mucous membrane of the nose is a strong point in favour of this view; for it is surely very unlikely that the asthmatic form of the same disease should be altogether different in its pathology. Sir Andrew Clark's theory of the nature of this supposed swelling has been already referred to. Störk is said to have actually observed with the laryngeal mirror that during an asthmatic attack the whole length of the trachea and part of the right bronchus were deeply congested. It is, however, not improbable that the smallest tubes may be affected both with hyperæmia and with spasm.

In 1871 Leyden discovered in the sputa certain pointed octohedral crystals, identical with those found in the blood and viscera in cases of leuchæmia, which are commonly known as Charcot's crystals, having been first described by him. Leyden's idea was that these crystals might perhaps constitute the starting-point of the asthmatic paroxysm, by irritating the peripheral ends of the branches of the vagi in the bronchial mucous membrane, and so setting up a reflex spasm of the muscular fibres beneath. But this is not at all probable, for they have also been observed in the sputa of patients suffering, not from asthma, but from other bronchial affections.

With regard to the *prognosis* in asthma, one very important fact is that when the disease occurs in childhood it generally subsides about the age of puberty, leaving the patient free for the rest of his life. On the other hand, persons above the age of forty or forty-five seldom if ever get rid of a liability to it. The longer and the more frequent the paroxysms the more serious is the case. It is also essential to notice whether in the intervals between the attacks there is any shortness of breath, or cough with expectoration. For such symptoms indicate that the asthma is complicated with chronic bronchitis, or emphysema, or dilatation of the right side of the heart; and the presence of any permanent organic lesion of the thoracic organs adds enormously to the gravity of the disease. The popular notion, if it exists, that "asthma is a lease of long life," rests upon no foundation

of fact. It only points to the fact that death in the fits is extremely rare, and that so apparently severe a disease does not kill rapidly. All life insurance offices know that asthma leads in time to emphysema and its consequences, and reject or heavily "load" such cases.

Asthma does not tend to develop phthisis; although, on the other hand, there are cases which prove that it does not prevent the supervention of that disease.

Treatment.—This falls under two heads: we have, first, to prevent the recurrence of the asthma, and, secondly, to relieve the attacks when they develop themselves, and, if possible, to cut them short.

In endeavouring to prevent the recurrence of attacks of asthma, by far the most important thing is to study its exciting causes in the individual patient, and as far as possible to remove him from their influence. The digestive organs and the diet demand the first attention; then the place of the patient's residence. A drug which is in some cases very serviceable, although its mode of action is obscure, is the iodide of potassium, in doses of eight grains thrice daily. It often utterly fails.

For the paroxysms of asthma different modes of treatment are useful, some in one case, some in another. Many patients are at once relieved when they are made faint and sick by an emetic dose of ipecacuanha or by smoking tobacco. The latter is said to be the best remedy for hay-asthma; unfortunately, those who smoke habitually are incapable of deriving benefit from it. In some cases nothing does so much good as strong hot coffee taken on an empty stomach, or hot whisky and water, or gin, or brandy. In other cases the inhalation of chloroform gives very rapid, but generally only temporary relief. Smoking the leaves of stramonium (or of one of the other species of Datura, the *D. ferox*, or *D. tatula*) is often very effectual. Or the patient may be given stramonium as a tincture or an extract, or the ethereal tincture of lobelia in full doses, or tincture of belladonna, or chloral. Some patients derive the greatest possible benefit from the fumes of nitre-paper, burnt so as to fill the room with white smoke. In other cases nothing does so much good as the inhalation of a green powder which is sold as a secret remedy in the United States, and which (it is said) may be imitated by mixing together nitre, powdered stramonium, and powdered aniseed. Chloroform and chloral hydrate are uncertain in their effects; galvanism is probably useless.

For details in the management of cases of asthma the reader should consult the late Dr Hyde Salter's work, based as it is upon a vast experience of the disease, as well as upon his own sufferings from it. It contains on almost every page practical hints of the greatest value.

DISEASES OF THE ALIMENTARY TRACT

AFFECTIONS OF THE MOUTH AND SALIVARY GLANDS OF THE THROAT AND OF THE GULLET

STOMATITIS.—*Vesicular ulcers of the mouth—Phagedænic ulcerative stomatitis—Gangrenous stomatitis or Cancrum oris—Thrush.*

MUMPS.—*Ptyalism—Metastatic parotitis.*

ANGINA.—*Catarrhal, ulcerative and herpetic forms—Acute tonsillitis or quinsy—Chronic follicular tonsillitis—Granular pharyngitis—Adenoid growths of the pharynx.*

STRICTURE OF THE ŒSOPHAGUS.—*Minor disorders—Spasmodic stricture—Simple stricture—Cancerous stricture—anatomy, course and treatment.*

THE diseases of the alimentary canal vary still more than those of the chest in their severity and importance as well as in their pathology. Some might be with more propriety classed with the specific fevers, some are due to malaria or to parasitic animals, others to disorders of the nervous system, while others again are, as far as we know at present, purely functional. Nevertheless, it is most convenient to consider them together, and the only classification attempted will be that of local distribution.

The diseases of the alimentary canal above the diaphragm come first.

AFFECTIONS OF THE MOUTH.—*Simple or herpetic ulcers.**—Among the trivial affections to which children, and sometimes adults, are liable, is one which consists in the formation of minute, shallow, round, or oval ulcers inside the mouth. They begin as small, raised, white spots, looking like vesicles. These in a few hours lose their roofs, apparently as a consequence of maceration in the fluid which constantly bathes them. The ulcers which result have an ash-grey or yellow surface, and a bright red border; they are painful, and very sensitive to the contact of particles of food, especially sugar and salt. A favourite seat is inside the lower lip, especially where it joins the gum; they may also occur upon the lining of the cheek or upon the tongue. Some persons are more liable to these ulcers than others, being troubled with them for a length of time at intervals of weeks or months. After a few days such ulcers heal of themselves, and it does not appear that any treatment is necessary, but touching them with a stick of lunar caustic relieves them.

Single ulcer of the palate.—In marked contrast with this affection is one which is described by Vogel (in 'Ziemssen's Handbuch') as occurring in weakly infants, especially those brought up in lying-in or foundling institu-

* *Synonyms.*—Aphthous ulceration of German and of some English writers—Follicular, vesicular, or herpetic ulcers—Ulcera mitia familiaria—Herpes oris.

tions. It consists in the formation of a flat ulcer at the back of the hard palate, just where the *velum* joins it. There is not usually any tendency to spread deeply, but neither is there any disposition to heal, and the ulcer remains until the child's death, which usually results from diarrhœa.

*Phagedœnic ulcerative stomatitis.**—Another form of ulceration of the mouth is a disease of considerable gravity, attended with great fœtor of the breath. The ulceration chiefly affects the gums, the edges of which become reddened and swollen, are detached from the teeth, and finally seem to break down into a grey pulp. So complete may be the destruction that the sockets of the teeth are sometimes exposed, and the teeth themselves become loose and fall out. The whole of the lining of the cheeks and lips becomes the seat of ashy ulceration. The tongue is large and doughy looking, marked at its edges by the teeth, thickly furred or ulcerated on the surface. A large quantity of acid fluid escapes constantly from the mouth, running out upon the pillow while the patient is asleep. All movements of the mouth are very painful, and food is taken with much difficulty.

The chief instances of this affection among the patients of Guy's Hospital have occurred in children between the second dentition and puberty; once, two sisters came with it at the same time. Among the soldiers of the French army it is stated to be frequent, occurring epidemically when they are overcrowded in close quarters. Whether it is then contagious appears to be doubtful. Begeron is said to have inoculated himself successfully with it in the lower lip.

The remedy for ulcerative stomatitis is chlorate of potass, which may be given in ten-grain doses at frequent intervals, dissolved in water. Or lozenges containing the salt, or Wyeth's compressed tablets, may be used with the object of securing its local action upon the mucous membrane, which action, however, appears after all not to be so essential as that resulting from its absorption into the blood. It is surprising in how short a time the affection is brought to an end; within three or four days the diseased parts begin to show a clean, healing surface. An example of this action in an adult is given in the 50th volume of 'Virchow's Archiv' (1870), p. 462, where, under the title "Gingivitis," three cases of ulcerative stomatitis are related.

Gangrenous stomatitis† is a disease almost entirely confined to young children. It does not follow the last-described affection, and differs from it in the fact that ulceration, or even phagedœnic stomatitis, begins in the mucous membrane, whereas noma begins as a slough in the submucous tissues. Its cause is unknown, and its treatment consists in destroying the diseased tissue by strong nitric acid or other surgical means.

Thrush.‡—In this country it has been usual to apply the term *aphthæ* to a condition of the mouth altogether different from that which general practice on the Continent has named *aphthous ulceration* (see last page). Confusion may be avoided by using the vernacular term "thrush" for the affection now to be described.

The earliest indication that thrush is setting in is a change in the mucous membrane lining the cheeks and other parts of the mouth; it becomes redder than natural, hot, and painful. Soon a number of minute milk-white spots appear upon its surface. These rapidly increase in size,

* *Synonyms.*—Phagedænic gingivitis—Putrid sore-mouth—Stomacace, a French term, "mouth-ill," often applied to scurvy.

† *Synonyms.*—Noma—Cancrum oris. *Germ.* Wasserkrebs, Wangenbrand.

‡ *Synonyms.*—Aphthæ, a good Greek word, and probably applied rather in the English than the German sense. Τοῖσι μὲν σμικροῖσι καὶ νεογνοῖσι παιδίοισιν, ἄφθαι. Hipp. Aph., iii, 24. *Fr.* Muguet.—*Germ.* Soor.

and run together; and in a day or two the whole surface may be covered with a nearly uniform adherent layer. At first there is some difficulty in detaching the white material. But after a time it becomes quite loose, and can be peeled off in large flakes without any bleeding. Microscopically it consists partly of layers of squamous epithelium, partly of the spores and mycelium of a fungus.

This fungus is commonly known as the *Oidium albicans*, although, according to Hallier, it is not really distinct from the *Oidium lactis*, which is the active agent in the souring of milk. It must not be supposed that the presence of the oidium in the human mouth is peculiar to cases of thrush. It has been found upon diphtheritic membranes, and in portions of fur taken from the tongue. Nevertheless, the essential cause of thrush is a vigorous and rapid growth of this fungus, leading to inflammation of the mucous membrane and detachment of epithelium, just as the fungus of ringworm produces a red and scaly condition of a part of the skin upon which it implants itself. There appears, however, to be one thing necessary before the oidium can germinate actively within the mouth. This is an acid state of the secretions which moisten its surface; and according to Vogel the mouth always gives an acid reaction at the very commencement of the affection, before any white spots are visible. The preponderance of mucus, which readily turns acid, over the alkaline salivary fluids in young infants, is perhaps the reason why thrush is so very much more common in infancy than at any other period of life.

In young children thrush may come and go with but little disturbance of the health, and without any danger to life. But in other cases it is associated with severe and even fatal diarrhœa. There is then a popular notion that the affection passes through the whole length of the alimentary canal, emerging at the anus. But although it is true that the œsophagus is sometimes affected in its entire length, there is no reason to believe that the oidium albicans is capable of germinating on any surface which is not provided with a squamous epithelium. Thus it never enters the nose or the larynx. But it does sometimes appear in the lowest portion of the rectum and upon the female genitals. And it may also be seen upon sore spots on the skin of the face and neck. The relation, therefore, of thrush to diarrhœa in young infants probably is that they are both effects of a weakly state of health from bad feeding or from some other cause. In these cases there is usually superficial dermatitis (or "eczema") of the nates, which must be carefully distinguished from syphilitic rash. In adults, thrush never occurs except in persons who are reduced to a state of extreme marasmus by a chronic malady (such as consumption or cancer), or who have passed through several weeks of pyrexia from enteric fever, or puerperal fever, or pyæmia. Thrush in adults is always a sign that the powers of life are nearly exhausted, and it is generally taken as warranting a most unfavourable prognosis; but patients may, nevertheless, recover after having had thrush, if the principal disease from which they suffer is not in itself incurable.

In the treatment of thrush all that is necessary is to wash out the mouth at frequent intervals with a weak solution of an alkaline carbonate or of borax, or to apply the *glycerinum boracis* freely to the surface of the mucous membrane.

Affections of the Salivary Glands.—*Salivation* or *ptyalism.*—In patients submitted to active mercurial treatment, the mouth is very apt

(unless due precautions are observed) to present an affection identical with that which was above described as "ulcerative stomatitis." But in addition there is also an extraordinarily profuse flow of saliva, so that the condition is known as salivation or ptyalism. On the other hand, salivation itself may occur independently of the administration of mercury, sometimes as a result of the action of other drugs (as iodide of potassium or pilocarpin), sometimes dependent upon irritation starting from distant organs (such as the uterus or the stomach), sometimes apparently spontaneous or idiopathic. But in practice such cases fall into insignificance in comparison with those that are due to the medicinal use of mercury. The quantity of saliva that is poured out is sometimes astonishing. The usual daily amount is one or two quarts; but as much as five quarts are said to have been collected in extreme instances. The patient is incessantly spitting it out of his mouth, or allowing it to dribble forth into a spitpot, which he keeps constantly by his side; at night it saturates his pillow. It is more or less viscid or glairy in consistence, and is said to have sometimes a specific gravity as high as 1059; but as the case goes on its specific gravity falls till it is scarcely above that of water. It contains little sulphocyanide of potassium, and less ptyalin.

Mercurial salivation is now very seldom seen. In administering mercury one watches the patient's mouth carefully, and the medicine is at once stopped when any disagreeable odour is perceptible in his breath, or when his gums become in the least degree inflamed, or his teeth tender on pressure. Or, if there should be special reason to anticipate the onset of salivation from the fact that one is using mercurial inunction, or calomel vapour baths, or full doses of blue pill instead of minute doses of the bichloride, one may often obviate it by giving at the same time chlorate of potass. When once salivation has developed itself chlorate of potass appears to have little direct influence upon that symptom, although it more or less quickly brings the gums and the mucous membrane of the mouth into a healthy state. The ordinary duration of salivation is from one to three weeks. While it lasts the patient generally becomes thin. His urine is scanty and his bowels are constipated. Relief may be given by washing out the mouth with astringent solutions, as of alum or gallic acid, to which some tincture of myrrh and tincture of opium may be added.

Mumps.*—The salivary glands, like the pancreas, are very little subject to the diseases which affect the liver and kidneys, or even the mamma and testes. The most frequent affection is one which, although it appears like a mere local inflammation, is in reality the expression of a specific disease, transmitted by contagion, occurring epidemically, and possessing the power of protecting against its repetition in the same individual. For this disease the popular name of Mumps is the best.

The earliest symptom of mumps is commonly an aching pain in the parotid region on one side, increased by every movement of the jaw, as in speaking, or in taking food. But sometimes malaise and pyrexia precede by a day or two all local signs of the affection. Swelling very soon sets in; the hollow between the mastoid process and the jaw is filled up, its place being taken by an ill-defined projection, which throws outwards the lobule of the ear, and extends over the cheek towards the angle of the mouth, and

* *Synonyms.*—Parotides—Cynanche parotidæa (Cullen)—Parotitis epidemica—*Scottice*, The Branks.—*Fr.* Les Oreillons.—*Germ.* Ziegenpeter.

downwards some distance into the neck. The submaxillary glands usually follow, and a little later the opposite side participates in the disease; so that in the course of from three to six days the whole of the face becomes surrounded by an immense mass of firm doughy infiltration, which gives to the patient a truly ridiculous aspect, there being an enormous double chin and the natural contour of the throat being altogether lost. The skin over the affected parts may either be slightly reddened or pale and waxy looking. Internally the swelling extends to the tonsil and the pharynx. The movements of the jaw are greatly impeded. The teeth can with difficulty be separated so as to admit the end of a spoon between them; the patient is obliged to confine himself to a fluid diet, with eggs, custards, and other things that require no mastication.

Sometimes the saliva appears to be very deficient, sometimes to be secreted in excessive quantities; but as a rule its quantity and quality are unaffected. The head is kept fixed in one position, with the face directed straight forwards. If, as sometimes happens, the affection remains all along confined to one side, the head is turned rather towards that side. Pain and tenderness continue more or less severe. Sometimes there is hardness of hearing, or the patient complains of shooting pains in the ears or of a continuous ringing sound in them. On about the fourth day the pyrexia ceases. It is seldom of much intensity, although temperatures of 104° are now and then recorded, and typhoid symptoms have even been known to develop themselves.

After three to six days the swelling begins to subside, and its absorption takes place so rapidly that within about an equal period of time it entirely disappears. Thus the whole duration of the disease is rarely beyond one or two weeks. It is sometimes followed by desquamation of the cuticle over the affected parts.

Mumps, like other specific diseases, protects from itself. But sometimes, when one side has been affected, the other follows suit within a week or fortnight. And, apart from these relapses, cases of a second attack are met with. In one case known to the editor a boy had mumps three times during his school life.

Complication.—In some cases, however, as the inflammation of the face and neck passes off, or even when it has for some days been at an end, the patient is attacked with acute "metastatic" affection of one *testicle*, generally (it is said) the right. The organ becomes swollen and painful, and there is sometimes effusion into the *tunica vaginalis*, with œdema of the corresponding side of the scrotum. After a short interval the other testicle may be attacked in its turn. When this complication occurs there is usually a return of the pyrexia. Trousseau has drawn attention to the fact that the secondary orchitis of mumps is now and then accompanied by symptoms of the most alarming character, though apparently without real danger to life. In one instance a condition of collapse was suddenly developed, in another the patient fell rapidly into a typhoid state. What rendered the diagnosis of the second case even more obscure than it otherwise would have been, was that the initial attack of mumps had been so slight and transient that no notice had been taken of it, and nothing could be learnt about it until consciousness returned. High fever and delirium are sometimes present.

Some years ago the writer saw a case of orchitis from mumps in which the pulse became extraordinarily slow and remained so for several days;

the temperature also fell to a low point and the breathing was much reduced in frequency.

The inflammation of the testicle usually lasts from three to six days, and then rapidly subsides. Not infrequently, however, it leads to a permanent atrophy of the organ. Urethritis has been noted as a complication. No explanation seems to be at present possible of the liability of mumps to set up orchitis; we can only refer it to that mysterious correlation of distant organs, in their proclivities to disease, of which we find so many other examples in pathology. Mr Stephen Paget has drawn attention to other instances of relation between disorders of the abdominal viscera (to which the testes belong) and parotitis ('Lancet,' April 17th, 1886).

In females affected with mumps it is said that the *mammæ* or the external genitalia sometimes exhibit a like tendency to swelling and inflammation. And this fact (if it be one) is of special interest, because of a case observed by Peter, in which a young woman who had amenorrhœa was several times attacked with parotitis at what should have been her catamenial periods, while on other occasions one of the labia became swollen and painful. According to some text-books, the *ovaries* may be the seat of "metastatic" inflammation after mumps. But this seems to be very doubtful.

Ætiology.—Mumps is most apt to occur in children from ten to fifteen years old, but it is not uncommon in adults. It is said to be more apt to affect males than females. The secondary orchitis is seen chiefly in boys about the age of puberty and in young men.

The contagion of the disease is supposed to be transmitted by the breath. The length of the period of incubation is variously stated by writers; it seems to be generally about fourteen days, but may range from six to twenty-two days.

Pathology.—With regard to the exact seat of the morbid process in mumps there is still some uncertainty. It is certain that the connective tissue outside the salivary glands is largely involved in the inflammatory œdema which must constitute the swelling in the disease; and some writers think that within the glands themselves the structure affected is rather the fibrous stroma which supports the glandular acini than the acini themselves. The presence of a microbe in the blood or saliva has not been discovered.

The *prognosis* is always favourable. Even when the fever runs high and orchitis is attended by delirium, there appears never to be any real danger. Neither the inflamed parotid nor the inflamed testes suppurate, though the latter may atrophy.

The only *sequela*, beside this last rare effect of the metastasis, is deafness; and this also is not common. According to Mr Dalby, it may be either "catarrhal"—when it passes quickly away, or "nervous"—when the condition appears to be permanent.

Very little *treatment* is required in cases of mumps. The patient should be kept indoors and out of the way of draughts. As has already been remarked, he can take only fluid food. Fomentations may be applied to the swollen parts.

Metastatic parotitis.—In marked contrast with mumps is an affection of the parotid gland which, instead of being a manifestation of a specific contagious disease, arises as a complication of other maladies, such as fevers, dysentery, or even local affections, such as intestinal obstruction from cancer of the sigmoid flexure, as in a case of the author's. This form of parotitis

is almost always unilateral. It sometimes subsides without suppuration, but it far more often leads to the formation of an abscess, which may either point behind the ramus of the jaw, or break into the external auditory passage, or burrow down into the neck or upwards in the pterygoid region towards the base of the skull.

The idea has been suggested that this "metastatic" parotitis is perhaps, after all, the result of dryness of the buccal mucous membrane, leading to an obstructed state of Steno's duct, with decomposition of the retained salivary secretion. But for this opinion there is little evidence.

It is said that the prognostic importance of parotitis occurring in the course of a fever depends very much on the period of the disease at which it is developed. At an early stage it is of very grave significance; at an advanced stage, or during convalescence, it is a comparatively trifling matter.

Gouty parotitis has been described. It must be excessively rare (see 'Med.-Chir. Trans.,' vol. lxx, p. 217, 1887).

Affections of the Fauces.*—Systematic writers on diseases of the throat recognise a great variety of affections of the fauces, all of which are attended with difficulty or pain in deglutition, and with more or less marked inflammation of the pharyngeal tissues. Some of these affections need detailed descriptions; but of others a cursory notice will suffice.

Thus *catarrh of the pharynx* is sufficiently characterised as redness and slight swelling of the posterior wall and of the palate, coming on after exposure to cold. In persons who have repeatedly had attacks of catarrh, a "relaxed sore-throat" is apt to be of frequent recurrence; the fauces feel dry and painful (especially in the morning on first waking), but the symptoms all pass off after breakfast, when nothing is to be seen beyond elongation of the uvula, a pendulous state of the palate, and perhaps some dilatation of veins in the mucous membrane.

Then there is an *ulcerated sore-throat* which is particularly apt to occur in nurses and medical students who are in close attendance on the sick, or in other persons if weakened by unhealthy hygienic conditions. In this affection are seen on the surface of the tonsils and other parts of the fauces small white superficial ulcers, which are vaguely called follicular or aphthous, and resemble in appearance the minute yellow ulcers of the mouth and tongue described above (p. 299). There is a considerable general disturbance, the breath is foul, and the tongue is furred. Wine, quinine, and tincture of iron are indicated in the treatment, and generally bring the affection to an end in a few days.

It is necessary to distinguish from ulcers on the tonsils certain circumscribed whitish-yellow patches, which are really masses of inspissated and protruding secretion. Patients who exhibit these patches often come for advice, complaining of a recent sore-throat. Guaiacum is very effectual in relieving such cases.

Another faucial affection that has been carefully studied by foreign observers is *herpes of the pharynx*. This consists in an eruption of opaline vesicles, sometimes few in number, but sometimes so thickly crowded together that a diphtheritic membrane may easily be supposed to be present. Sometimes it recurs again and again in the same patient; and sometimes it alternates with a herpetic eruption on the skin or on the genitals.

* Sore-throat.—*Fr.* Mal de gorge.—*Germ.* Halsweh.—The terms *angina* and *cynanche* are often applied somewhat indiscriminately to all inflammatory affections of the fauces.

*Quinsy.**—Among the inflammatory diseases of the fauces set up by cold, there is one that mainly affects the tonsils, and is well known by the popular name of "quinsy." Mackenzie gives figures, based upon an analysis of 1000 cases, showing that it is far more frequent in persons between fifteen and twenty-five years old than at any other age; it is comparatively seldom seen in children, or in adults beyond the age of forty. Some persons are exceedingly liable to this affection, being attacked by it whenever they get a chill, or sometimes as the result of slight stomach disorder, or (in the case of women) of menstrual disturbance. Acute tonsillitis sometimes precedes rheumatic fever. Not infrequently the pyrexia precedes the local inflammation by a day or two, so that in servant-girls and young adults generally, the possibility of quinsy coming on should always be remembered. In many persons there is habitually a chronic enlargement of the tonsils, and upon this acute inflammation from time to time supervenes.

Quinsy appears to be rather more common during autumn than at other seasons. It is attended with an enormous increase in size of the tonsil, which forms a red, shining, globular mass, projecting into the fauces, and also distinctly to be felt in the neck at the angle of the jaw. When both tonsils are involved, they may come into contact in the middle line, being flattened and even (as Bristowe remarks) ulcerated from mutual pressure. But more often one of them is alone affected; and sometimes, just when the inflammation is subsiding in one, the other is attacked in its turn. The uvula, the soft palate, and the pillars of the fauces all partake more or less in the swelling. The patient complains of severe pain in the throat whenever he moves his jaw, and especially in attempting to swallow. The pain may radiate to the ears. The amount of pyrexia often seems disproportionate to the severity of the local affection. Even if there is no actual rigor, the patient experiences alternate chills and flushes of heat, and complains of headache, malaise, and pains in the limbs; the pulse ranges from 100 to 120 in the minute, and is full and bounding; the temperature rises to 102°, 103°, or even 105°. The tongue is thickly furred and the breath foul.

The writer once made an autopsy in the case of a young child who had died in Guy's Hospital during the previous night of suffocation as the result of severe swelling of the tonsils from quinsy. Such an occurrence, however, is exceedingly rare. The disease almost always ends favourably, either subsiding more or less rapidly, or else advancing to the formation of an abscess in the tonsil, which breaks and discharges a thick fœtid pus that is generally swallowed. There is then immediate relief to all the symptoms, and the patient soon feels perfectly well. The ordinary duration of the disease is three or four days; but if the two sides of the throat are attacked in succession, it may be prolonged over a week or ten days. In some cases it is said that suppuration starting from a tonsil extends down into the neck, and burrows until it may even reach the chest. Cases also have been published in which tonsillar abscesses have eroded the carotid artery and given rise to fatal hæmorrhage. And in one reported case suffocation occurred as the result of the breaking of the abscess, which filled the upper air-passages with pus. Mackenzie, who cites this case, remarks

* *Synonyms.*—Cynanche—Acute suppurative tonsillitis or amygdalitis—Angina tonsillaris—Angina phlegmonosa. Κυνάγχη or Συνάγχη refers to the choking sensation produced by this and similar inflammations of the throat. *Angina,* from *angio,* has a similar meaning. The French *Esquinancie* and English *Quinsy* are corruptions of Cynanche.

that such an accident is much to be dreaded should the abscess give way during sleep.

Some French writers, especially Maingault and Gubler, have maintained that tonsillitis is now and then followed by paralysis of the soft palate, like that which is so often seen after diphtheria. But one would be rather disposed to think that in such cases the specific disease was really present, for marked swelling of the tonsil, and even suppuration in it, is by no means rare in diphtheria.

In the *treatment* of quinsy, sucking ice or the application of cold to the throat often gives more relief than anything else. But when an abscess is in process of formation, the course of the disease may apparently be hastened by the use of fomentations and poultices externally and by steam inhalations. As soon as fluctuation can be felt an incision should be made with a guarded bistoury. Störk remarks that the best way of detecting a soft spot is to push the parts inward with one forefinger placed at the angle of the jaw, while the other forefinger in the mouth is carefully passed down over the inflamed structures from point to point. In opening a tonsillar abscess the cutting edge of the knife must be directed inwards and not outwards, lest the internal carotid artery should be wounded. Mackenzie remarks that when the patient refuses surgical interference, an emetic often leads to the immediate rupture of the abscess.

In many cases, however, it would seem that by suitable treatment quinsy may be made to abort, and the occurrence of suppuration be prevented when it would otherwise have taken place. Aconite has been recommended for this purpose, but according to many experienced practitioners guaiacum is more efficacious. Mackenzie administers it in the form of lozenges, each of which contains three grains of the resin; one of these, taken every two hours, seldom (he says) fails to arrest the disease at its first onset.

Chronic enlargement of the tonsils.—A not uncommon affection of the tonsils is chronic overgrowth of their substance, or (as it is often called) "chronic hypertrophy." They may be as large as chestnuts or even larger, and they are very firm and fleshy, smooth on the surface, but sometimes with cheesy or calcareous masses projecting from cavities in their interior. The affection may exist from early infancy, or develop itself during childhood or at puberty. In some cases it subsides as adult life is reached, and it seldom persists after the age of thirty. In children it is attended with many inconveniences; the mouth has to be kept open during sleep, and a snoring sound accompanies the breathing; some even say that a pigeon breast may result from the obstruction to the entrance of air into the lungs. This condition often goes with a narrow and high-arched palate, with crowded teeth and a straitened passage through the nostrils. Such children snore at night and breathe through the mouth during the day, and thus constant faucial irritation is kept up. There is something about the physiognomy of such patients that enables one to see at a glance what is the matter with them; Mackenzie speaks of their "open mouth, drooping eyelids, and dull expression;" and in addition to these peculiarities the voice is thick and nasal, while the act of deglutition (as in swallowing the secretions that accumulate in the mouth) is performed in a clumsy manner and with obvious effort. Another effect of chronic enlargement of the tonsils is deafness, which is attributed to a coincident swelling of the mucous membrane of the Eustachian tube. Mackenzie says, too, that the senses of smell and of taste are often impaired; but sometimes the

affection exists to a very marked extent without giving rise to any symptoms whatever.

The *treatment* may at first consist in the administration of cod-liver oil, iron, and other tonics, while the tonsils are every day brushed over with a solution of perchloride of iron, or smeared with powdered alum or tannin. But if there is much overgrowth of solid tissue, excision by means of the guillotine is almost always necessary. If hæmorrhage follows the operation, it is generally easily checked by making the patient suck ice; if not, Dr Mackenzie recommends that he should slowly sip half a teacupful of a strong solution of tannic and gallic acids, containing ʒvj of the former and ʒij of the latter to the ounce of water. The wounded parts remain sore for some days; during this time the food must be soft and bland, and the use of marsh-mallow lozenges is recommended as soothing.

Granular pharyngitis.—A very common affection of the back of the throat is one in which the pharyngeal surface appears dotted over with small prominences of about the size of millet-seeds, which may be either scattered, or closely packed together, or confluent into ridges of varied breadth and length. At the same time the smaller blood-vessels of the mucous membrane are seen to be dilated and tortuous. With regard to the nature of the granulations, there appears to be still room for further investigations. It is generally supposed that they consist of enlarged glands which have taken more than their share of a process of hypertrophy that likewise affects other structures, but not to so great an extent. Störk, however, says that the mucous membrane generally is in such cases often thinner than natural, and that what characterises the granulations microscopically is the absence of a superficial stratum of epithelium, large round swollen cells lying uncovered and exposed. As a rule, the surface of the throat is in such cases dry, there being apparently a deficiency of the normal secretions. But Mackenzie describes an "exudative form" of the affection in which viscid mucus is seen adhering in patches to the follicles, or in which their orifices are filled with a white material resembling cream cheese in appearance.

Dr Horace Green, of New York, was the first writer to give such an account of granular pharyngitis as fixed itself upon the attention of the profession; his names for it were "follicular disease" and "follicular inflammation" of the throat and air-passages. It is, indeed, by no means limited to those regions which are directly visible at the back of the mouth. Sometimes it spreads upwards towards the vault of the pharynx, sometimes downwards towards the larynx. Michel has recently insisted on the importance of thoroughly exploring with the laryngeal mirror every part of the fauces; for example, he says that when the rest of the surface is quite healthy, there may be a small patch of disease just behind one or both of the posterior arches of the palate; this may have been overlooked by one medical man after another, and yet it may really be the cause of a great deal of suffering to the patient, as is shown by touching it with a probe, and afterwards by the results of applying treatment to it.

The subjective symptoms of granular pharyngitis vary widely in different cases. Probably it very often causes no discomfort at all. Sometimes, however, it gives rise to a very troublesome feeling of stiffness or dryness in the throat, to a constant need for swallowing, to a pricking pain during deglutition (especially deglutition of the fluid secretions of the mouth), to a

tickling sensation compelling a frequent and often painful cough, or to incessant hawking, in the hope of getting rid of mucus, until actual retching may occur. In other instances, the chief complaints are associated with exertion of the voice. Speaking or singing may be attended with a sense of painful effort; the patient may be obliged to stop from time to time, in the middle of a sentence even, to swallow or to clear the throat. Such cases have given to the disease the name of "clergyman's sore-throat;" it is indeed very common in those who earn their living by means of the voice, as in preachers and public singers, as well as in street hawkers and costermongers. As a rule, exposure to cold plays a conspicuous part in bringing out and in aggravating the symptoms of granular pharyngitis. According to some writers, irritation of the fauces by excessive smoking or by alcoholic liquids should be mentioned among the exciting causes of the affection. An over-sensitive state of the nervous system appears in many cases to contribute largely towards increasing the severity of its subjective symptoms; some patients appear to be almost incapable of forgetting, even for an instant, the morbid feelings which they experience in the back of the throat; they are ever flying from one medical adviser to another, and they thoroughly deserve the name of hypochondriac. Another element in the ætiology of granular pharyngitis appears to be an inherited predisposition. Dr Green speaks of three brothers, all clergymen, who were compelled by it to give up their official duties, and whose mother was also affected with it; and he also alludes to a large number of cases, recorded in his notes, of two or three members of a single family having been treated for it.

The age at which it is most apt to occur is from twenty-five to thirty-five, and in men more often than in women. But Mackenzie has seen it in children who were eight, six, or even only three years old.

In the *treatment* of granular pharyngitis, the essential point appears to be the destruction of the granulations. So long as no local applications were used except gargles, inhalations, and brushing over the fauces with solutions of nitrate of silver, or even with the solid caustic, it was admitted by all candid observers to be an exceedingly intractable affection, and one that often ran on for years in spite of all that was done to cure it. In fact, as gargling with fluids is commonly practised, it seldom brings them into contact with any part of the fauces behind the anterior pillars of the palate. Guinier, of Cauterets, has clearly proved, in his brochure entitled 'Étude sur le Gargarisme Laryngien,' that when a person with his head thrown backwards makes fluid bubble about in the back of the mouth, while he goes on inspiring at regular intervals through the nose, the fluid is supported by the base of the tongue, the uvula, and the anterior pillars of the palate; any portion of it which passes further backwards is instantly swallowed. On the other hand, he has also shown that it is possible to teach patients to gargle in quite a different manner, and that the fluid then passes not only into the pharynx, but even into the larynx itself, resting directly upon the upper surface of the vocal cords. The directions are that the head should be slightly raised, the mouth but little opened, the lower jaw thrown forwards, so as to lift the chin. Having taken a small quantity of fluid into his mouth, the patient is to draw a deep breath through the nose, and then to allow the fluid to fall back into the fauces, while he endeavours to emit the sound of the vowel *ê* (or the English *a* long). This, of course, means that the cords are brought together, and

that the act of expiration is begun; while at the same time the epiglottis is raised so as to throw widely open the upper part of the laryngeal cavity. A bubbling sound is produced by the thin stream of air which passes outwards between the cords; this sound, Guinier says, is quite unlike that of ordinary gargling; it resembles rather the rattle in the throat made by persons who are moribund. The act of gargling within the larynx can be continued only so long as a slow expiration is being maintained. Before a fresh breath can be taken, the fluid must be thrown up into the pharynx, whence it often passes out through the nostrils. The writer has himself seen Guinier demonstrate with the laryngoscope the presence of a layer of fluid resting upon his own vocal cords. And he seems to have gained a considerable amount of success in the treatment of granular affections of the pharyngeal and laryngeal mucous membranes by making patients gargle in this way with the sulphur water of Cauterets.

Of late, however, far better results than had ever before been attained in this troublesome disease have followed the adoption of much more active measures. Mackenzie applies a caustic paste to each granulation separately, touching on the same day only two or three, and sometimes only one, of them. It is easy to imagine that this practice must be tedious and must in many cases cover a long space of time. But in 1873 Dr Michel, of Cologne, drew attention in the 'Deutsche Ztschrft. f. Chirurgie' to the success which he had attained in about seventy cases by the use of the galvanic cautery. Since then many other observers have adopted this method. It consists in applying a heated platinum loop to the granulations, so as just to destroy their surface; and, as a rule, the operation has to be repeated only three or four times, inasmuch as the effect is not limited to the part immediately cauterised, but extends to some distance around. There is little or no pain at the time, except when the pillars of the fauces are the parts touched by the instrument; the inflammation which follows can be kept within bounds by making the patient during the first few hours suck ice at intervals; there is nothing to prevent his continuing his usual avocations. Dr Foulis, of Glasgow, employed a small gas cautery for the same purpose; but this has the disadvantage that it must be heated before being passed into the mouth, whereas the platinum loop of the galvanic cautery is cold at the times of its introduction and of its withdrawal.

Adenoid vegetations in the vault of the pharynx.—In 1869 Dr Wm. Meyer, of Copenhagen, drew the attention of the Royal Medical and Chirurgical Society (vol. liii) to this affection, which he was almost the first to recognise, but which has proved to be exceedingly common. It consists in the presence of masses of various shapes and sizes, growing most frequently from the posterior wall or from the roof of the pharynx, but sometimes also from the sides of that cavity, or even from the upper surface of the soft palate, but never from the back of the nasal septum. They are described as occurring in three forms, the *cristate*, the *cylindrical*, and the *flat*. They are sometimes soft, and sometimes hard. They contain many vessels, especially veins; and they are otherwise made up of a scanty areolar network, having its meshes filled with lymph-cells. Their epithelium may either be ciliated, or of pavement form, according to their exact seat. Their colour is generally the same as that of the more or less congested mucous membrane in their vicinity, but they may have a slightly yellowish hue.

It is often possible to tell by the way in which a person speaks and even

by the expression of his face, that he is affected with pharyngeal vegetations. The peculiarity of the speech consists partly in an inability to utter the nasal sounds *m, n, ng,* so that instead of "common" the patient says "*cobbod,*" instead of "nose" "*doze*" instead of "song" "*sogg*;" partly in a loss of the resonance naturally given to the voice in the nasal cavities. This depends upon the occlusion of the posterior nares preventing the nasal cavity from acting as a resonator. Hence the explosive vocal sounds take the place of the continuous vocal or "nasal" sounds. The same effect is produced by closing the anterior nares.

The peculiarity of the facial expression depends upon the obstruction to the passage of air through the nostrils in breathing. This causes them to appear narrow and collapsed, and the nose itself looks thinned and flattened from side to side. It also compels the patient to keep the mouth more or less constantly open ; and since the orbicularis oris no longer gives support to the other muscles of the face, the countenance acquires a vacant stupid aspect, often increased still further by an odd trick of twisting and pouting the lips. Other symptoms are a feeling of fulness in the upper part of the fauces, as though there were a foreign body there, a secretion of thick greyish or greenish mucus which glides down the pharynx and compels the patient to be constantly clearing the throat, the presence of blood in the mouth, especially on first waking in the morning, and a more or less habitual headache.

But what most commonly causes patients who have vegetations in the vault of the pharynx to seek medical advice is the impaired state of their hearing. Sometimes there is only occasional deafness with a tinnitus, especially if they happen to take cold, but in many cases the aural symptoms are far more serious and lasting; there is chronic catarrh of the tympanic cavity, and the drum may even become perforated, giving exit to a purulent discharge. It is to be observed, too, that various affections of the fauces are apt to be associated with the presence of pharyngeal adenoid growths. The tonsils may be enlarged, there may be granular pharyngitis, the uvula and the soft palate may be thickened. There may also be catarrh of the anterior parts of the nasal cavities, though in the majority of cases the secretion of the Schneiderian membrane is rather deficient than excessive.

The easiest method of detecting with certainty the presence of vegetations in the vault of the pharynx is to explore the upper part of the pharynx with the forefinger. This is to be passed between the tongue and the roof of the mouth, and insinuated by the side of the uvula until it glides upwards behind the velum. It is then carried along the posterior edge of the septum of the nose, and turned in various directions until every part of the space has been thoroughly examined. If necessary a probe introduced through the nostril may be used to bring the individual vegetations in contact with the finger. The examination may cause some nausea, and may even be followed by pain in the back of the head ; there is often a good deal of bleeding from the growths when they are touched. Rhinoscopy is seldom of much assistance in diagnosis. The vegetations can be most satisfactorily seen in cases of cleft palate, an affection that appears to be rather frequently associated with their presence.

The systematic examination of children at schools in Denmark, England, and Holland has shown that from 1 to 5 per cent. of them are affected with pharyngeal adenoid growths. The disease is said to be more frequent

when the climate is cool and damp. It is seen chiefly in persons under the age of twenty-five years, and it appears often to be congenital or to date from very early childhood. It frequently affects several members of the same family; Meyer thinks that it is more common in boys than in girls. It is said to be often a sequela of measles, and also to be traceable in many cases to "colds." Its relation to scrofula is doubtful; according to Wiesener, of Bergen, it may lead to an infiltration of the cervical glands such as is commonly called scrofulous.

When adult life is reached, adenoid vegetations in the vault of the pharynx appear commonly to shrink and to disappear spontaneously. Nevertheless, it is important to remove them as soon as their presence is detected, especially on account of the damage which they do to the organs of hearing. Sometimes cauterisation with solid nitrate of silver suffices to destroy them, but they generally have to be scraped off by suitable instruments, of which descriptions may be found in Meyer's several papers, and in the 'Transactions of the International Congress of 1881.' The chief of them consists in a small oval ring, which has a sharp though not absolutely cutting edge, and is mounted on a slender stem. This is passed backwards through the patient's nostril, and is guided to the bases of the vegetations by the operator's left forefinger introduced through the mouth. The operation causes little pain but profuse hæmorrhage, which is easily checked by the injection of cold water containing salt and carbolic acid. It is often followed by headache, and even by slight stupor for a few hours. It sometimes has to be repeated once or oftener, because all the vegetations are not completely got rid of on the first occasion. If any remains of them are left they are sure to grow again. Meyer therefore insists upon the importance of after-treatment, consisting in cauterisations with nitrate of silver and daily injections of a solution of bicarbonate of soda, or of chlorate of potass. So long as there is any soft tissue to be felt which bleeds when touched, these measures should not be discontinued. The effect of the complete removal of pharyngeal adenoid growths is quickly to restore the natural speech, to change in a surprising manner the expression of the patient's face, and in many cases to bring back the sense of hearing.

Diseases of the Œsophagus.—This part of the alimentary canal is remarkably free from the slighter inflammatory affections which are so common both above and below it. Its thick layer of squamous epithelium seems to protect it from all but the most violent irritation, and it has neither the rich blood-supply, nor the active secreting functions, nor the abundant lymphatic tissue, which elsewhere in the alimentary tract become occasions of disease.

Chronic *inflammation* of the gullet with thickening of the mucous membrane is, however, seen either as the result of external pressure, *e. g.* from a thoracic tumour or an aneurysm, or in cases of habitual congestion from chronic valvular disease of the heart. In the former case the thickened and opaque mucous membrane is, in external appearance as in pathology, like the "corns" produced by friction on the pericardium, or the white patches of the tongue where it is touched by a tooth or other mechanical irritant. In the latter case extreme venous congestion may be discovered after death, with desquamation of the upper layers of epithelium, a condition comparable to the far more important congestive catarrh of the stomach in similar disease of the heart.

Occasionally, when no source of pressure or irritation can be ascertained, the mucous membrane is found (at times over a considerable space) to be covered with minute papillæ which may be large and circumscribed enough to deserve the name of a *papilloma*.

Functional stricture. — The above pathological conditions are without clinical significance; there are, on the other hand, functional affections of the gullet which are at present without an anatomical explanation. Of these the most important is what has been called "Spasmodic Stricture." The patient is usually young, most often a girl at the age between puberty and childbearing, when functional neuroses are most common. It also, however, occurs in male subjects, and one of the most marked and obstinate cases the writer has seen was in a boy of fourteen. In most cases the neurotic or "hysterical" character of the affection is sufficiently evident, and the easy passage of an œsophageal sound completes the evidence.

Regurgitation.—Another condition which is probably at first functional is rejection of food after it has passed the constrictors of the pharynx. This differs from the gastric regurgitation, or, as it may be termed, "rumination," which will be described under disorders of the stomach; for here the food never reaches the gastric cavity, but is detained in the gullet. Œsophageal regurgitation appears to begin rather as a bad habit than a disease; but whether or not there be any structural lesion as its original cause, there is frequently, or perhaps always in confirmed cases, a pouch formed in the lower part of the gullet, in which food collects before its regurgitation. *Dilatation of the œsophagus* is said to be more frequent in men than in women. It usually involves the whole thickness of the tube; but cases have been described in which the mucous coat alone has protruded between the muscular fibres, so as to form a hernial pouch. A classical case of this curious condition was published in the thirteenth volume of the 'Medico-Chirurgical Transactions' (1849) by Mr Worthington, of Lowestoft.

An œsophageal pouch is most frequently met with, not as a primary lesion, but as the result of a stricture immediately below it. It has occasionally proved to be the result of a mere narrowing of the gullet at its cardiac end. Such a case is described and figured by the author in the 'Guy's Hospital Reports' (3rd series, vol. xvii, p. 414), where at last a cancerous growth developed and ended in the patient's death at eighty-four, forty years after the appearance of dysphagia. In the same paper is figured a dilated œsophagus resulting from a simple, non-traumatic stricture of the cardia, which was described by Dr Wilks in the seventeenth volume of the 'Pathological Transactions' (p. 138). Regurgitation and dysphagia had existed all the life of the patient, a healthy farmer, who had once consulted Sir Astley Cooper for œsophageal stricture, and who died at seventy-four of acute pneumonia. Mr Durham has discovered two cases of dysphagia and simple stricture, recorded by Sir Everard Home ('Practical Observations on the Treatment of Stricture in the Urethra and in the Œsophagus,' 1821, vol. ii, p. 398). One of these showed, *post mortem*, a fold of mucous membrane, which narrowed the gullet just opposite the cricoid cartilage.

Dysphagia lusoria, as it has been called, demands a word of notice. The term was first applied by Dr Bayford, of Lewes, to a case (probably of

spasmodic stricture of the gullet) in which the right subclavian artery arose from the third part of the aorta, and passed to its distribution between the œsophagus and trachea. The existence of this abnormality was probably a mere coincidence. The much more frequent irregular arrangement of the right subclavian arising from the third part of the arch, and passing *behind* the trachea and œsophagus, between the latter and the vertebræ, is found, *post mortem*, in persons who have never experienced difficulty in swallowing.

Organic strictures of the œsophagus may be divided into the simple or non-malignant, and the cancerous.

Simple stricture can in most cases be traced to a *traumatic* origin, most frequently to irritant poisons, such as the mineral acids. Constriction of the gullet by external cicatrices or pressure of diseased vertebræ, abscesses, aneurysms, cancerous mediastinal glands, or possibly a distended pericardium, will of course have the same results as true stricture or contraction of its walls, and will only be distinguishable during life by evidence of the presence of the external compressing cause.

Stricture is occasionally due to contraction of a *simple ulcer* of the œsophagus, which is, however, a very rare affection compared with the corresponding lesion in the stomach, or even the duodenum. There is no doubt that *syphilitic* ulcers, probably of a tertiary gummatous nature, may give rise to contraction of the œsophagus ('Guy's Hospital Reports,' vol. xvii, ser. 3, 1872, p. 413). Above a stricture there may frequently be seen hypertrophy, with or without dilatation of the muscular walls of the gullet.

Malignant stricture of the œsophagus is always primary and is usually of the epithelial, keratoid, or flat-celled variety. Adenoma or glandular cancer has occasionally been observed, but true examples of the encephaloid or scirrhous variety are extremely rare.

The most frequent position is generally stated to be in the middle of the tube, opposite the bifurcation of the trachea. Less in frequency is the upper portion, where cancer spreads so as to be described indifferently as pharyngeal or œsophageal. Lastly, a malignant stricture is occasionally met with at the entrance into the cardiac orifice of the stomach. It is here that its presence is most apt to be overlooked, the diseased portion being left in the diaphragm when the stomach and œsophagus have both been removed, as Virchow long ago observed. The foregoing statement is that of Wilks, Rindfleisch, and Klebs. Many text-books, however, follow Rokitansky's original assertion that the upper part is the most frequent seat and the lowest the rarest. Förster, Moxon, Payne, and Coats say that the commonest seat of cancer of the œsophagus is its lowest third; and this statement is confirmed by the careful analysis of Petri and Zenker. In 58 cases collected by these two writers 4 were in the upper, 14 in the middle, and 24 in the lower third. The remaining 16 cases spread over the middle and adjacent parts also. The latter group of cases is the disturbing element which has probably affected the classification of statistics. It still, however, remains true that the part which corresponds to the bifurcation of the trachea is a frequent seat of œsophageal cancer, and that cancer of the extreme cardiac end is rare. In 13 cases collected by Dr Moore, of St Bartholomew's Hospital, the lower third was affected in 7, the middle third in 5, and the upper in one.

The malignant growth is sometimes a mere cartilaginous ring like an annular stricture of the colon, but more frequently it forms an ulcer which only partially encircles the tube, and infiltrates and spreads up and down for an inch, or even more. Hughes Bennett recorded a rare case of double cancerous stricture ('Prin. and Pract. of Medicine,' p. 453).

The growth of epithelioma of the gullet is slow, and it rarely affects more than the neighbouring lymph-glands of the mediastinum; but occasionally secondary nodules are found in the viscera,—least rarely, perhaps, in the liver or the lung. It may produce fatal hæmorrhage, or may perforate the œsophagus by sloughing, involve the vagus nerves, or open anteriorly into the trachea or lung.

Myomata, polypi, besides the warty growths above mentioned, and other non-malignant growths, have occasionally been observed in the œsophagus.

Malignant stricture is more commen in men than in women, and is rare before middle life.

Its early *symptoms* are slight and its progress insidious. Difficulty in swallowing solid food is commonly the first complaint. Pain, though occasionally severe, is often long before it appears, and is sometimes almost absent. Gradually the patient finds it more and more difficult to swallow soft food, and at last even liquids, and increasing emaciation is the result. So latent may the symptoms be, that the disease has sometimes been only discovered after death, though a tradition of a diagnosis once made by Sir Astley Cooper shows how the aspect and age of a person suffering from this disease may lead to its recognition by experienced observation, or by a shrewd guess.

In most cases, when a man of middle age, or older, comes complaining of inability to swallow food, he can indicate the exact spot where he feels it stop; and the negative result of examination of the other organs, with his progressive loss of flesh, makes the diagnosis easy. The way in which he eats, taking very small pieces of bread at a time, is very suggestive. Soft food, like bread and milk, can be eaten when ordinary diet is rejected, and it is often weeks or months later before difficulty is experienced with liquids. The regurgitation of a little blood with the food is very characteristic.

The only possible diagnosis between simple and malignant stricture depends upon the age of the patient and the history of previous injury.

The aspect of a patient in the advanced stage of the disease is very characteristic: the extreme marasmus, without jaundice or cyanosis or dyspnœa, the look of starvation and of long-endured suffering from thirst, the hollow eyes, dilated pupils, sunken cheeks and temples, and, above all, the deeply concave abdomen, feeling empty when examined, and allowing the aorta, the vertebræ, and the kidneys to be plainly discerned by the fingers. The skin is dry and rough, the condition known as pityriasis tabescentium. The bowels are confined for days or weeks. The urine is scanty, high coloured, and often offensive.

The cautious passage of a bougie is the only decisive proof of the nature of the disease, and also gives a criterion of its position and of the calibre of the tube. Hamburger's methods of diagnosis by auscultation deserve mention (Erlangen, 1871. See also Dr Allbutt's paper, 'Brit. Med. Jour.,' ii, 1875). Of less practical importance is the ingenious attempt to obtain a

view of the gullet by Waldenburg; his instrument is figured in the 'Berlin. klin. Wochenschrift,' 1870, p. 580.

"Tandem, post Tantali pœnas diu toleratas, lente marasmo contabescunt" (Boerhaave). Death, however, often occurs from intercurrent pneumonia or pleurisy. In one case under the writer's care the primary stricture was latent, and the first symptoms were pain, and afterwards paraplegia, caused by secondary cancer of the vertebræ.

The *duration* of these cases is very variable; it is to be counted by months, and some patients have lived on for two years, or perhaps for longer. The most rapid cases may prove fatal by perforation within a few weeks of the first appearance of dysphagia.

The *treatment* of stricture of the œsophagus is purely mechanical. When free from ulceration the stricture will usually be benefited by the frequent passage of a bougie; in fact, by the same method of dilatation as is adopted for stricture of the urethra. In cases of simple traumatic constriction this is sometimes an effectual cure; but even in cancerous strictures, so long as there is no ulceration, the occasional passage of an olive-shaped bougie frequently affords great relief. The utmost care and gentleness is essential, or fatal perforation may ensue. As Mr Bryant well puts it, when a patient complains of difficulty in passing food onwards down the gullet after the act of swallowing, and of its return into the mouth, the practitioner should first think of thoracic aneurysm, then of cancer, and then of some other kind of ulceration. The dread of such a catastrophe as perforating an aneurysmal sac, or thrusting a bougie into the pleural cavity, will, however, be the best safeguard against its occurrence. No force which an intelligent hand could use will perforate an intact mucous membrane. When perforation, or even ulceration, has already taken place, no one would willingly risk the passage of a tube. But when there is no evidence of ulceration the practice is defensible and beneficial.

The *sonde œsophagienne à demeure* is an instrument which is passed through a stricture, and then left in it as a catheter is left in a stricture of the urethra. It was advocated by Mons. Krishaber at the International Medical Congress of 1881 ('Transactions,' vol. ii, p. 392). He advises its passage through one of the nostrils rather than the mouth, and in proof of the tolerance of the instrument relates four cases in which the tube remained continuously *in situ* for 46, 126, 167, and 305 days respectively. Feeding, of course, takes place entirely through the hollow œsophageal sound. A similar plan has been carried out by Mr Symonds at Guy's Hospital with good success.

When obstruction has become complete, life may be preserved for a time by nutrient enemata. For this purpose small quantities of peptonised food without salt or alcohol are best employed; and in many cases raw eggs, beef-tea, and pancreatised milk, are well retained and absorbed. But often the rectum rejects the nutriment; after a time this result is almost sure to occur, and even in the most favourable cases the patient is insufficiently nourished. In acute cases of disease, or of injury or operation about the mouth and throat, or even while a gastric ulcer is given time and rest to heal, the treatment by rectal alimentation is most valuable. But when, as in stricture of the œsophagus, the disease is progressive, it is far better, so soon as occlusion occurs, and no liquids even can reach the stomach, for the operation of opening the stomach to be faced before the patient's strength and endurance have been exhausted.

The operation of gastrotomy, or *gastrostomy*,* was first performed by Sédillot in 1849, and was introduced into England by Mr Cooper Forster. It has since been amply justified by the long periods of life and comfort which it has afforded to patients who would otherwise have died by one of the most painful deaths—that from thirst. In a case brought before the International Congress in 1881 ('Trans.,' p. 456) the patient survived a year and a half, and in a second case (for malignant stricture), by the same surgeon, life was prolonged for 128 days. For similar cases see a paper by Dr Gross, jun., in the 'American Journal of Med. Sc.' for 1884.

The safety of the operation has been increased by the plan introduced by Mr Howse, of first cutting down to the stomach and fastening it by sutures to the abdominal walls, and a day or two afterwards, when adhesions have formed, opening it and introducing the cannula.

* Gastro-stomie (*i. e.* making a mouth in the stomach) was the French surgeon's original term. Mr Bryant follows Dr Pooley, of New York, in defining "gastrotomy" as opening the stomach for removal of a foreign body.

DISEASES OF THE STOMACH

ACUTE DYSPEPSIA.—*Acute catarrhal gastritis—Its symptoms, causes, histology, and treatment—Acute paralytic distension—Acute suppurative gastritis.*
CHRONIC DYSPEPSIA.—*Atonic and chronic catarrhal forms—Symptoms, causes, anatomy, diagnosis, and treatment.*
Gastralgia.—Anorexia, &c.—Eructation and Pyrosis—Vomiting, &c.—Hæmatemesis.
GASTRIC ULCER.—*Anatomy—Pathology—Ætiology—Symptoms—Event and duration—Treatment.*
CANCER OF THE STOMACH.—*Carcinoma of the pylorus and of the body of the stomach—Sarcoma—Colloid cancer—Symptoms—The tumour—Consequent dilatation—Gastro-colic fistula—Duration—Diagnosis—Treatment.*
Fibroid Induration of the Stomach—Gastric concretions.

IN health, as everyone knows (or has known) by experience, the process of digestion is unattended with any kind of sensation; we ought not to be conscious that we have stomachs. But under morbid conditions, it may be accompanied with unpleasant feelings, varying in degree from a slight sense of weight or discomfort up to agonising pain. These may be symptoms of severe disease of the stomach, or of some slight organic change, or of a mere functional derangement. Many of the less serious disorders of the stomach are commonly grouped together as dyspepsia, or indigestion.

1. ACUTE GASTRIC DYSPEPSIA.—It is not uncommon in persons who were before perfectly well, for the stomach to resent some particular meal, on account either of its quality or its quantity. Such cases must have come within the knowledge of every medical man, and the following may be taken as examples:—A schoolboy eats hastily a large quantity of grapes, skins and all; in a few hours he feels ill, and presently rejects the contents of his stomach; next morning he is well. A man, in the month of July, has the usual order of his meals disturbed by social engagements; he partakes twice in the same day of a variety of dishes, and probably eats much more than he really requires; he goes to bed with an uneasy feeling at the epigastrium, and in the night he vomits in surprising quantity the food that he had swallowed, almost unchanged, but dry, without the liquid with which he had washed it down; his discomfort is then at an end, and he falls asleep. Each of these patients brings up a little bile when his stomach has emptied itself of its accumulated contents. Formerly this fact would have been regarded as a sufficient ground for calling the complaint a "bilious" attack. But we now know that, in consequence of the antiperistaltic movements induced in the duodenum, bile enters the stomach whenever there is much vomiting, and is of course rejected in its turn.

To attacks like these the name of acute indigestion is applicable. But it is usual to describe under that name a somewhat different class of cases,

which are of longer duration. And since these are believed to depend upon a catarrhal inflammation of the gastric mucous membrane, the terms "acute indigestion" and "acute catarrh of the stomach" are commonly regarded as synonymous.

Acute gastric catarrh.—In different cases, the symptoms vary somewhat. In addition to a sense of weight and oppression at the epigastrium, which is common to all forms of dyspepsia, there is almost always actual pain. This is usually a dull aching, but sometimes it assumes a burning, stabbing, or griping character. One patient who came to me complained chiefly of a soreness at the left side of the sternum and about the left scapula; another felt more pain in the back than in the abdomen. In some cases, however, the epigastrium is tender, and it often feels full and tense. The patient is thirsty and eager for cold and acid drinks; but he has generally no appetite, and often experiences a loathing for all kinds of food. One man told me that his appetite was good, but that he felt afraid to eat anything, knowing what discomfort it would cause him. The breath is offensive. The tongue is often surprisingly foul and coated with a thick yellowish fur; this is one of the most characteristic indications of the disease. Nausea and retching are generally prominent symptoms, or patients may complain rather of the sour gases which come up by eructation. Partially digested food, with a very acid taste, is vomited; or a quantity of whitish mucus. Aching in the back and limbs, malaise, and depression of spirits are also complained of. There may be slight fever, but the skin is generally moist. In one case the attack began with shivering. A herpetic eruption sometimes breaks out upon the lips and chin.

An attack of this kind may last from a day or two to a week, or even longer; while if injudiciously treated, it may pass into chronic gastric catarrh.

Various *causes* of acute gastric catarrh are mentioned by writers. The cases most readily accounted for are those in which it follows the ingestion of some irritating substance. In one patient a severe and protracted attack came on the day after he had dined on venison and champagne. In infants improper food frequently causes attacks of this kind, which are commonly, but not always, complicated with diarrhœa. Prostration is a very marked feature in such cases, and death often results. Decomposing meat or vegetables, and shell-fish under certain conditions, are especially apt to give rise to acute catarrh of the stomach. The same effect is produced by the inhalation of dust or vapour given off from wall papers containing arsenic. The late Dr Wilson Fox mentions the case of a healthy child who, after sleeping in a room lined with such a paper, was seized with severe vomiting, and even brought up blood. The direct ingestion of irritants like arsenic and antimony, cause violent inflammation of the stomach, as described in works on toxicology: for this there is no analogy in the effects of ordinary disease.

Exposure to changes of temperature is said to be another cause. Dr Fox quotes from Guipon the case of a workman, who on several occasions was seized with vomiting and pain in the stomach after being exposed to the heat of a furnace. And he says that attacks of this kind are especially apt to occur in changeable weather, as in the spring and later autumn, when cold and high winds prevail, as well as during the severe heats of summer and early autumn. Epidemic influences are also mentioned. Dr Fox cites Chomel as having observed that gastric catarrh was frequent where cholera was raging, and had himself noticed the same thing in 1866. Lastly, it is

said to be a frequent complication of scarlatina, erysipelas, measles, diphtheria, smallpox, puerperal fever, &c. And at any rate every medical man is aware that each of these diseases is apt to be ushered in by repeated acts of vomiting, which, however, last only till the characteristic rash makes its appearance.

Since acute gastric catarrh has no tendency of itself to destroy life, one might fairly expect that its *morbid anatomy* would be unknown. But, half a century ago, a remarkable case occurred, in which an American named Alexis St Martin, after receiving a musket-shot wound in the left side, acquired a fistulous opening into his stomach, so that part of its mucous membrane was permanently exposed to view. The case was carefully investigated by Dr Beaumont, and he relates that deep red pimples sometimes appeared, which afterwards became filled with purulent matter; and at other times irregular circumscribed red patches, small aphthous crusts, and abrasions of the lining membrane, leaving the papillæ bare. These diseased appearances, when considerable, were attended with dryness of the mouth, furring of the tongue, thirst, and acceleration of the pulse, and the secretion of gastric juice was suspended, so that food remained undigested for twenty-four or forty-eight hours, or more; although liquids were absorbed as soon as they were swallowed. Mucus was also poured out by the surface of the stomach, and slight hæmorrhages sometimes occurred. Now, it is true that symptoms were by no means constantly present when the mucous membrane presented these appearances, but the case is nevertheless one of the utmost value, as showing that the stomach is susceptible of morbid changes which, if they could generally be seen, would arrest attention. For, in the *post-mortem* room, such changes can seldom be observed satisfactorily. After death, the lining membrane of the stomach is commonly acted upon by its contents, so that it becomes softened and pulpy, or may be entirely dissolved over a more or less extensive area. All the coats of the organ, indeed, may in this way be perforated, a large ragged aperture being left. As might be expected, this is generally at the back of the stomach, on which the contained matters rest while the body lies with its face upwards. Moreover, in the stomach, as in all other parts, congestion is very apt to subside after death, so that it can no longer be detected. We have, however, seen that in obstructive diseases of the heart the gastric mucous membrane is found intensely reddened and ecchymosed and lined with mucus; and great reddening is also seen in the bodies of those accustomed to drink spirits to excess.

It is probable that the changes which Dr Beaumont described would no longer be visible in a *post-mortem* examination. Writers, however, describe certain other appearances as characteristic of acute gastric catarrh—a milky opacity of the mucous membrane, which is soft, thick, and lacerable. Under the microscope, the secreting cells and the nuclei are swollen, irregularly distending the tubules; they are filled with granular matter, and in severe cases they often break down into a granular *débris*. Dr Cayley and Dr Fenwick have found in fluids from the stomach tube-casts comparable with those which are well known to occur in the urine in Bright's disease. A drawing of these is to be seen in the 47th volume of the 'Med-Chir. Transactions.' Infiltration of leucocytes between the tubules has been also observed and proves the existence of acute inflammation.

In addition to these changes, Dr Fox describes an increase in the size of the solitary follicles of the stomach, which appear as small white specks, scattered over its surface, and which sometimes ulcerate, forming little cup-

shaped depressions. The interstitial tissue between the tubules also becomes infiltrated with leucocytes.

Now, it is important to note that the cases in which these appearances have been found have not been simply cases of gastric catarrh; for these do not terminate fatally. The observations in question were made in the bodies of those who had died of scarlatina, diphtheria, pneumonia, or some other disease. They undoubtedly prove that the diseases in question are attended with morbid changes in the stomach, but not, it would seem, that these diseases are to be regarded as so many causes of acute gastric catarrh, in the clinical sense of that term. For, although in the instances in which the morbid changes were found after death, the patient may have been more or less sick and have had a furred tongue, yet it is probable that the catarrh of the stomach was only secondary and did not in any way modify the course of the disease.

Clinical varieties.—We may clinically recognise acute catarrhal gastritis under the following conditions. First the cases due to irritant *poisoning*, as by arsenic. The possibility of this as a cause must never be forgotten; it may be accidental, suicidal, or homicidal in origin, and it may be the result of a single dose or of gradual absorption suddenly producing its accumulated effect. Irritability of the eyes or actual ophthalmia, diarrhœa, and severe pain in addition to the vomiting are the most important symptoms. If no arsenic can be discovered in the vomited matters or digesta, Marsh's or Reinsch's process of identification will probably have already pointed to antimony, should that metal (as in the well-known Balham case) have been the cause of gastritis. Mercury would be almost certain to produce salivation at the same time. Of vegetable irritants, poisonous fungi are the most frequent causes of acute gastritis in adults, various berries in children. The poisonous symptoms from eating "tinned lobster" or other kinds of decomposing food merge into those of ordinary acute dyspepsia.

Secondly, apart from *gastritis ab ingestis* are the cases above mentioned of gastric catarrh which accompanies all *febrile* states, but is strictly symptomatic and secondary to the more important disease. Variola is perhaps, of all specific fevers, that in which the gastric symptoms are most constant and most severe, and next to it come scarlatina, pneumonia, and acute gout.

Thirdly, there are the cases of acute and subacute gastritis which constitute so marked a feature of the morbid anatomy of *cardiac* disease.

Lastly, there is a clinical group of cases in which somewhat severe epigastric pain is accompanied by vomiting, first of the contents of the stomach, then of a considerable quantity of mucus, and lastly, of bile. There is a thickly coated tongue, complete anorexia, considerable thirst, and, as a rule, constipation. These symptoms occur in elderly rather than in young patients and may come in the course of chronic Bright's disease (quite apart from uræmic vomiting) or other lingering malady. But they are most often seen in combination with bronchitis and myalgia. In such cases we find the heart and the kidneys unaffected and no evidence of more than moderate catarrh of the large bronchial tubes. The temperature is not raised, but the pulse is quick and irritable, and the "muscular rheumatism" severe, particularly in one or other shoulder and in the back and loins; or there is decided pleurodynia but no pleuritic rub or effusion. The patients appear to be more ill than any physical signs discovered account for; and one is anxious lest some primary lesion has been overlooked, or lest apoplexy or pneumonia is

impending. In most cases, however, with careful nursing, warmth, and strict diet, with brandy well diluted if the strength begins to fail, these cases do well.

The *diagnosis* of acute gastric catarrh is generally easy. Both Bamberger and Dr Wilson Fox observe that enteric fever in its early stage is the disease which is most likely to be confounded with it.

In the *treatment* of an attack of acute indigestion, the first thing to be attended to is the diet. In mild cases, complete abstinence for twenty-four hours often effects a cure. In severe and protracted cases, nutrient enemata may be used with advantage. If any nourishment is given by the mouth it should be milk in very small quantities, and diluted with soda-water or lime-water. Persons who cannot take milk must have veal or chicken broth. Sometimes this is less grateful than warm milk and water with a little cinnamon, or barley-water, or rice-water. As the symptoms subside, light farinaceous puddings may be allowed, but no solid meat, until they have entirely passed off. Unless there be great prostration the patient should abstain altogether from alcoholic liquids; champagne is not so suitable in this as in other forms of irritability of the stomach. If a stimulant appears absolutely necessary, brandy well diluted is the best.

In sucking infants, the quality of the milk must be carefully inquired into. Sometimes it is advisable to keep the child from the breast for a few hours, giving it only a little rice-water or very diluted milk at intervals. In infants brought up by hand, the substitution of asses' milk for cows' milk is sometimes effectual; but too often one can save the child only by engaging a wet nurse at once.

Hot fomentations or linseed poultices may be applied with advantage; or a hot-water bottle. Or spongiopiline may be used, soaked in hot water, squeezed out, and sprinkled with laudanum.

But the most important questions concern the administration of emetics and purgatives. Two cases of Sir Thomas Watson's may here be quoted. One was that of a person who had been taking large quantities of cream with his tea and coffee. After suffering for several days with severe gastric pain and disorder, he threw up a mass of hard curd like a small cream cheese, and he was at once completely relieved. In the other case a similar fit of indigestion terminated in the ejection of a mass of snuff. It is certain, therefore, that irritating matters may remain for a considerable time in the stomach. But, on the other hand, as Bamberger remarks, one must not trust too much to the circumstance that the patient continues to experience uneasy sensations, for these may continue long after their cause is removed, just as one goes on imagining that there is a foreign body in the eye long after it has been got rid of. Bamberger, indeed, gives a caution against the use of emetics or purgatives, which, he says, have caused catarrh of the stomach much more often than they have cured it. Dr Fox says that an emetic may be administered "when the presence of undigested food is indicated by cramp-like pain, nausea, ineffectual attempts to vomit, and faintness;" but adds that antimony and even mustard are to be avoided, and that ipecacuanha with large draughts of lukewarm water or of infusion of chamomile is the best emetic in these cases. He recommends, however, rather active purgatives; from three to eight grains of calomel followed by a black draught, or by castor-oil; or a dose of blue pill and compound colocynth pill, with a seidlitz powder afterwards. Dr Beaumont certainly found in the case of Alexis St Martin, already referred to, that the administration of

calomel (which he introduced into the stomach in rather large doses) was followed by a subsidence of morbid appearances in the gastric mucous membrane.*

But whatever may be said as to the propriety of beginning the treatment of a case of this kind with an emetic or with a purge, all writers are agreed that when once given it should not be repeated. Sedatives and antacids are then the remedies, and sucking small pieces of ice often gives much relief. For severe vomiting, dilute hydrocyanic acid is the best drug. In ordinary cases bismuth is particularly useful; ten grains of the subnitrate, with as much carbonate of soda and a little morphia, will often give immediate relief. Seltzer or Vichy water may be prescribed with much advantage, a pint or a pint and a half being given daily. Effervescing medicines are also useful. But the patient should not be allowed to take liquids in such quantities as to distend the stomach. During convalescence the remedies that will hereafter be recommended for chronic dyspepsia become applicable. But the use of the bitter tonics in protracted cases of acute catarrh of the stomach is said to "perpetuate a chronic inflammatory action."

Acute gastric distension.—In adults acute catarrh of the stomach is itself unattended with danger. But it may be a question whether certain very rare cases, in which rapidly fatal collapse occurs after moderate gastric symptoms have lasted a few days or a week or two, ought not to be regarded as due to the supervention of a further morbid state upon one of catarrhal inflammation. The author has himself seen two instances of the kind. They were described in the 'Guy's Hospital Reports' for 1872–73, under the name of "acute dilatation of the stomach," but a better title would perhaps be "acute paralytic distension." One case occurred in a man, aged thirty, who had for some time been in the hospital under Dr Owen Rees and was supposed to have incipient phthisis. He was seized with persistent vomiting; he passed no urine; and he gradually became collapsed. On examining him on the third day I found that the abdomen was retracted and that its walls were rigid. There was dulness above the pubes and half way up to the umbilicus. This might have been attributed to distension of the bladder, but a catheter had been passed and no urine could be obtained. Moreover, a splashing sound was obtained by manipulation of this region or of the iliac fossæ. He died the same afternoon. On *post-mortem* examination the stomach was found to fill the whole abdomen and to contain a large quantity of fluid. But when removed from the body it shrank back to about its natural size, showing only a number of

* We ought to be very careful in prescribing purgatives in cases of supposed acute gastric catarrh. I can never forget a case which I diagnosed as of this nature, and which proved to be one of acute suppurative peritonitis. A bank clerk felt poorly one day after having eaten some pears in the afternoon. In the night he woke up with epigastric pain and vomiting. A medical man was not sent for for two days, and when he came he gave a mild aperient. This operated, and a day or two later the sickness subsided. There was a little delirium about the third day. The pulse was at no period of the case over 100; the temperature ranged from 100° to 101°. I was asked to see him on the sixth day. He then appeared to be better, the sickness and pain had ceased, he had begun to take food again. The pulse was about 90, of fair volume; the temperature exactly 100°. Except that the countenance was sunken, and that the eyes were surrounded by deep brown rings, there appeared no reason for alarm, and I concluded that the attack had been one of acute indigestion, and that the patient was in a fair way to recover. Within twenty-four hours, however, he died, and it turned out that there was diffused peritonitis, set up by ulceration of the vermiform appendix.—C. H. F.

fine white striæ on its serous surface, apparently analogous to "lineæ gravidarum." The other case I saw in consultation with Mr Hooper, of Bermondsey. A man, aged twenty, had for a fortnight been suffering from abdominal pains and repeated vomiting. Two days before my visit the sickness ceased entirely, but he nevertheless became worse in all other respects. I found him with a sunken countenance, his eyes glassy and surrounded by deep rings of pigment, his breath nauseously sweet. His abdomen was generally distended, but the right hypochondrium was flat, and passing downwards and to the right above the navel a line could be seen which I recognised as indicating the upper border of the stomach. On manipulation of the lower part of the abdomen, I detected fluctuation and obtained a splashing sound. I therefore diagnosed dilatation of the stomach, and as soon as I could procure a stomach-pump I had a long tube passed down the œsophagus. A greenish fluid was ejected through it and by its side with considerable force. The pump was then connected with it, and no less than *seven pints* of the same fluid were removed by its means. The abdomen became deeply hollow while this was being done, The patient said that he felt much relieved, but he died four hours afterwards. The *post-mortem* examination showed, as I expected would be the case, that the stomach had returned to its natural size and form; but there was a sloughing abscess behind the duodenum, communicating with the bowel. This must doubtless have caused the patient's death under any circumstances. But it may reasonably be hoped that in an uncomplicated case like the former one the prompt use of the stomach-pump would afford a chance of saving the patient's life if an early diagnosis could be made. It seems clear that the cessation of vomiting is due to a paralytic state of the gastric muscular coat, comparable with that which occurs in the bladder in cases of retention of urine. It is not clear what is the origin of the large quantity of fluid which the stomach contains in these cases. Unless it is merely what has been swallowed, it must indicate an irritated or inflamed condition of the lining of the organ. Unfortunately the mucous membrane was not examined microscopically in either case.

The physical characters which indicate acute paralytic distension of the stomach during life are: (1) A rapidly increasing distension of the abdomen, which is unsymmetrical, the left hypochondrium being full while the right is comparatively flattened. (2) The presence of a surface marking which descends obliquely from the left hypochondrium towards the umbilicus and which corresponds with the lesser curvature of the stomach. This seems to move up and down each time the patient breathes. (3) Dulness and fluctuation in the pubic region with resonance over the front of the abdomen. (4) The production of a splashing sound on manipulation. It is to be observed, however, that in one of my cases the first two of these signs were absent.

Phlegmonous gastritis.—Another rapidly fatal disease of the stomach is acute diffused suppurative inflammation: a still more rare affection than paralytic distension. Bamberger mentions it in Virchow's series of handbooks (1855), but Rokitansky had previously described it as suppurative inflammation of the submucous connective tissue, and refers to older cases recorded by Monro, Lieutaud, and others, which were collected by Albers. Sometimes the process instead of being diffuse is confined to one or two spots, and may then be defined as submucous abscess of the stomach; such

abscesses, or "gastric carbuncles," as Virchow calls them, may burst into the cavity of the viscus.

Ackermann collected thirty cases, mostly puerperal. It has been compared to phlegmonous erysipelas or "pseudo-erysipelas" of German pathologists. Wilks and Moxon mention hepatic abscess as a result. A typical case of this rare disease is recorded by the author in the 'Path. Trans.,' vol. xxvi, p. 81.

The rarest form of all the anatomical forms of gastritis is *membranous* or "*croupous.*" It is sometimes the result of extension of diphtheria down the œsophagus, as observed by Jenner, but has also been seen in Bright's disease by Wilks, in phthisis by Fox, and apparently as an idiopathic affection by Niemeyer and Delafield. It is sometimes found associated with a similar membranous inflammation of the colon.

Sloughing, or, as German writers call it, "diphtheritic" gastritis, is described by Billard and Bednar as occasionally seen in newborn children.

Chronic Dyspepsia.—The most common disorders of the stomach are those which are chronic in their course; and these are usually referred to when indigestion is spoken of. Writers on gastric affections describe two forms of chronic dyspepsia; one they call "atonic," the other "chronic gastric catarrh," or "chronic inflammatory dyspepsia." It is admitted, however, that there is great difficulty in distinguishing between them.

To begin with the former,—a person who has *atonic dyspepsia* complains of a sense of weight and uneasiness after food, which may last for some hours, or even up to the next meal. The seat of these unpleasant sensations is usually the upper part of the abdomen; but sometimes, particularly by ladies, they are referred to the chest behind the sternum, so that a feeling of dyspnœa is experienced, or to the back between the shoulders, so that an impression is produced that food sticks in the gullet. There is rarely actual pain, unless it be as the result of flatulence. There is no tenderness of the abdomen, and pressure rather gives relief. Eructations of gas, and even of undigested food, are not uncommon; and these often cause an offensive or rancid taste, perhaps due to the formation of butyric acid. The appetite is generally deficient; there may be a distaste for food of all kinds, even though the want of it gives rise to a sense of exhaustion. Thirst is generally absent; indeed, the ingestion of fluids often seems to aggravate the symptoms. The tongue is broad, pale, and flabby; it is marked at its edges by the teeth, but is not often thickly furred. The bowels are always constipated. According to Trousseau this is due to the close sympathy between the stomach and the intestine, of which sympathy he gives an odd illustration in the fact that an enema used immediately after a meal may produce dyspepsia in a person unaccustomed to such a proceeding. Flatulent distension of the colon, with borborygmi, is also a frequent effect of dyspepsia.

Beside these symptoms, referable directly to the digestive organs, there is a general depression of power. The patient complains of a sense of languor and weariness of the limbs, especially after his meals. His spirits are depressed, morose, or irascible. His pulse is soft and compressible, slow when he is at rest, but quickened by any exertion. Palpitation is commonly complained of, and occasionally the heart's action becomes intermittent. The skin is moist and there is no fever. The complexion is often pallid, sallow, and muddy, but there is seldom marked anæmia or loss of flesh, except in very

chronic cases, and even then it is remarkable how well nourished the dyspeptic patient may be. The urine is copious and clear.

In *chronic gastric catarrh* the symptoms are in most respects very similar. Even in this affection the patient complains after his meals of weight and discomfort rather than pain; and tenderness of the abdomen, although often present in some degree, is not marked. On the other hand, thirst is a very prominent and distinctive symptom, especially in the intervals between meals. The patient often experiences a sense of exhaustion or of internal heat, which is relieved by drinking. The appetite is capricious. The breath is often offensive. A nasty taste in the mouth is often complained of, especially on first rising in the morning. The gums are spongy, red, and inclined to bleed. An excessive secretion of saliva is not uncommon, and at night it may escape from the mouth and wet the pillow. When this is the case, the dyspepsia is not infrequently complicated by excess in smoking. The tongue is often of a bright red colour, and raw-looking, the papillæ standing out as bright red points. Or this condition may exist only at its sides and tip, the rest of its surface being coated with a white or brownish fur of greater or less thickness. The lips are often dry and cracked. The mucous membrane of the pharynx may be granular and inflamed, and it may secrete a tenacious mucus, which is a source of great annoyance and discomfort to the patient.

Emaciation is said to be almost constant in chronic catarrh of the stomach. Slight febrile disturbance, preceded by rigor and malaise, is very common. Sometimes it occurs at night, and then it is often followed by copious perspirations during sleep. The urine is generally scanty and deposits lithates or oxalates; but sometimes it is alkaline and throws down phosphates. The expression is anxious and careworn.

Vomiting does not necessarily occur in chronic catarrh of the stomach any more than in atonic dyspepsia. Even nausea is generally not much complained of. In the dyspepsia of habitual drunkards, however, vomiting of mucus, especially in the morning, is one of the principal symptoms, and it is probable that this kind of vomiting is always an evidence of a catarrhal state of the gastric mucous membrane. Some caution appears to be required in concluding that vomited matters contain mucus from their naked-eye appearances only; Frerichs has shown that starchy substances are sometimes converted in the stomach into a tenacious glutinous material which may resemble mucus very closely.

In some dyspeptic patients in whom vomiting occurs with considerable frequency the ejecta are intensely sour. This is generally due, not to an over-secretion of acid by the stomach, but to the formation of lactic, butyric, and acetic acids by fermentation from the starchy and saccharine elements of the food. These acids are often developed with great rapidity, and in such large quantity, that when the patient vomits the throat burns, the teeth are set on edge, and the eyes smart, just as though strong acetic acid had been taken into the mouth. At the same time the sour smell of an acid, volatile at a low temperature, is diffused through the air. A further evidence that fermentation is the cause of the formation of acid in such cases is the fact that gas is evolved, which has been found to consist of a mixture of carbonic dioxide and a volatile hydrocarbon. Sulphuretted hydrogen is also found whenever eggs or other articles of diet containing sulphur have been eaten in considerable quantity.

The morbid appearances presented by the mucous membrane are described

as being different in atonic dyspepsia and in chronic gastric catarrh respectively. In the former the lining of the stomach is thin and transparent. In the latter it is almost always thickened and indurated. It may even be so tough that it can be stripped off the subjacent tissue in large pieces, or the submucous tissue may at the same time be white and fibrous, in which case there is increased rather than diminished difficulty in separating the coats. Near the pylorus the mucous membrane is often mammillated in cases of chronic gastric catarrh. But it is important to note that this condition is not necessarily the result of inflammation, for it may be found even in a healthy organ, being then caused by the contraction of the muscular layer which exists round the bases of the secreting glands. The most characteristic change in the appearance of the interior of the stomach in cases of chronic catarrh is, however, its ash-grey pigmentation. This, when closely examined, is seen to depend upon the presence of numerous minute specks scattered thickly over it. Under the microscope they are seen to consist of granules of pigment (doubtless originally derived from hæmatin), which are deposited in the connective tissue between the tubes, or even in the epithelial cells.

The gastric glands appear to present morbid changes in both forms of chronic indigestion. At least it seems to be certain that such changes are often observed in cases which in all other respects would come under the head of atonic dyspepsia. The secreting tubes are then found to be shrunken and wasted, and to have undergone fatty degeneration. They are often irregular in form and calibre. Their epithelium may have almost entirely disappeared, being represented only by granules and fat globules. In chronic gastric catarrh the changes in the epithelium appear to be the same. Cysts are not uncommonly found, which are probably the result of distension of parts of the tubes that were constricted off from the rest. Dr Wilson Fox observes that fatty degeneration is especially apt to affect groups of the glands one or two lines in diameter, which are then visible as small dead white spots in the mucous membrane. Dr Habershon, Dr Handfield Jones, and Dr Fenwick are the other observers who have worked most at this branch of morbid histology. Several of Dr Jones's cases were in persons advanced in life, so that the fatty changes in the tubes might be attributed to a process of senile degeneration. Dr Fox asserts that the gastric glands may be replaced by a fibro-nucleated tissue in atonic dyspepsia; but this appears hardly probable, and, as he himself remarks, simple atony is often itself the result of a pre-existent inflammation. Dr Fenwick has demonstrated the fact that when the secreting tubes are atrophied the digestive power of the dead mucous membrane is much less than under normal conditions. He found changes in the glands especially frequent in those who died of cancer of the breast, and he is disposed to regard this as one cause of the rapidly increasing anæmia which occurs in such cases.

Redness of the mucous membrane has often been mentioned as one of the appearances characteristic of catarrhal gastritis; but in many cases of this kind there is no redness, at least in the dead body. There are cases in which the most intense injection of the gastric mucous membrane is an almost constant appearance. But there are cases of chronic or subacute catarrh from obstructive *disease of the heart*. The stomach is then found lined with a thick layer of mucus; and after this is washed away, the surface is seen to be of the most vivid crimson colour, which may either affect a large part of its surface uniformly or occur in spots. Ecchymoses

are often present at the same time, and still more frequently they are simulated by small patches, due to arborescent injection of the branches of some minute vessel. When effusion of blood occurs into the submucous tissue it would seem that the gastric juice sometimes dissolves off the corresponding part of the mucous membrane. A little ulcer is the consequence, the floor of which is occupied by a layer of black coagulum. This process is known by the name of *hæmorrhagic erosion*. The greater part of the redness and congestion in these cases is due not to inflammatory but to passive congestion.

Another form of gastric catarrh in which the stomach is often found intensely reddened is that which results from alcoholic *intemperance*. An unskilled pathologist may easily be led to suspect the presence of an irritant poison in cases of this kind. Some time ago the author made an autopsy in a case of a young man who had suddenly died in a railway train early in the morning. There was no cause for his death discovered, but the stomach was most intensely reddened and ecchymosed. It was clearly ascertained that there had been no foul play, and there appeared to be little doubt that the abuse of stimulants had been the cause of the gastric irritation. As Wilks and Moxon remark, no mere redness and injection of the gastric mucous membrane is enough to prove the presence of an irritant poison. There must either be actual ulceration or chemical evidence of its presence.

The causes of atonic dyspepsia and of chronic gastric catarrh are very similar. Possibly disposition to either may be inherited. In some persons, age is an element in the causation of atonic dyspepsia; as life advances the digestive power of the stomach undergoes diminution. Hot seasons, relaxing climates, exhausting discharges, sedentary occupations, venereal excesses, prolonged anxiety of mind, long-continued depressing emotions, are also enumerated as causes of chronic dyspepsia. Except intemperance, they probably all act, if they act at all, by interfering with proper meals.

Other undoubted causes of indigestion are the habit of waiting too long between the meals, *imperfect mastication* of the food (the state of the teeth should always be looked after), taking too much fluid (especially cold water) with the meals, the *abuse of stimulants*, of condiments or of tea, excessive smoking, and taking bodily exertion or making mental efforts while digestion is going on.

The quality of the food may also be concerned. Dr Chambers cites the case of a poor needlewoman who had subsisted for a year on bread, potatoes, and tea, getting sometimes a little bacon but hardly ever other kinds of meat, and who suffered so much from dyspepsia that she dreaded to eat. In other cases indigestion always follows some particular article of diet, such as fatty matters or soups. Dr Chambers has attempted to describe "indigestion of vegetable food," "of albuminoid food," "of fatty food," and "of watery food" separately, but with no great success. One curious instance that he gives is that of a lady who from childhood had never been able to take roast beef without afterwards having heartburn. This he attributes to the fat which lies between the muscular bundles in stall-fed bullock's flesh. With regard to digestion, however, many idiosyncrasies are met with, of which it is not possible to give explanations, but which the physician must not ignore. Strawberries disagree with some persons, eggs with others, melted butter with many. Twice-cooked meats, potatoes, new bread, cauliflower, and jams or candied fruits are some of the most frequent sources of indigestion.

Although atonic dyspepsia sometimes attends senile decay, the ordinary flatulent dyspepsia with constipation which comes on after a meal is by far most frequent in young adults. It is very rare in children, but young men and unmarried girls are extremely liable to it, and it often continues through the first half of adult life. In women it is aggravated, or sometimes begins, about the menopause; but most men suffer less after forty or forty-five, and in old age, notwithstanding loss of teeth and sedentary habits, it usually disappears, so that, as Dr Michael Foster puts it, the old man who suffered martyrdom from dyspepsia throughout his active life, "now eats with the courage and the success of a boy." This undoubted fact seems to show that while indigestible food is certainly the occasion of dyspepsia, it is not its essential cause; and also that the histological changes described above do not exist or are not operative in the majority of cases. Irregular meals, fast eating, and the mental interest and anxiety which belong to active life, appear to spoil digestion by a kind of inhibition, either of the vaso-motor or of the secretory nerves, or possibly of the muscular walls of the stomach. The careless and unharassed periods of life,—youth before the toil and strife is begun, and age when both have passed, are the periods of unconscious and therefore happy digestion.

The *diagnosis* between atonic dyspepsia and chronic gastric catarrh is often unsatisfactory. But, after all, it is of little consequence, for with a little practical experience one probably learns to adjust one's remedial measures to the necessities of the case without attempting to draw a fine distinction between affections that are really near allied.

The diagnosis between chronic dyspepsia and the serious organic diseases of the stomach is of infinitely greater importance. It will best be discussed after they have been described; but we must remember that their early symptoms are very commonly attributed to mere indigestion, and that the possibility that one or other of them may develop itself must never be overlooked in any dyspeptic case that seems to be protracted or severe. Vomited matters should always be subjected to the closest scrutiny, and we must make it a rule to examine the patient in the recumbent posture, and with the surface of the abdomen exposed to view and manipulation*

The *prognosis* in dyspepsia depends chiefly on whether its causes are or are not capable of being completely removed, on the age and sex of the patient, and on the degree of severity and the duration of the symptoms. Proper treatment scarcely ever fails to give some relief; most patients are greatly benefited, but it seldom happens that a case of long standing is permanently and absolutely cured.

In regard to the *treatment* of these affections, it will be most convenient to take first chronic catarrh of the stomach. For this affection sedatives are the remedies that should be first employed, and especially bismuth. A drachm of Schacht's solution, or ten grains of the subnitrate, may be administered three times daily. At the same time small doses of some alkali, and of morphia may be given with great advantage; a very useful formula is the Mist. Bismuthi Sedativa of the Guy's pharmacopœia.†

* I can never forget the case of a gentlemen in whom, as soon as his shirt was raised, the existence of obstruction at the pylorus was indicated by the obvious peristaltic movements of a dilated stomach, but who assured me that his abdomen had not hitherto been examined, although he had been under the care of more than one specialist.—C. H. F.

† It must be kept in mind that the preparations of bismuth, like those of iron, give a black colour to the fæces.

Another valuable remedy in cases of this kind is magnesia; it may be prescribed with three-minim doses of dilute hydrocyanic acid, and equal parts of lime-water and cinnamon-water. Again, the oxide of zinc (in doses of two or three grains) is of service, especially when the cause of the gastric disorder is alcoholic intemperance. In long-standing cases, Dr Wilson Fox recommends the oxide of silver (in doses of one grain to two grains), alum (in doses of two to five grains), tannin or decoction of oak bark, and matico. This writer gives a caution against the use of purgatives in cases of chronic irritative dyspepsia; which he says, "are often aggravated by a persistence in their use." He advises that, when necessary, the action of the bowels should be solicited by the daily use of enemata of cold water, although he allows castor-oil in some cases, and in others the decoction of aloes, or the useful dinner pill containing aloes and extract of nux vomica.

Among the natural mineral waters of Great Britain, the most useful in cases of chronic gastric catarrh are said by Dr Fox to be those of Harrogate, Bath, and Leamington. Trousseau recommends Plombières, Vichy, and Bagnères de Bigorre, in France; as well as Pougues for anæmic patients.

In atonic dyspepsia, the treatment must be such as will tend to restore the functional activity of the stomach. The most important point of all is the regulation of the diet. The number of the meals, and the quantity of food taken at each meal, should be carefully adjusted, according to the idiosyncrasy of the patient. As a rule, there should be three meals daily; at two of which freshly-cooked meat should be eaten. Beef and mutton, poultry and game (but not hare or rabbit) are to be recommended; pork and veal, and salted or preserved meats are to be forbidden. Eggs agree well with some dyspeptic patients, whereas others are unable to take them. Vegetables must not be omitted from the dietary, but are to be eaten with caution. Potatoes should be taken sparingly, if at all; they must be well boiled and floury and not young; turnips, parsnips, and Jerusalem artichokes, in fact all solid roots are better avoided; but green vegetables, as spinach and asparagus, and in some cases onions, may be taken in moderation. Peas and beans are famous for causing flatulence and so are cauliflower and other cruciferæ. When vegetables are found to disagree, their place may be supplied by rice or macaroni, and such fruits as stewed prunes, grapes, and strawberries. New bread should never be eaten by persons who are subject to indigestion; "aerated bread" is in many cases to be preferred to that made with yeast. Sometimes it is of great importance to substitute toast or biscuits for bread. Light farinaceous puddings generally agree well with dyspeptic patients. Sugar, says Dr Fox, "may be used in moderation;" but Dr Chambers objects to its being taken "in such quantity as to cause a sweet taste." Everyone is agreed that lobsters and crabs, cheese, nuts, and pickles are to be strictly forbidden. A large amount of fluid should not be drunk at meals. Cocoa, or milk and water, may be used as substitutes for tea or coffee. Dr Fox advises that a moderate quantity of wine (sherry, claret, hock or champagne) should be taken twice daily; but the more usual practice is to limit the patient to weak brandy (or whisky) and water with his meals. Malt liquors are often injurious, but in certain cases a light, well-hopped and not effervescing bitter ale is an excellent stomachic; and some young women can take porter without discomfort who bear no other form of stimulant. Whatever causes flushing of the face after the meal is bad. Food is to be taken slowly, time being allowed for mastication and the due admixture of

saliva; and on this account it is advisable that the patient should have his meals in company with other persons.

General hygienic treatment is of great importance in cases of atonic dyspepsia. The patient should retire early to rest, and sleep in an airy room. During the day he should be in the open air whenever he can, and he should take as much exercise as possible, short of fatigue. Riding on horseback is often to be recommended, and for some patients yachting, or a sea voyage. A daily tepid bath is generally advisable, with the use of the hair glove or flesh brush. Cold, sponge, or shower baths are to be taken only when they are followed by good reaction.

Dr Fox speaks of Brighton as the best place for persons affected with this form of dyspepsia; next in order he places Scarborough, Dover, Folkestone, Margate, Eastbourne, Malvern, Tunbridge Wells. He also speaks highly of Ilfracombe.

The medicinal treatment of atonic dyspepsia consists mainly in the administration of remedies which tend to assist the process of digestion. Among these an important place belongs to the alkalies, which have been shown by Blondlot and Bernard to increase the secretion of gastric juice. One of the simple or aromatic bitters may often be given at the same time; such as chamomile, or calumba. A useful formula is one which contains carbonate of soda, rhubarb, and calumba. Gentian is more stimulating and sometimes too "strong." Nux vomica is exceedingly useful, but should not be taken continuously for more than six weeks. A good way to prescribe it is in the form of a pill, with a grain of sulphate of iron and a grain of extract of aloes or of compound rhubarb pill, to be taken just before one or more of the meals.

In many cases of atonic dyspepsia, and particularly in the later stages of the affection, the dilute mineral acids are very useful, especially the hydrochloric. In doses of ten or fifteen minims, properly diluted and taken with or after the meals, it prevents the sense of weight and oppression which would otherwise be experienced by the patient, and relieves acidity and flatulence arising from fermentation of the food. Trousseau speaks very highly of this remedy, which appears to be little known in France. He mentions that he learnt its use by sitting at a dinner next to a tourist who said that he never travelled without a little bottle of the acid, of which he took a few drops after each meal.

Pepsine, again, is often useful in cases of atonic dyspepsia. But Dr Pavy has shown that care is required in obtaining it at the druggists, since much of what is sold in London is devoid of any active properties. A plan which this physician has proposed, consists in digesting meat artificially (by means of an infusion of dried pig's stomach, acidulated with hydrochloric acid) before it is swallowed. The acid solution of pepsine in glycerine, taken in drachm or two drachm doses after a meal, is often useful in atonic flatulent dyspepsia.

Even the milder preparations of iron (although indicated when the patient is anæmic) sometimes disagree with dyspeptic patients, and, according to Dr Fox, the same is the case with quinine, particularly if it be given with sulphuric acid. When there is much flatulence, creosote, thymol, or carbolic acid are useful in the form of pills.

Children are, as before noticed, comparatively little liable to dyspepsia. So common, however, is the complaint, that even in them cases frequently call for treatment. These depend almost always on one of two causes:

either unsuitable food or general anæmia, in which the stomach shares. The first we see in the discomfort of an infant fed too soon on starchy food, or given material fit only for a grown child; and again in the acute gastritis of a schoolboy who has eaten too many apples or tarts or other "trash." The immediate remedy is an emetic, if the stomach has not unloaded itself, and the subsequent treatment is better choice of food.

The anæmic or atonic form of dyspepsia in children is seen in cases of rachitic, tubercular, or other kinds of marasmus, and is best treated by steel wine, fresh air, and sometimes by wine or malt liquor with meals.

Senile dyspepsia, when not the direct expression of organic disease of the stomach, is usually atonic, and benefited by wine and some such dinner pills as rhubarb or aloes with capsicum.

The dyspepsia of young women, associated with chlorosis and constipation, is cured by saline laxatives, aloes, and steel.

Alcoholic dyspepsia is recognised by the morning sickness or nausea, the frequent diarrhœa, the frontal headache, the acid eructations, and the temporary relief from a dram. The only treatment is entire abstinence from liquor, aided by the exhibition of alkalies and the milder bitter infusions.

The indigestion which comes of habitual excesses at the table, often combined with sedentary habits, is cured by saline aperients, with an occasional blue pill, more exercise, and more self-control. These are the cases which are so much benefited by the drinking the purgative waters of Carlsbad and other baths.

There are certain effects of gastric disorder, which although sometimes associated with the ordinary symptoms of dyspepsia, yet often occur by themselves, and may reach such a degree of intensity that they have to be regarded as independent affections.

Gastralgia.—Perhaps the most important of these is *pain*. This has been already mentioned as one of the symptoms of dyspepsia, particularly in its inflammatory form; but it also frequently occurs without any indication of impairment in the digestive process. Several names are applied to pains situated in the stomach, but unfortunately different writers use them in different senses. Cullen employed *cardialgia* for the less severe varieties which would commonly be called "heartburn" or "acidity," while he described as *gastrodynia* a more violent but also more transient pain, such as would usually be spoken of as "cramp" or "spasm" of the stomach. Most English writers follow Cullen in the use of these names, but the Germans employ them with meanings exactly reversed. *Gastralgia* is a term which is used chiefly by the French, and with a very wide range of application. The late Dr Anstie proposed to limit it to a particular kind of pain, namely, to that which comes on when the stomach is empty, half an hour or so before the time appointed for a meal.

This last kind of gastric pain is mentioned by all writers on the subject. It is often quickly relieved by even a small quantity of food. Sir Thomas Watson mentions the case of a clergyman whom he knew, and who was much harassed by its recurrence several times daily until he found by accident, after having tried a round of drugs, that eating a small biscuit would at once appease it. This writer also says that a drachm of the aromatic spirits of ammonia, or half a drachm of magnesia, will sometimes remove the pain

in a moment as if by magic. According to Dr Budd, the gastric pain which occurs when the stomach is empty is also accompanied by slowness of the pulse and by coldness of the surface of the body; the recumbent posture helps it away; hydrocyanic acid is the medicine which he recommends for it. On the other hand, Dr Anstie regarded it as a form of neuralgia, and he speaks of strychnia as the most valuable remedy for it. He prescribed five or ten minims of the tincture of nux vomica three times a day, or sometimes gave $\frac{1}{80}$ to $\frac{1}{50}$ of a grain of strychnia by subcutaneous injection. One case in which this remedy effected a cure was that of a patient who had actually attempted suicide on account of the agonizing pain which he endured. Arsenic was also recommended by Dr Leared for this affection. I have occasionally made trial of it, but without any marked success.

When gastric pain comes on soon *after* food it may be due to a variety of causes. Its diagnosis always requires great care, for the pain arising from organic disease of the stomach is usually of this kind. The strongest indication of the presence of such disease is the circumstance that the pain begins as soon as food is taken, and lasts until digestion is completed or until vomiting occurs. But, as we shall presently see, these characters may be wanting. On the other hand, very severe and protracted pain is sometimes complained of after every meal by nervous or hysterical persons, in whom there is no serious disease.

Another form of gastric pain—described by Abercrombie—begins from two to four hours after a meal, and lasts for several hours. This writer thought that its seat was in the duodenum. But Sir Thomas Watson points out that one can generally remove it by giving an alkali, or by letting the patient swallow a cup of warm tea. He therefore supposes that it is due to the continued secretion of gastric juice after the food has passed through the pylorus, and then adds that the onset of the pain may often be prevented by a small quantity of alkali in some aromatic water taken immediately after dinner. Trousseau speaks of this kind of pain as being often attended with a sense of sinking at the stomach, a craving appetite, and a great feeling of weakness. Constipation usually accompanies it, but sometimes diarrhœa, which he attributes to the circumstance that in cases of this kind the food is propelled into the duodenum before the digestive action of the stomach is completed. The treatment which Abercrombie found most useful consisted in giving two grains of sulphate of iron, with one grain of aloes and five grains of aromatic powder, three times daily. The sedative bismuth mixture is also useful in such cases.

But pain in the neighbourhood of the stomach, arising from disorder of that viscus, sometimes bears no relation whatever to the times at which the meals may be taken, or to the stage which the process of digestion may have reached. Pain in the left side, under the nipple and running round to the shoulder, has been repeatedly removed by bismuth and morphia, although not a single fact could be elicited to prove its having been due to gastric irritation. For this kind of pain—coming on at uncertain intervals in most violent paroxysms—Cullen reserved the name of "gastrodynia." Sir Thomas Watson says that it is often accompanied by a sensation of distension, much anxiety, and extreme restlessness. In females hysterical symptoms are frequently present, and the stomach is sometimes distended with enormous quantities of gas.

Gastrodynia may recur at irregular intervals for a very long period without appreciably affecting the general state of the patient's health. It

cannot in such cases be due to any active disease. Probably it is sometimes the indirect result of pathological changes long since come to an end. Bamberger speaks of the cicatrices of gastric ulcers as giving rise to paroxysmal attacks of pain, and it seems exceedingly probable that they may do so by irritating the filaments of nerves embedded in them. Not long ago the author made an autopsy in the case of a lady who had for years suffered from a pain in the back, which was supposed to be connected with an abscess near the sacrum she had when a child. All the parts in front of the spine—the aorta, the vena cava, and the nerves—were embedded in a dense mass of cicatricial fibrous tissue. This had doubtless been in some way the cause of the pain. In another case the destruction of a hydatid in the liver was followed by severe pain, evidently due to interference with nervous filaments during the contraction of the cyst.

On the other hand, a person may suffer from gastrodynia for many years and yet if one should have an opportunity of making an autopsy one may find nothing to account for it. Bamberger gives a case of this kind. It occurred in a powerful man, who for nine years had been subject to frequent attacks of the most violent pain in the stomach, lasting for days, or even weeks, and attended with great prostration and temporary loss of flesh. He died of acute phthisis. A slight dilatation of the stomach was the only morbid change in that organ.

Gastrodynia has to be distinguished from several other kinds of pain. In the first place pain arising in the colon may resemble it somewhat closely; this will be discussed when we deal with colic (*infra*, p. 368).

Again, according to Briquet, the abdominal muscles are often the seat of pain, without any affection of the subjacent parts. He lays stress on the circumstances that superficial tenderness is present, that the left recti and obliqui abdominis are the muscles principally affected, that not only their fleshy parts but also their tendinous attachments are concerned, and that dorsal pain and tenderness in the vertebral groove often exist at the same time. But we shall hereafter see that rigidity of the upper part of the rectus with tenderness is a very common effect of organic disease of the stomach, and there seems to be no reason why it should not occur even when the pain is of functional origin. In one very striking instance, a pain in the left hypochondrium, which had long resisted other treatment, was again and again removed by quinine and iron, and in that case the pain was probably myalgic. True myalgia of the abdominal muscles would be recognised by its being increased with movement of the body.

Lastly, pain situated in the epigastrium, over the stomach, may be continuous, and may last for a great length of time; but of this kind of pain one may say that it is comparatively seldom caused by organic disease of the stomach, and perhaps never by mere gastric disorder. There are doubtless many conditions that may give rise to it; but two in particular must always be borne in mind,—aneurysm of the abdominal aorta and incipient disease of the dorsal vertebræ. Some very striking instances in which epigastric pain was due to spinal disease are related by Hilton. One was that of a boy who for two months had been complaining of severe pain just above the pit of the stomach, and who used to walk about with his hands placed over that region, and with the body a little inclined forwards. It seemed as though he were suffering from irritation of some of the abdominal organs, and he had been treated on that supposition, but without much benefit. The pain was relieved when the boy lay down. Its seat was not

to one side of the body more than to the other. Disease was detected between the sixth and seventh dorsal vertebræ, and pressure on their spines excited the pain in front. He was kept in a recumbent posture for four or five months, at the end of which time he was completely cured. Another case was seen by Mr Hilton with Dr Addison. A Westminster boy had pain at the pit of the stomach and occasional vomiting. He was found to have disease between the same two vertebræ ; he too was easy when lying in bed. He was made to lie down uninterruptedly for two or three months, and from that time he got well.

True gastrodynia is often very intractable. Watson recommends the application of a mustard poultice to the epigastrium, and the administration of a carminative (such as a few drops of cajeput oil suspended in mucilage) or of sedatives, among which he assigns the chief place to hydrocyanic acid.

Alterations of the appetite are commonly due to gastric disorder. Loss of appetite (or *anorexia* as it is called*) may indeed be a symptom of almost any kind of disease. But it doubtless depends upon the loss of digestive power which accompanies so many morbid conditions, particularly those attended with fever. Beaumont found that when Alexis St Martin was feverish, the secretion of the stomach was diminished or suppressed, and food remained undigested for twenty-four or even forty-eight hours. Accordingly, patients suffering from acute diseases do not have regular meals, and are allowed only fluid nourishment in small quantities and at intervals which are often extremely short. In other persons the appetite may be greatly improved by the administration of bitters and mineral acids ; a pill of capsicum, nux vomica and gentian is very useful for this purpose ; but such medicines should never be prescribed until it has been ascertained that the anorexia is not due to disorder of the digestive organs, which must be first corrected.

Excessive appetite, on the other hand, is called *bulimia.** It is sometimes produced by the presence of worms in the intestines, and is a symptom in diabetes and in some diseases of the mesenteric glands. It has already been mentioned as accompanying one of the forms of pain in the stomach.

Lastly, *pica** is the name given to a perverted state of the appetite, in which substances that have no nutrient properties are swallowed greedily, or filthy matters, such as excrement, horsedung, &c. It occurs either as a variety of the "longings" of pregnant women, or in hysterical and half-demented girls.

Regurgitation.—Other symptoms of gastric disorder are those which consist in the regurgitation of gaseous or fluid matters upwards through the œsophagus. Of these the simplest form is *eructation* or belching. Generally gas alone arises, but sometimes a small portion of imperfectly digested food as well, which, however, is instantly carried back into the stomach. In rare cases a large part of the food is habitually brought back into the mouth, to be deliberately remasticated and swallowed a second time. Some years ago there was a patient of Dr Pavy's in Guy's Hospital who possessed this habit of ruminating. Dr Copland collected a number of cases of this kind, some

* Anorexia (*ἀνορεξία*), want of desire of food.—Bulimia (*βουλιμία*), or excessive hunger.—Pica is a modern translation of *κίσσα*, the jay (*Pica glandaria*), a word applied by the Greek physicians to the indiscriminate greediness of a morbid appetite.

of which came under his own observation. In one patient the rumination began in from fifteen minutes to an hour after almost every meal. Each bolus of food came up during an act of expiration; it had the same taste and flavour as when first swallowed; there was neither nausea nor pain; he masticated it a second time with pleasure. No treatment seems to help persons who ruminate, beyond advising them to eat very slowly and with moderation.

A somewhat analogous affection is what is called waterbrash or *pyrosis*.* This is not very common in England, but the people of Scotland are very liable to it, and still more so those of Norway, Sweden and Lapland. It is supposed to be caused in the Scotch by the oatmeal of which they eat so largely. Cullen, who was very familiar with pyrosis, described it as coming on usually in the morning when the stomach is empty. According to him, it begins with a severe pain at the pit of the stomach, which, after continuing for some time, brings on the eructation of a thin watery fluid in considerable quantity. The fluid is sometimes acid, but is often absolutely tasteless. The repetition of the eructation seems at length to give relief to the pain, and the attack is then at an end. It is apt to return more or less frequently for a considerable length of time. Cullen says that the complaint occurs chiefly among the lower classes, in women more often than in men, and between puberty and middle age rather than at any other period of life. He speaks of it as often unattended with any symptoms of dyspepsia.

In England pyrosis seldom appears as an independent malady, apart from other effects of gastric disorder. Sir Thomas Watson speaks of one remarkable case in which no less than three pints of a thin tasteless liquid were brought up every day. Writers differ with regard to the nature of this secretion. Some, with Dr Handfield Jones, regard pyrosis as a catarrhal affection of the gastric mucous membrane, analogous to bronchorrhœa. But the liquid is often ejected without any effort of vomiting; and, moreover, this sometimes occurs immediately after or even during a meal. Dr Chambers gives the case of a retired surgeon, who often had to leave the room at mealtimes, and would throw off as much as five or six ounces of frothy clear liquid. The contents of the gastric cavity never came up at the same time, although the ejection of the fluid sometimes made him retch. There seems, therefore, to be much probability in the opinion suggested by Dr Chambers that the fluid is really saliva, which trickles down the œsophagus, and, being arrested by spasm of the cardiac orifice, collects there until it gushes back into the mouth. Frerichs, indeed, is said to have detected sulpho-cyanide of potassium in liquid of this nature. One may perhaps object that Dr Pavy found it to have the power of digesting, but this might have been due to the admixture of a little gastric juice. The last-named observer showed that water injected into the stomach of an animal quickly becomes charged with pepsine, so that if the possession of digestive power should create any difficulty in the application of Dr Chambers's theory, this might be removed by supposing that the saliva had passed down into the stomach before being ejected. Dr Pavy, however, believes that the fluid is often secreted by the stomach.

The preparations of bismuth are very useful in the treatment of pyrosis. Sir Thomas Watson recommends opium combined with an astringent, as, for

* Etymologically, pyrosis ($\pi\tilde{\upsilon}\rho$ = fire) should mean the same as heartburn; but in England it is never used in this sense. In Greek, the word means inflammation.

instance, in the pulvis kino compositus. Dr Pavy is in the habit of prescribing the liquor opii sedativus in eight-minim doses, with an ounce of the compound infusion of gentian, three times a day.

Vomiting.—Another effect of gastric disorder is *emesis* or *vomitus*. This has already been mentioned as occurring in certain forms of dyspepsia, and we shall find that it is a principal symptom of the organic diseases of the stomach. But it may also be met with independently of these conditions.

The act of vomiting is usually preceded by a peculiar feeling, which is termed *nausea* (literally "sea-sickness"). In persons unaccustomed to being sick, this is accompanied with sensations of giddiness or faintness, coldness of the surface, pallor of the lips and face, and a small and feeble pulse. After some seconds, or a few minutes, retching occurs; and this is followed by the expulsion of the contents of the stomach. But it is by no means always the case that vomiting is preceded by such painful sensations and efforts. Some persons, chiefly women, are subject for years to occasional sickness, which in them may be attended with scarcely any discomfort. This kind of vomiting is especially apt to occur at night or in the early morning. So far it resembles the chronic vomiting of alcoholic dyspepsia, but it is a mistake to suppose that morning sickness is necessarily an indication of intemperance.

Sometimes habitual vomiting, independently of gastric pain and of any indication of dyspepsia, may reach such a point as to be alarming, and even dangerous to life. At Guy's Hospital we have had several cases of this kind. Some striking instances are related by Dr Chambers. In one of them the affection had been of three years' duration, and it was stated that the food was always returned, unchanged in appearance, within ten minutes after being swallowed. Another patient was said for five years to have hardly ever kept down a whole meal. This kind of sickness is almost confined to the female sex, and occurs chiefly in young women. It is very frequently associated with disorder of the menstrual functions; in one of Dr Chambers's cases, just quoted, it was attributed to a chill, by which the catamenia had been suppressed for several months. These patients, too, are often hysterical. They sometimes reject the food before there has been time for it to be swallowed. According to Dr Chambers the tendency to vomit can sometimes be checked by a strong effort of the will. Like other neuroses it is sometimes catching. But the most remarkable feature of many of these cases is that, although the vomiting is so constant, yet there is little or no loss of flesh. It is evident that a good deal of food must in reality be retained by the stomach.

In cases of this kind the *diagnosis* cannot be carried further than "irritability of the stomach," or "hysterical vomiting." But it is of extreme importance for the practitioner to remember that there are several organic diseases—affecting parts far distant from the stomach—of which a similar form of vomiting may be one of the symptoms. There are, indeed, few serious diseases in the course of which the stomach does not occasionally reject its contents. But the present point is that in certain diseases irritability of the stomach is often the earliest, and, for a time, the only indication that the patient is ill.

The most important of these is perhaps incipient pulmonary *phthisis*. The lungs should always be most carefully examined before one arrives at the conclusion that habitual vomiting is merely due to functional disturbance

of the stomach ; and, if there be any other ground for thinking that tubercular disease is likely to develop itself, a guarded opinion should be given, even though there may be no discoverable stethoscopical evidence of its presence (cf. *supra*, p. 212). Another morbid state, of which vomiting is a principal symptom, is that due to *Addison's disease* of the suprarenal capsules. Again, the possibility of the presence of *cerebral disease* must never be overlooked ; in some cases of abscess of the brain there are few other symptoms than sickness. According to Romberg, the vomiting which accompanies affections of the brain is characterised by the absence of nausea and of retching, and by its occurring when the head is moved, as in swinging, shaking, or stooping, or in suddenly rising ; it also occurs when the patient is erect rather than when he is recumbent. Affections of the spine seldom cause gastric disturbance. In female patients the possibility of *pregnancy* must never be forgotten, particularly if the vomiting should be only of a few weeks' duration. All these varieties of sickness are commonly attended with constipation.

When chronic vomiting and diarrhœa occur in the same case, the presence of some form of *Bright's disease* of the kidneys should always be suspected. In one instance the observance of this rule led to a correct diagnosis which might not otherwise have been reached, for the quantity of albumen in the urine was very small, and would probably have been overlooked.

Another possible cause for the existence of vomiting and diarrhœa in the same patient is chronic *poisoning* by small doses of arsenic or antimony, or other irritant poison.

The diagnosis of "irritability of stomach" having been arrived at, its *treatment* is often highly successful. In many cases the best plan is to give the stomach entire rest for two or three weeks, the patient being fed solely by enemata. Or minute quantities of milk may be administered by mouth, as in the well-known case related by Dr William Hunter. A boy was brought to him in a state of the most extreme emaciation, who vomited up almost everything that he swallowed, in spite of the treatment of three very eminent physicians. Dr Hunter recommended that only a single spoonful of milk should be given at a time. The boy was never sick afterwards ; he gradually became able to take more and more nourishment, and he ultimately recovered entirely. A most graphic and interesting account of the case is given in the sixth volume of Dr Hunter's 'Medical Observations and Inquiries.'

The application of a blister to the epigastrium is often highly serviceable in cases of this kind, particularly if it be kept open by an Albespeyres' plaster, or in some other way. Sedatives may often be prescribed with advantage, but particularly morphia or opium. Occasionally the oxalate of cerium (in doses of one to two grains) has succeeded when bismuth and hydrocyanic acid had failed. Creasote is sometimes serviceable. In the cases related by Dr Chambers a daily shower-bath appeared to do much towards the restoration of vigour of mind and body, and in some of them the preparations of valerian were useful. Lately two, three, or four drops of tincture of iodine in a teaspoonful of water has become a favourite remedy in our wards, and it certainly appears sometimes to be efficacious.

Other symptoms.—Gastric disorder may manifest itself by producing reflected disturbance of other parts. Thus *palpitation* of the heart and *cough* are occasionally produced by overloading the stomach.

A somewhat similar affection is hiccough or *singultus*. This is due to a sudden spasmodic contraction of the diaphragm, repeated at more or less regular intervals, and attended with a clicking sound which is caused by the abrupt passage of air through the glottis. Its recurrence can often be stopped by holding the breath. Hiccough is not usually a matter of any consequence, and lasts only a few minutes, or at most an hour or two. But in dangerous illnesses it sometimes continues for days together, and it may then exhaust the patient and appear to be the immediate cause of death.

Dr Edward Liveing mentions that he had a man past middle age under his care in whom hiccough occurred in paroxysms of twelve hours' duration about twice a week for four years; and he quotes a case of a little girl of twelve, who for nearly three years was subject to fits of violent hiccough, even during sleep, which lasted from ten minutes to an hour and returned three or four times during a day and night. She was cured after taking turpentine.

In all probability hiccough is due to the presence in the stomach of food which is incapable of being digested.

No one can make frequent autopsies without observing how often the stomach contains a pint or more of brandy and egg mixture or of some similar substance, which must include all that had been given by the nurses for some hours before death. In one case of fever, as the end was approaching, the relatives began to flatter themselves with vain hopes that the patient would recover because he took all his nourishment; but it evidently was not absorbed, for a splashing sound could be constantly produced by manipulating the upper part of the abdomen.

HÆMATEMESIS.—As the result of gastric catarrh, matters of various kinds are sometimes ejected from the stomach, but vomiting of blood, or *hæmatemesis*, is very rare except as the effect of acute irritant poisons or as the result of some organic lesion. This, therefore, appears to be the most fitting place to discuss it before we pass on to describe the organic diseases of the stomach.

Diagnosis.—In investigating a case in which blood is said to have been vomited, one must, in the first place, make sure that the blood was really rejected from the stomach. Strange as it may appear, there is often considerable difficulty in distinguishing between hæmoptysis and hæmatemesis, and particularly so when one has to rely upon the statements of the patient and does not see the blood actually brought up. When blood escapes into the air-passages in large quantities it may issue from the mouth in gushes; while some of it may pass back into the pharynx and there excite retching and vomiting. Indeed, a portion of the blood may run down the œsophagus. In a recent autopsy on a young child who died of hæmoptysis, we found an ounce of coagulated blood in the stomach. On the other hand, when sudden and profuse hæmatemesis occurs, the blood may irritate the larynx in passing over it, and so provoke a paroxysm of cough. The patient's statements, therefore, may afford a very unsafe basis for distinguishing between vomiting of blood and hæmoptysis; the diagnosis must be founded upon a consideration of the conditions under which these two forms of hæmorrhage severally occur.

The stomach is a large cavity into which a great quantity of blood may ooze before it excites vomiting. Hence hæmatemesis is commonly preceded by the characteristic symptoms of hæmorrhage—pallor of face, dimness

of vision, giddiness, or fainting. On the other hand, hæmoptysis occurs quite suddenly, the patient finding the hot blood in his mouth or feeling a tickling in his throat, or a sensation as of bubbling in the chest, immediately before he begins to cough up the blood.

Again, after hæmatemesis, any blood that may be left in the stomach passes downwards, and (if not completely digested) it is ultimately discharged from the bowels. But when any part of the air-passages is the seat of the hæmorrhage, the blood that is almost necessarily sucked into the smaller bronchial tubes is afterwards got rid of by expectoration, and the sputa remain discoloured by it for several hours or even for some days.

Again, the appearance of the blood after its ejection is generally different in cases of hæmoptysis and of hæmatemesis respectively. In the former it is usually frothy from admixture of air, it has an alkaline reaction, and it is of a bright red or "arterial" hue. In the latter it is acid and darker coloured, or brown, from being mixed with the gastric juice. Dr Chambers mentions a case in which the application of test-paper to a woman's clothes over which she had thrown up blood, showed that it had come from her stomach. When the hæmorrhage is very profuse—and particularly if a large artery be opened—blood rejected from the stomach may be fluid and of a scarlet colour. But whenever it is retained for any length of time in the cavity before being vomited, it undergoes a peculiar change. It often coagulates, and sometimes solid masses of it are rejected which are so tough as almost to choke the patient. More frequently the blood, whether clotted or not, is acted on by the gastric juice, the acid of which decomposes the hæmoglobin and produces hæmatin. The presence of comparatively small quantities of blood in vomited matters thus gives them an appearance like that of coffee grounds; while pure blood altered in this way resembles a black pudding or has more or less the appearance of tar.

Blood which passes from the stomach into the intestines is found in the stools altered in exactly the same way. It is often perfectly black or (as it is called) "tarry." Evacuations having this character were supposed to consist of "black bile" by the older writers, who described them as characteristic of a special disease which they called *melæna*. But it has long been known that this affection is one of the results of hæmorrhage into the stomach, being, in fact, almost as certain a sign as hæmatemesis. Addison long ago taught that when blood having this black colour was discharged from the bowels the source of the bleeding was always the stomach. An exception to this rule ought perhaps to be made for the duodenum, although it is doubtful whether the blood discharged from an ulcer in that part of the bowel would be sufficiently long in contact with the gastric juice to undergo the peculiar change. But when the hæmorrhage occurs from the rest of the intestine, the blood is always more or less distinctly red.

The dark appearance which the fæcal evacuations so constantly present when a patient has been taking a preparation of iron or bismuth medicinally must be distinguished from that due to blood. It is of a more slaty hue, and the fæces are usually remarkably dry. In a doubtful case one might apply the guaiacum test or that of the spectroscope.*

It is important to note that hæmorrhage into the stomach may, and often does, occur without any blood being vomited. Thus a case was

* The detection of iron in an acid solution of the dark pigment by yellow prussiate of potash would distinguish melæna from the effect of bismuth. On one occasion a hysterical or malingering schoolboy, whose case is referred to in the first volume (p. 842), said

observed at Guy's Hospital in which a patient (who had once before brought up a large quantity of blood) became blanched, called out that she was dying, and did actually die in twenty-five minutes, after a convulsive seizure. The stomach was full of clotted blood. Other instances of a similar kind have occurred at the hospital, and one such is mentioned by Sir Thomas Watson. Doubtless therefore it is a frequent occurrence for small quantities of blood to escape into the gastric cavity without exciting vomiting. One can, indeed, see no reason why small or even moderate quantities should be rejected unless there be also some other condition making the stomach irritable. Up to a certain point, it is probable that blood undergoes digestion and is absorbed before it has passed down the whole length of the intestine ; so that the stools may themselves fail to afford evidence of the hæmorrhage. Still it is very important that whenever a patient's symptoms can possibly be attributed to bleeding into the stomach, the fæces should be most carefully examined.

A case in point is given by Dr Chambers. A woman, aged thirty-three, who had suffered from well-marked symptoms of gastric disease, grew rapidly weaker and paler, and her tongue became dry and furred, "as in hæmorrhagic fever," to use his expression. It was long suspected that she was passing blood, but she constantly denied that this was the case. She was made an in-patient, and then it was discovered that blood came from the bowels every time they acted.

On the other hand, true hæmatemesis may occur without there being in reality any hæmorrhage from the vessels of the stomach ; so that in investigating a case in which blood is vomited, one must in the first place determine whether the blood comes from the gastric blood-vessels or from some other source. Attacks of hæmatemesis have sometimes been (so to speak) manufactured, the patients having first secretly drunk the blood which they afterwards vomited in the presence of others. Sir Thomas Watson mentions two cases of this kind. Again, when blood escapes into the back of the mouth or into the pharynx it is often swallowed by the patient unconsciously, particularly during sleep ; and the first indication of the fact may be the occurrence of profuse hæmatemesis.

But in most cases of hæmatemesis the blood really comes from blood-vessels which run in or beneath the mucous membrane of the stomach ; and we have now to consider what are the causes that may give rise to this form of hæmorrhage.

Causes.—In the first place gastric hæmorrhage may occur in certain *general diseases*, as a consequence (it is supposed) of changes in the blood itself : for example scurvy, purpura, malignant smallpox, yellow fever, acute yellow atrophy of the liver. Then, again, it may be a subordinate symptom of organic *disease of the heart*, or of the high arterial tension produced by chronic *Bright's disease ;* but in the latter case the arteries are often morbidly brittle as well. It is said, too, that hæmatemesis may be caused by *disease of the spleen.* Sir Thomas Watson quotes from Latour the case of a man whose spleen was immensely enlarged, as the result of obstinate ague. Latour foretold that hæmorrhage from the stomach would occur, and his prognosis was justified by the result. The bleeding recurred several times ; in a month

that he had passed blood from the bowels, and produced very dark motions. There was no evidence of disease, and it was found that the scybala were of natural colour, except on the surface. The stools gave no iron reaction, and the colour was found to be due to a silver solution which had been poured into the nightstool from a bottle used in photography

the spleen could no longer be felt, and the patient had good health for twenty-five years afterwards. Sir Thomas Watson thinks that he has more than once seen the spleen diminish in size in proportion as blood was poured out of the stomach. We may doubt, however, whether in his cases the spleen was primarily diseased, and whether the enlargement of that organ and the hæmatemesis were not joint results of portal congestion, itself due to hepatic disorder.

The two principal organic diseases of the stomach—simple ulcer and cancer—are each frequently attended with hæmatemesis. In cases of *cancer*, however, it very rarely happens that any considerable quantity of blood is vomited until the disease has reached an advanced stage, or has, at any rate, declared itself by well-marked symptoms. On the other hand, a *simple ulcer* of the stomach is sometimes latent up to the time when a large hæmorrhage takes place; the patient having either had no symptoms previously, or only such slight indications of gastric disorder as appeared to be of no consequence. Latent gastric ulcer, however, seems to be of decidedly less frequent occurrence than latent intestinal ulcer: perforation of the peritoneum from the latter cause may take place unexpectedly in persons who had appeared to be in good health. Perforation of a gastric ulcer is particularly common in young subjects; sudden large hæmorrhage seems rather to occur in persons advanced in years. This kind of hæmatemesis is very seldom immediately fatal. In some cases it returns at intervals of a few hours, and the patient lives five or six days. Sometimes no fresh bleeding occurs for several weeks or even months. The ulcer generally presents certain special characters which will be fully described further on, but Dr Murchison has placed on record two cases, in each of which it was a mere pore-like aperture leading directly into a large branch of artery. A preparation of a similar kind is contained in Guy's Hospital museum. It is worthy of note that in all these instances the seat of the ulcer was not the lesser curvature, but the cardiac pouch of the stomach. A small ulcer of this kind might easily be overlooked, and it is possible that such may have been the explanation of some of those cases, in which a *post-mortem* examination has failed to reveal the source of the hæmorrhage. Some time ago a carman, aged thirty-two, who was in the hospital for gout and albuminuria, died suddenly from hæmatemesis. The stomach was full of blood, but we could not discover from what part of the stomach it had come. The vessels of the stomach were not found much congested, for the hæmorrhage had doubtless emptied them. The liver appeared to be quite healthy.

Vomiting of blood is very frequent in cases of *cirrhosis of the liver*, and it is often the earliest symptom. Many cases in which ascites has already appeared terminate by the sudden and fatal hæmatemesis.

All systematic writers mention among the varieties of hæmatemesis one in which the effusion of blood is *vicarious* to the menstrual flow. Sir Thomas Watson relates a case of this kind which came within the knowledge of the late Dr Latham. A girl about the age of fourteen became the subject of hæmatemesis, recurring at monthly periods. She married without ever having menstruated, and became pregnant; the hæmatemesis then ceased, and did not return until she had been confined, and had suckled and weaned her infant. Sir Thomas Watson also quotes Mr North as having met with two instances in which suppressed menstruation was followed by repeated and at length fatal hæmorrhage. I have searched in vain for the original record of these two cases, so that I cannot tell

whether the presence of a gastric ulcer was disproved by an autopsy. All modern clinical observers are agreed that vicarious hæmatemesis is, to say the least, exceedingly rare. And probably many cases which were formerly supposed to be of this kind would now be explained differently.

The occurrence of profuse hæmatemesis in a person who presents no other well-marked symptoms of disease is then generally the result either of a latent ulcer, or of congestion of the organ; this last being itself often, but not always, dependent upon latent cirrhosis of the liver. The next question is whether these two conditions can be distinguished from one another. And this must be answered in the negative. Both Dr Murchison's cases, for instance, occurred in persons who had been intemperate; one of them was a plethoric woman, aged fifty, the other a soldier aged twenty-eight, whose liver was cirrhosed. It might well have been thought that in both instances the hæmorrhage was due to mere congestion.

Another cause for hæmatemesis, independent of visceral disease, is atheroma of the blood-vessels. In one case it followed profuse epistaxis and was itself the precursor of cerebral hæmorrhage.

Treatment.—The question of diagnosis is one of no little importance, for the practice recommended in cases of hæmatemesis from congestion is hard purging. Sir Thomas Watson directs that five grains of calomel should be given every night and a black dose every morning, till the stools lose their pitchy colour. And he says that he has pursued this plan with perfect success, even when the patient had been blanched by previous hæmorrhages, and when the pulse was feeble and irregular. But it is evident that such treatment must do harm if the blood has come from an ulcerated artery; and, on the other hand, when the case is one of mere venous congestion, the occurrence of hæmorrhage shows that the vessels are on the way to relieve themselves, even if they have not already done so. The administration of astringents at this stage might indeed be injurious. The best course, therefore, is to wait a few hours, or even for a day or two, until we see whether the hæmorrhage returns. Even if the patient should pass several tarry evacuations in succession, this is not a proof that bleeding has occurred more than once, for they may all have arisen from a single hæmorrhage.

But whenever repeated attacks of hæmorrhage occur in a person already blanched by loss of blood, it is clear that they depend on something more than portal congestion; astringents must then be used, and acetate of lead may be prescribed in doses of three or four grains, with a quarter of a grain of opium, every two or three hours. Some writers have spoken highly of oil of turpentine, twenty or thirty minims of which are given every four or six hours. Other valuable styptics are gallic acid and dilute sulphuric acid. Of the former ten grains may be administered every two or three hours, or even oftener: of the latter ten or twenty minims. Dr Chambers records a case in which it appeared certain that a gradual oozing of blood, causing continued melæna, was arrested by dilute sulphuric acid, with Battley's solution of opium.* The patient may suck small pieces of ice, but he should be allowed to take scarcely anything into his stomach. Starvation is indeed the cardinal point in the treatment of hæmatemesis, nutrient enemata being given if

* It is doubtful whether sulphuric acid can act as an astringent in cases of diarrhœa, of hæmoptysis, or of profuse sweating; for in medicinal doses it must always be neutralized by the bile and pancreatic juice, or, if absorbed from the stomach, by the soda of the blood. It may be of service when applied directly to the mucous membrane of the stomach; even in cases of hæmatemesis, however, it is rarely prescribed without opium, catechu, or some other astringent, so that it is difficult to judge of its efficacy.

support appears necessary. If the hæmorrhage is profuse the patient's head should of course be kept low. The application of a bladder partially filled with ice on the epigastrium is often serviceable. Brinton recommends that it should be removed when it has been kept on for a few minutes and that it should be reapplied from time to time; but no harm has arisen when it has been left in its place for several hours continuously.

GASTRIC ULCER.*—We have seen that vomiting, gastric pain, and hæmatemesis may each or all be symptoms of ulcer of the stomach.

This disease may occur under a variety of conditions. Hæmorrhagic erosions in cardiac disease have been already mentioned (pp. 327–8), and both in enteric fever and in diphtheria the surface of the stomach sometimes, though very rarely, presents a number of small ulcers.

In these cases, the ulceration of the stomach is due to an acute process, but the organ sometimes presents a large number of chronic ulcers. A striking instance of this kind occurred some years ago at Guy's Hospital in a man of colour, aged thirty, who had recently come from the Southern States of America. He died of thoracic aneurysm, after an illness of twelve months' duration, his principal symptom having been vomiting of his food about an hour after its ingestion. Dr Moxon found that almost the whole of the stomach was diseased. There were numerous recent ulcers with raised irregular edges, and there were also many thick, puckered cicatrices.

Anatomy.—The affection now to be described differs altogether from this. It is limited to a small part of the surface of the stomach. Most frequently there is only a single ulcer; sometimes there are two; rarely, three, or four, or even five. Rokitansky found the ulcer solitary in sixty-two out of seventy-nine cases, and in twelve of the rest there were two. When there are more than one, they differ in size and in other characters, so as to prove that they commenced at different times.

One of the most remarkable circumstances connected with gastric ulcers is that their seat is, in the great majority of cases, along the lesser curvature of the stomach. Sometimes an ulcer lies across the curvature itself; more often it is situated either in the anterior or posterior wall, but almost always close to that line. According to Brinton, ulcers are found on the posterior surface of the stomach eight times as often as on its anterior surface. But our *post-mortem* records at Guy's Hospital by no means bear out this statement. Not infrequently two ulcers are found in the same stomach exactly opposite one another, one on each surface. Since they generally appear to be of different dates, it has been supposed that one of them has been set up secondarily to the other, as the result of its coming into contact with the opposed surface of mucous membrane. Sometimes the pylorus is the seat of the ulcer, sometimes the cardiac pouch. Sometimes, lastly, an affection exactly similar occurs in the first portion of the duodenum; this belongs, of course, to diseases of the intestines; but it is convenient to take it with ulcer of the stomach.

A gastric ulcer has a sharply-defined edge. This at first is entirely free from thickening. Its floor may be formed by the muscular coat, or the ulceration may extend through this, forming a pit, which is always considerably smaller than that in the mucous membrane, and at the bottom of which the peritoneum is visible. Very frequently, unless a different process should be started, the serous coat in its turn becomes attacked; a minute

* *Synonyms.*—Simple chronic ulcer of Cruveilhier—Perforating ulcer of Rokitansky.

yellow slough forms in it; and the detachment of this allows the contents of the stomach to escape into the general peritoneal cavity, setting up fatal diffused peritonitis. The ulcer is found in the *post-mortem* room to have the form of a flattened cone, the base corresponding with the mucous surface of the stomach. It is often described as a "punched-out" ulcer, on account of its regular circular form, and of the evenness of its margin. The acute perforating ulcer is always of small size, seldom larger than a sixpenny-piece. Not infrequently, instead of eating its way into the peritoneal cavity, it erodes some large vessel in passing through the coats of the stomach, and thus gives rise to hæmatemesis.

So far there is nothing in the characters of a gastric ulcer different from those of a similar affection occurring in other parts of the alimentary canal as is sometimes (though rarely) the case. Thus Mr Flower has recorded an instance in which a small round ulcer developed itself in the œsophagus, and passed straight through into the descending aorta. In the duodenum similar ulcers occasionally follow severe burns of the skin, as Mr Curling first pointed out; and isolated cases have been published of similar ulcers in the jejunum, the ileum, and the descending colon.

But in the great majority of cases an ulcer of the stomach goes on to acquire further characters which are almost peculiar to it. Its edge, although still perfectly even and regular, becomes slightly thickened. For a little distance beyond it all the coats of the organ are matted together. These changes evidently depend on the occurrence of a chronic inflammatory process. The peritoneum covering the floor of the ulcer also becomes thickened and opaque, and adherent to whatever part may be opposed to it.

From the locality affected by the chronic gastric ulcer, its floor almost always becomes attached either to the under surface of the left lobe of the liver, if it be in the anterior wall of the stomach, or to the pancreas and the adjacent connective tissue and vessels, if it be on the posterior wall. Thus perforation of the serous cavity is for the time prevented, while the ulcer gradually increases in size. Its growth in different directions is not always uniform, and thus it often loses its circular shape and becomes oval or irregular in form. This last, however, is frequently due rather to the circumstance that when two or more ulcers are present they come into contact as they grow larger, and finally coalesce. As before mentioned, two ulcers are often found just opposite to one another, one on each side of the lesser curvature; these, when they run together, give rise to a single sore, of dumb-bell shape. The size to which an ulcer of the stomach attains is sometimes very considerable; the 'Pathological Transactions' contain a record of one which measured five and a half by three inches. While thus expanding in circumference, gastric ulcers also increase in depth. The peritoneum is gradually worn through where it is adherent, and the surface of the pancreas or of the liver comes to form part of the wall of the stomach. This at first takes place only at a small spot, but the area of adhesion and that of destruction gradually become more extensive. Thus the greater part of the pancreas may in time become exposed in the floor of the ulcer; it is covered only by a thin film of connective tissue, through which its lobulated character can be plainly identified.

In comparatively rare cases the floor of an ulcer in the anterior wall of the stomach becomes adherent, not to the liver, but to the abdominal walls, and these may in time be perforated, so that a gastro-cutaneous fistula is formed. Murchison collected twenty-five cases of this kind ('Med.-

Chir. Trans.,' vol. xli), of which, however, only twelve were originally instances of simple gastric ulcer; six of them were cases of cancer, seven cases in which the penetration of the walls of the stomach was due to wounds or injuries of the corresponding part of the surface of the abdomen.* A gastro-cutaneous fistula may remain open for several years. It sometimes closes of its own accord.

But, as might be expected, this process of adhesion of the floor of the ulcer to different parts is by no means unattended with risk. In the first place the protective process of adhesion may at any time fail to keep pace with the spread of the ulceration, and perforation into the peritoneal cavity may take place, or the adhesions may be broken through by some muscular effort made by the patient, when the same result of course follows. For obvious reasons perforation is more apt to occur when the ulcer is in the anterior than in the posterior wall of the stomach; indeed, in the former position ulcers seldom attain any considerable size.

In other cases danger arises from the penetration of blood-vessels. We have seen that the recent "punched-out" ulcer often erodes an artery of some size; but in the chronic cases now described it is no uncommon thing to see a large artery, or even more than one, with its coats abruptly cut across, lying in the floor of the ulcer, and plugged with a little cylinder of clot that can be pushed out with little difficulty. In other cases, in which death has been directly due to hæmorrhage, the vessel is patent. The artery itself is sometimes a branch of the coronary artery of the stomach, or the trunk of that vessel, or a pancreatic branch of the splenic artery. Even the trunk of the large splenic artery itself is not infrequently penetrated by a gastric ulcer.

Another change to which ulcer of the stomach is liable is its cicatrisation. This occurs not infrequently. Indeed, one seldom sees a large ulcer which has not healed over in some parts of its surface. Brinton speaks of cases in which the whole extent of the ulcer has been found cicatrised, with the single exception of a point in the centre, occupied by an eroded artery, hæmorrhage from which had caused death. But, in most instances, when a gastric ulcer heals, the patient has good health afterwards. Should he die from some other disease, the cicatrix varies in appearance according as the coats of the stomach were more or less deeply and widely destroyed; it may show merely a little thickening of the submucous tissue, or it may form a hard, puckered mass, with radiating processes extending into the surrounding mucous membrane.

When an ulcer is seated at the pylorus its cicatrisation may give rise to narrowing of that orifice, and obstruct the passage of food through it. The result is that the cavity of the stomach becomes dilated and hypertrophied, exactly as when the pylorus is narrowed by scirrhous growth.

Again, the cicatrix of a large ulcer occupying the middle of the stomach may constrict it, and so cause what is termed an hour-glass contraction.

Pathology and origin.—The credit of having originally described gastric ulcer is commonly assigned to Cruveilhier, who published his account of it in 1830; or to Rokitansky, whose work appeared in 1839. But Dr

* Of these last, the most remarkable of all is perhaps one recorded by Dr Murchison himself of a woman who for three years kept a penny pressed into the sore left by a seton, until an opening into the stomach was formed. In one case, the only one which has come under the editor's care, the nature of the epigastric ulcer was proved by its discharging a clear liquid of strong acid reaction. Although firm adhesions had taken place, death ensued as the result of profuse hæmatemesis, possibly from a second ulcer.

Abercrombie had in reality pointed out all its distinctive characters in 1828. It must, however, be said of Rokitansky that he laid the foundation of the most modern view in regard to this affection, by suggesting that it arose out of a hæmorrhagic erosion. Virchow, in 1853, adopted this hypothesis and developed it. He attributed the destruction of the coats of the stomach to the corrosive action of the gastric juice. This, he said, cannot dissolve the mucous membrane, so long as the circulation is maintained, for the alkaline blood will neutralize the acid as it penetrates the tissues. He therefore supposed that the starting-point of the affection was some morbid change in the blood-vessels of that part of the stomach, whether obliteration of an artery, or obstruction of a vein. He also traced the conical form of the ulcer to the distribution of the tuft of vessels arising from a single arterial rootlet. And he discovered a further argument in the fact that, when perforation occurs, the aperture in the serous coat is always to be found in a particular direction, away from the centre of the ulcer. This he attributed to the circumstance that the apex of the vascular cone is likewise excentric, being always directed towards whichever is the nearer of the two curvatures of the stomach, along which the main arterial trunks run. Virchow's hypothesis has since been accepted by many other writers. Panum endeavoured to support it by artificially injecting little globules of wax into the branches of the abdominal aorta in dogs. He found that when they found their way into the arteries of the stomach the mucous membrane presented ulcers which resembled pretty closely the affection now under consideration in its earlier stage.

It is evident that Virchow's theory consists of two distinct parts. First, there is the question whether ulcer of the stomach at its commencement is caused by arrest of circulation in the corresponding part of the gastric mucous membrane, and begins in a hæmorrhagic erosion. This must be denied, on the ground that if such were its origin it ought to be frequent in those who suffer from obstruction of the portal circulation, as a result of disease of the heart or liver. Hæmorrhagic erosions are in fact common in cases of this kind, but not gastric ulcer. So that the commencement of the latter affection remains unexplained.

But a second question is whether the corrosive action of the gastric juice has anything to do with the further development of the affection. That it is not concerned in its commencement is clear, not only for the reason given by Virchow, but also because in its early stage it is exactly like ulcers which may occur in other parts of the alimentary canal. But a *chronic* ulcer of the stomach presents characters which are seen nowhere else, with the single exception of the first part of the duodenum; and this is exposed to the influence of the gastric juice. The only affection with which such an ulcer can be compared is a thick-walled chronic ulcer on the leg. It is quite conceivable that the frequent contact of an acid secretion with the surface of an ulcer of the stomach may not only retard its cicatrisation but also set up a process of chronic inflammation in its edge and floor that may give it its special characters. A further argument in favour of this view may be found in the fact that truncated branches of artery are often exposed in the floor of a gastric ulcer, whereas in all other parts of the body the walls of arteries show a remarkable power of resisting the ulcerative process.

Another point requiring explanation is that the great majority of ulcers occupy the lesser curvature of the stomach, or its close neighbourhood;

probably, as Sir William Gull suggested, it in some way depends upon the fact that this part is more fixed than the rest of the organ.

With regard to the *predisposing causes* that favour the development of ulcer of the stomach very little is known.

Sex.—All writers are agreed that it occurs much more often in females than in males; according to Dr Wilson Fox, two or three times as often.

But of 100 fatal cases of gastric ulcer extracted from the *post-mortem* records of Guy's Hospital by Mr Keiffenheim for this second edition, there were 58 in men and only 42 in women.

Of 100 other cases (viz. 93, which were not fatal, collected by the same gentlemen from our clinical records, and 7 others added from notes of private cases to make up the 100), there were 33 in men and 67 in women.

Age.—It is commonly believed that gastric ulcer is a disease of young women, and rare in either sex before puberty or after forty. This, however, was not the opinion of the late Dr Brinton, who maintained that it occurred at all ages from sixteen to sixty and upwards. The following table, drawn up by Mr Keiffenheim from the clinical and the *post-mortem* records of Guy's Hospital, may be of service in determining this point. Ninety-three of the cases are those of patients who left the hospital more or less benefited; the other 101 are those of patients who died and in whom the diagnosis of gastric ulcer was verified by an autopsy.

	Clinical Cases.				*Fatal Cases.*			
Age.	Male.	Female.		Totals.	Male.	Female.		Totals.
Under 9	0	+ 0	=	0	2	+ 2	=	4
10 to 19	2	+ 9	=	11	3	+ 4	=	7
20 „ 29	6	+ 24	=	30	7	+ 5	=	12
30 „ 39	9	+ 14	=	23	7	+ 8	=	15
40 „ 49	12	+ 10	=	22	15	+ 7	=	22
50 „ 59	4	+ 3	=	7	15	+ 15	=	30
60 „ 66				0	9	+ 0	=	9
70 and 72				0	1	+ 1	=	2
	33	+ 60	=	93	59	+ 42	=	101

There is one class of cases of gastric ulcer particularly prone to attack girls at or soon after the age of puberty; namely, those cases in which the affection remains latent until fatal perforation occurs. Dr Buzzard has recorded an instance of this kind in a girl nine years old. But it is a great mistake to suppose that the risk of perforation is limited to such cases, and ulcers that have already been recognised by the characteristic symptoms of the disease appear to be equally apt to destroy the patient's life in this way at all ages and in both sexes.

Symptoms.—Of these, the most significant, and generally the earliest, is *pain.* This may be of every possible degree of intensity, from a mere feeling of weight or tightness in the epigastrium, up to the most severe sensations of burning or gnawing or boring, attended with feelings of sickening depression. According to Brinton, it is rarely or never described as stabbing or lancinating. In the great majority of cases it comes on in from two to ten minutes after the ingestion of food, and remains an hour or two, subsiding when digestion is accomplished. If vomiting occurs, this almost always brings the pain to an end. Sometimes the pain does not begin until half an hour or an hour after a meal. It is often

distinctly increased by the ingestion of hard or indigestible matters, by food which is hot, and (according to Brinton) sometimes particularly by tea and by beer. In very rare cases, on the other hand, it comes on chiefly when the stomach is empty; and it is relieved by food, by hot water, or by brandy. The pain of gastric ulcer, however, is not always intermittent. It may be continuous, lasting for days or weeks together. These are generally cases in which the ulcer is already of long standing. Whatever its other characters, the seat of the pain is most frequently the epigastrium, immediately below the ensiform cartilage, but sometimes it is described as being behind the cartilage, and sometimes it is three or four inches lower down. Occasionally it is outside the median line, in one or other hypochondriac region. It is usually limited to a very small area, which, according to Brinton, is rarely more than two inches in diameter and is sometimes a mere spot less than half that size.

Scarcely less important than the pain already described is a pain in the back, to which Cruveilhier first drew attention as a symptom of gastric ulcer. This is of a gnawing character and is generally referred to a single spot between the eighth or ninth dorsal vertebra and the first or second lumbar.

Dr Brinton devoted great pains to the elucidation of the question whether variations in the seat of the pain in different cases could be traced to differences in the position of the ulcer. He collected some twenty-five cases in which the pain, having been referred to one or other hypochondrium, the ulcer was afterwards found to occupy the corresponding extremity of the stomach. He also ascertained that in some cases the position in which the patient chose to lie, as affording the greatest ease to the pain, was a guide to the seat of the ulcer; the prone position indicating that this was on the posterior, and the supine that it was on the anterior wall of the stomach; whereas, again, when the patient found relief by lying on the right side, the ulcer was at the cardiac end, and when on the left side, it was at the pylorus. But he was obliged to admit that in most cases, whatever the seat of the ulcer, the recumbent posture gave ease, and no information could be elicited as to the effects of changes of position.

Pressure on the spot to which the pain is referred almost always aggravates it, and in many cases there is the most extreme tenderness, so that not the slightest contact with the clothes, nor the gentlest touch from the physician's hand, can be endured. Sometimes pressure on the epigastrium increases the pain in the back. A very few instances have been recorded in which pressure has given relief to the epigastric pain.

A second symptom of gastric ulcer is *vomiting*. In the most marked cases this does not occur for some weeks after the patient has begun to suffer pain. It takes place when the paroxysm of pain induced by food has reached a certain height; and as soon as the stomach is emptied the patient is free from discomfort, or at most experiences a slight burning sensation for two or three minutes longer. The expulsion of the gastric contents is seldom attended with violent retchings. In rare cases, sickness occurs independently of the ingestion of food, but it would appear that this is generally due to some other cause than the ulcer; such, for instance, as alcoholic intemperance.

The vomited matters do not necessarily present any characteristic appearance. But whenever the presence of gastric ulcer is suspected they should always be very carefully examined, and on several different occasions;

for proper search may reveal the presence of blood. As above stated, *hæmatemesis* frequently is caused by ulcer of the stomach—in four fifths of Lebert's 252 cases observed at Breslau. But sometimes the blood is present in such small quantities in the vomited matters that it fails to attract the patient's notice. It is important that specimens of vomit should be selected for examination which contain as little as possible of food, and, of course, that they should contain no animal food that contained blood-corpuscles. If these precautions be taken, a fluid which had seemed comparatively clear will often throw down a sediment containing blood-corpuscles. In other cases, the amount of blood effused into the stomach is larger. Being altered by the gastric juice, it gives to the vomited matters a brown colour, or (to use the common expression) the appearance of "coffee grounds." This is due to the presence of hæmatin. It ought perhaps to be noted that the administration, as medicine, of a preparation of iron may give a blackish colour to the contents of the stomach, if the patient should about the same time take tea, or anything else containing tannic acid.

It must not be supposed that the presence of blood in the vomited matters, or even the occurrence of distinct hæmatemesis, is a proof that an artery has been exposed in the floor of the ulcer. The blood often comes either from the minute vessels which supply its surface, or from those which go to the adjacent part of the mucous membrane.

Other symptoms of gastric disorder frequently occur when an ulcer is present, which can hardly be regarded as symptoms of this disease. Such are dyspepsia, flatulence, pyrosis, and constipation. The appetite is generally defective, but in some cases excessive and ravenous, particularly when there is habitual vomiting of all that is taken ; sometimes, although there is great desire for food, the patient is afraid to eat, on account of the pain which follows every meal. In young women amenorrhœa attends ulcer of the stomach so frequently that some writers have endeavoured to trace a causal relation between them. The fact was pointed out long ago by the late Dr Crisp. In older women menstruation often goes on regularly, although they may continue for several years to be affected with gastric ulcer ; but if there should be anæmia from profuse hæmatemesis the catamenia may be suppressed for a time.

In long-standing cases there is often extreme wasting ; by the constant pain, and the deprivation of food, sharp lines are worn in the patient's face, and cause a peculiar physiognomy. Dr Brinton says that he was often able to recognise the disease at a glance in a crowded hospital out-patient room.

When a gastro-cutaneous fistula has developed itself the patient usually ceases to vomit. Food almost always escapes from the orifice as soon as swallowed ; to prevent this the patient has to wear a plug of lint or gutta-percha unless the mucous membrane should happen to protrude, so as to form a kind of natural valve. The general health, however, is often excellent, so that the patient may be able to go about almost as well as usual.

The characteristic symptoms of gastric ulcer, then, are the occurrence, soon after the ingestion of food, of a peculiar kind of pain which is relieved by vomiting, or subsides when digestion is completed, and the presence of blood in vomited matters or in the fæcal evacuations.

Duodenal ulcer.—When an ulcer is seated in the first part of the

duodenum, the pain is said to come on between half an hour and two or three hours after meals, or even later still; vomiting is not very common, but (according to Krauss, who has published a monograph on this disease) hæmorrhage has occurred in one third of all recorded cases. I may take this opportunity of mentioning that Krauss collected fifty-eight cases of duodenal ulcer in men and only six in women.

Latency.—Gastric ulcer is not infrequently altogether latent until it erodes a large artery; and the same is true of perforation into the serous cavity. It may indeed sometimes be a question whether the ulcer was absolutely unattended with symptoms during any part of its course, even when a patient who had seemed in perfect health is suddenly attacked with an illness which proves fatal in a few hours. In such cases the ulcer often presents no sign of inflammatory reaction in its walls; and there appears to be no reason why all the coats of the stomach should not be destroyed in a few days, or even in a few hours. Niemeyer relates the case of a young medical man who died rapidly of perforation, and who declared positively that for just a week previously there had been some trifling symptoms which had seemed to him to indicate a slight catarrhal affection of the stomach; and this writer appears to conclude that the time during which the ulcer had been forming must also have been a week. But this conclusion is very doubtful, for often when perforation occurs in persons apparently in good health, the ulcer has smooth rounded edges, and must certainly have existed for a considerable time; and it not infrequently happens that this affection is found accidentally in the body of a person who has had no gastric symptoms, and has died of some other disease.

But in other instances, ulcer of the stomach is overlooked, not because the patient has shown no signs of gastric disorder, for such signs may have been observed for several years, but because they have been of so slight a character that the case has been regarded as one of mere dyspepsia. The absence of vomiting is particularly apt to lead to this mistake. Brinton mentions one case in which there was no vomiting in four years during which an ulcer remained active; and he speaks of other cases in which it was represented by slight nausea only, or was limited to a single attack, or occurred only at the very close or the disease. According to this writer, it is upon the size of an ulcer of the stomach that the frequency and intensity of vomiting depend, rather than upon any of its other characters. No absolute rule can be laid down for the diagnosis of gastric ulcer in cases of this kind. Much depends upon the acumen and judgment of the physician. The existence of the disease may be suspected in many cases, in which the evidence of its presence falls far short of absolute proof. The diagnosis between simple gastric ulcer and cancer will be best considered in the section which relates to the latter disease.

Events.—An ulcer of the stomach may have several different terminations. Cicatrisation sometimes occurs, and the patient regains his former state of health. Several years ago some pathologists at Prague noted the state of the stomach very carefully in a large number of autopsies, and according to their statements both cicatrices and unhealed ulcers were found very frequently, and in the proportion of 147 of the former to 156 of the latter. Scars in the stomach have sometimes been discovered in the *post-mortem* theatre at Guy's Hospital, but very much more rarely. Moreover, since open ulcers also are sometimes found unexpectedly in the dead body, we must not lay too much stress on the more or less frequent discovery of cicatrices, as proving

that there is much chance of recovery by a patient who suffers from well-marked symptoms of the disease, or at least not without careful treatment. Certainly few would endorse Cruveilhier's statement that "simple ulcer of the stomach tends essentially to a cure." However, many cases are on record of symptoms followed by cicatrisation. One such is that of the anatomist Béclard, who suffered from pain in the stomach and vomiting, from which he gradually recovered; when he died, many years afterwards, the scar of an ulcer was found in the lesser curvature of the stomach.

The healing of a gastric ulcer is not infrequently partial. Cicatrisation may take place on one side, while in the opposite direction it goes on spreading. The pylorus may become narrowed, or the stomach may acquire an "hour-glass contraction," while part of the surface still remains unhealed. Thus symptoms of obstruction may arise, in addition to those which indicate the presence of the ulcer. Or the sore may heal for a time and afterwards again break out. Probably this is one cause of the remarkable circumstance that there may be a complete intermission of all the symptoms of gastric ulcer for many months, after which they return. In other cases, however, an apparent intermission is really due to the fact that, the first ulcer having finally cicatrised, a fresh one afterwards develops. Lastly, it appears probable that the inclusion of nervous filaments in a cicatrix is sometimes the cause of the continuance of pain after the subsidence of the other symptoms; and, indeed, after the cure of the disease.

The *duration* of this disease is often exceedingly protracted. Cases have been recorded in which symptoms were present uninterruptedly for twenty or thirty years, or even longer.

There are several different ways in which gastric ulcer may prove fatal. Sometimes the patient dies by gradual exhaustion. This, however, seems to be rare; three instances are recorded at Guy's Hospital out of twenty cases in which the immediate cause of death is noted. Brinton speaks of having seen three or four cases of this kind within a few months.

On the other hand, in eight of our twenty cases, death was traceable directly to hæmorrhage, and in nine to perforation. One is therefore compelled to regard these occurrences, not as accidents, but as the ordinary terminations of the disease. Other results of gastric ulcer are much more rare. In some cases the destruction of the gastric coats is followed, not by general peritonitis, but by a circumscribed abscess, which generally occupies the left hypochondrium, and which may in its turn perforate the diaphragm and set up fatal pleurisy. Still more rarely, the disease sets up pyle-phlebitis and abscesses in the liver.

It is an interesting question whether cancer ever develops itself secondarily in the floor or edge of a simple ulcer of the stomach. Trousseau speaks of the two diseases as antagonistic. But Brinton is disposed to think that the one may pass into the other; we have had at Guy's Hospital more than one case in which the stomach presented part of the circumference of what was apparently a simple ulcer, of which the remainder had been replaced by a malignant growth. If this is correct, the occurrence is probably more frequent than might appear from the absence of direct observations of it; for in many cases the extensive development of cancer would doubtless obliterate all traces of the previous ulcer.

Treatment.—There are some cases of ulcer of the stomach in which all that can possibly be hoped for is the palliation and relief of its symptoms. The medicines that are then to be prescribed are those which have already

been mentioned at pp. 336, 338, and 343, under the heads of gastric pain, of vomiting, and of hæmatemesis respectively. Brinton found in certain cases a preparation of bismuth, or the kino powder with opium, as effectual in arresting hæmorrhage from the stomach as the more powerful astringents. This writer expressed a very strong opinion as to the absolute uselessness of the oxide or nitrate of silver in this disease; but although it is no doubt impossible that these medicines can act upon the surface of the ulcer as lunar caustic does upon a sore to which it is directly applied, it is certain that they sometimes give relief to the pain. On the other hand, Dr Brinton spoke very highly of the value of opium, attributing to it, beyond its influence in relieving pain and sickness, a direct power of buoying up the nervous system and supporting the patient's strength. He in fact believed it to be as essential to the cure of an ulcer of the stomach as some surgeons have found it to be in cases of chronic ulcer of the leg. His way of using it was to give a small pill of the watery extract, or a few grains of the compound soap pill, two or three times a day. He prescribed it especially for patients of advanced age, or of broken-down constitutions, in whom the disease was of long standing and the ulcer probably of large size.

The use of purgatives requires much caution in cases of ulcer of the stomach, and there is no doubt that they should as far as possible be avoided. But Dr Brinton more than once noticed a definite and repeated coincidence between the occurrence of a paroxysm of pain and vomiting and an accumulation of fæces in the colon; and for such cases he recommends the use of castor-oil. In common with other writers, he speaks very strongly against the use of mercury in any form, saying that he is certain that he has witnessed relapses which could only be attributed to its administration.

A blister often relieves the symptoms of gastric ulcer, but it is said that in some patients it causes increased pain. Dr Brinton speaks of having observed this in cases in which there were old adhesions between the stomach and the abdominal walls. This writer thought that leeches should never be used, but Dr Wilson Fox says that the application of two or three of them sometimes gives marked relief to pain. A blister applied to the back has been found to relieve the pain in that region.

But in the great majority of cases something more may be fairly aimed at than the mere relief of symptoms. If there is any truth in the hypothesis that cicatrisation of gastric ulcers is prevented by the action of the gastric juice, the rational treatment is evidently to keep the stomach quite empty for a time, supporting the patient by enemata alone. In cases of obstruction of the œsophagus life may be maintained in this way for at least two or three weeks, a period which is probably long enough to enable a gastric ulcer to take on a healing action, even if it is not sufficient for its complete cicatrisation. Whenever the diagnosis is clear, and the patient can be induced to submit to this method of treatment, it would be reasonable to carry it out for a certain period at once. However slight the symptoms may be, one never can tell how near the peritoneum or some large artery may be to the floor of the ulcer; and every week's or month's delay must necessarily add something to its size, and so far diminish the prospect of benefit, if the patient should afterwards submit to the same plan of treatment.

Again, in many cases, the very urgency of the symptoms affords an argument for giving complete rest to the stomach, and feeding the patient by nutritive injections: for instance, when all that is swallowed

is shortly afterwards rejected. Vomiting, indeed, as Dr Brinton remarks, is much less amenable to treatment than any other symptom of gastric ulcer, and often resists the action of every reputed remedy. It is also by far the most dangerous and important of these symptoms, on account of the risk that it entails of the supervention of perforation, from the rupture of the protective adhesions. One can therefore never hesitate to recommend that the stomach should be kept perfectly empty for a time, when there is such obstinate vomiting. In some cases, indeed, it may not be absolutely necessary, so far as the mere relief of this symptom is concerned. There is the well-known case of Dr Hunter, before quoted (p. 338), in which vomiting that had been uncontrollable was checked by the limitation of the food to milk, given a spoonful at a time. A similar plan was suggested by Cruveilhier, for cases of gastric ulcer. Dr Brinton says that sometimes the milk is better borne when it has previously been boiled, or when it is mixed with lime-water; in some cases he has found a little fresh curd, mixed with a thin pulp of arrowroot boiled in water, better than anything else. As convalescence advances, ground rice may be substituted for the arrowroot, and afterwards biscuit powder. Sugar was specially objected to by Cruveilhier, and subsequent writers have endorsed his opinion; it is thought to produce flatulence.

Some persons, however, are unable to digest milk, and according to Dr Wilson Fox there are some elderly people whom it fails to nourish. Animal broths must then be given in its place, or peptonised milk may be tried. But many patients suppose themselves to have digestive idiosyncrasies, who are afterwards found to do just as well as anyone else upon the most rigidly restricted diet.

During convalescence from ulcer of stomach the most extreme care should be exercised as the patient gradually extends his range of diet. The quantity of food taken at one time must be such as will not distend the stomach; and all hot food or drink must be avoided. Complete abstinence from alcoholic stimulants is desirable, although Dr Brinton admits that he remembers more than one case in which one or two glasses of sherry, or even one or two tumblers of beer, were taken daily without seeming to impede the cure of what was probably an ulcer of the stomach.

Lastly, it must not be forgotten that pressure upon the epigastrium may do harm when there is a gastric ulcer. A woman should not be allowed to wear her stays, nor a shoemaker to press the last into the pit of his stomach. Care must be taken that, in manipulating the abdomen, only very gentle pressure is used. For similar reasons all violent exercise and sudden efforts must be most carefully avoided.

Cancer of the Stomach.—This is in every way a disease of great importance. Dr Brinton estimated that it caused 1 per cent. of the total mortality in London Hospital practice, and at Guy's Hospital the proportion is even higher, namely, 79 in 5990 autopsies, or more than 1·3 per cent. According to Dr Brinton it is less common than simple ulcer; but an analysis of the *post-mortem* records at Guy's Hospital during the last twenty years shows that there have been rather more than twice as many fatal cases of cancer as of simple ulcer of the stomach.

Anatomy.—It has been stated that there are three local varieties of this disease, as it affects the cardiac orifice, the body of the stomach and the pylorus. But as regards the first of these, almost all the cases that

have been set down as examples of cancer affecting the cardia have really been instances of cancer of the end of the œsophagus, extending into the adjacent part of the stomach. The 'Pathological Transactions' seem not to contain a single example of cancer beginning in the stomach at its œsophageal end; nor does the museum of Guy's Hospital show any specimen, in which the lower end of the œsophagus is not also affected. Moreover, in every one of the four or five cases that have at different times been recorded under the name of cancer of the cardia, in the reports of our *post-mortem* examinations during twenty years, there is room for supposing that the disease began either in the œsophagus or in the lesser curvature of the stomach. Indeed, on *a priori* grounds we might expect that a part at which the digestive tube is opening out into a large cavity would have little or no tendency to be affected with the disease, in comparison with the narrow passage above it.

Cancer of the body of the stomach, however, and cancer of the pylorus must be carefully distinguished from one another. And as the latter is the more uniform affection of the two, it may conveniently be first described.

The *pylorus*, when affected with cancer, becomes greatly thickened, so that it forms a rounded swelling, which is often somewhat lobulated, and which is almost always sharply defined towards the duodenum, while it passes gradually into the wall of the stomach, or extends for some distance along its lesser curvature. The disease generally involves the whole circumference of the orifice, which is consequently much narrowed. It grasps the finger tightly, or may be too narrow to admit it; but cases are very rare in which a large catheter cannot be passed into the duodenum. The mucous surface is generally much reddened; it may either be smooth or present nodular excrescences, and sometimes distinct villous growths. Most frequently it is more or less extensively ulcerated.

On making a longitudinal section, one finds that the several coats of the stomach are still plainly to be recognised. The thickest part of the mass is that which corresponds with the submucous connective tissue; this generally makes up two thirds of the whole. Next comes the muscular layer, which is likewise greatly augmented. This appears as a row of pinkish-grey translucent striæ, regularly arranged with opaque bands between them, which bands often consist of cancerous tissue. Still further outwards the subserous tissue is also thickened, but to a less extent; and it likewise is generally infiltrated with the new growth. The peritoneal surface may either be unaffected, or it may be the seat of an inflammatory process binding it to the adjacent parts, or, again, it may present more or less numerous cancerous nodules.

Cancer of the *middle of the stomach* is much more variable in its characters. In many cases it begins along the lesser curvature, and then it may either remain limited to that part or spread to one or both surfaces. In one case of this kind only a narrow border along the greater curvature was left untouched by the invading growth. In some instances it forms a more or less broad ring completely surrounding the middle of the organ. Sometimes, again, it appears as a large patch, with or without ulceration. Lastly, it may affect the whole of the stomach almost uniformly.

Whatever part of the stomach may be affected with cancer is very liable to become adherent to any other structure with which it happens to be in contact. Most commonly this is the under surface of the liver, and the

growth may then extend into that organ, and afterwards it may undergo ulceration; thus a large cavity may be produced, in which food (such as grape skins) may lodge. Or the diseased portion of the stomach may become fixed to the surface of the abdomen. In one instance the anterior part of the abdominal wall had altogether disappeared, being fused in a mass of cancer two or three inches thick. A gastro-cutaneous fistula may result, but this is even more rare than with non-malignant gastric ulcer. Or, again, the growth may become continuous with a mass of diseased glands near the pancreas, and with that structure itself. Or, perhaps more frequently, the first portion of the duodenum is drawn into adhesion with the back of the diseased pylorus; and sometimes an ulcerated opening forms between them, behind the proper orifice. This depends upon the fact, pointed out by Luschka, that the normal direction of the first part of the duodenum is from before backwards. In other cases the diseased part of the stomach becomes adherent to the colon, and a fistulous communication between them may afterwards develop itself.

Histology.—Cancer or malignant disease of the stomach is with rare exceptions true *carcinoma.*

Virchow only mentions two instances of primary *sarcoma* of the stomach, one of them being a case recorded by Dr Wilks in the tenth volume of the 'Pathological Transactions.' In each of these cases, and also in a third case of Dr Wickham Legg's (in the twenty-third volume of the same 'Transactions'), the patient was a young girl; and both the ovaries were at the same time affected with sarcoma. The disease seemed to have begun in the lesser curvature. Virchow remarks on the comparatively slight tendency to ulceration as a distinction between this form of disease and carcinoma of the stomach. But in a fourth case of Dr Cayley's, recorded in the twentieth volume of the 'Pathological Transactions,' there were large nodules of the growth projecting into the cavity of the stomach and these were extensively ulcerated. The patient was a man aged fifty-seven.

Virchow does not mention any instance in which disease occupying the pylorus was of a sarcomatous nature, but three or four cases of this kind have been observed at Guy's Hospital. One was in a woman aged forty-seven; the submucous tissue of the pylorus was three quarters of an inch thick. Dr Moxon describes it as thick but flabby, of a milk-white colour, yielding a clear fluid when scraped, consisting mainly of a well-developed fibrous tissue, but also containing some delicate spindle-cells with very large tails. In another case, which occurred in a man aged sixty-six, the pylorus was the seat of a new growth of yellowish look and of firm consistence, which proved to be a round-celled sarcoma. A third case came under the author's observation in June, 1876. The patient was a man aged sixty-seven. The pylorus, through which the finger could readily be passed, presented a large ulcer, seven inches in circumference, the base and sides of which were formed by a homogeneous pinkish-white substance, which yielded no juice, and consisted of round and oval cells, and spindle-cells, embedded in an intercellular substance containing mucin. Probably the cases which Sir Thomas Watson and others have described under the name of simple hypertrophy of the pylorus were examples of sarcoma. Dr. Wilks used formerly to lay considerable stress upon the fact that in some cases of so-called scirrhous pylorus the disease was really only a local thickening of the submucous tissue, with consequent hypertrophy of the muscular coat. He based this opinion partly upon the dry, juiceless character of the growth, consisting

mainly of fibrous tissue, partly upon the absence of secondary cancerous nodules. This last fact, however, is not by itself conclusive; for, out of forty-one cases of true carcinoma of the pylorus in succession at Guy's Hospital, there were at least five in which no cancer existed elsewhere in the body. On the other hand, in one case, which would otherwise have been designated simple fibrous thickening or hypertrophy, there were secondary nodules in the liver, the exact nature of which, however, is not specified in the report. One must bear in mind that carcinoma of the stomach (unlike carcinoma of the breast) is situated in what is termed a vital organ. Secondary growths would doubtless be found much more constantly if death did not so quickly ensue.

As above stated, in cancer of the stomach the submucous tissue is more thickened than any of the other layers. The older pathologists therefore supposed that the disease began there, but this appears to be a mistake. According to Waldeyer it always commences in an overgrowth of the glands in some one spot in the mucous membrane. These become elongated and dip down into the subjacent connective tissue; when they have reached it they proliferate actively, and so give rise to a cancerous nodule, which spreads out horizontally and may reach a large size, but which is nowhere connected with the superficial glandular layer, except at its starting-point.

Carcinoma of the stomach, as of all other parts, presents in different cases characters which in some respects differ very widely. These in part depend upon the proportion between the amount of fibrous stroma forming the alveoli and that of the nests of cells contained in them. If the stroma be abundant and the alveoli small, the growth has a tough fibrous appearance and yields but very little juice. If the stroma be scanty and the alveoli large, the growth is soft and of a milk-white colour, and yields much juice when scraped. But between the former (which would be termed "scirrhous cancer") and the latter (which would be called "encephaloid or medullary") all gradations exist, and in some cases it may be difficult to say under which head the disease should be placed. As a rule, however, carcinoma of the stomach belongs rather to *scirrhus*. According to Dr Brinton, three cases out of four belong to this form of the disease, and among cases affecting the pylorus the proportion would be still higher. As already stated, the new growth passes towards the serous surface between the bundles of the hypertrophied muscular coat. On the other hand, as Dr Moxon has pointed out, *medullary carcinoma* often destroys the muscular coat over a considerable area; so that the whole thickness of the wall is converted into a uniform mass of disease presenting elevations on each surface, but especially towards the cavity of the stomach. Sometimes branching processes sprout from the mucous membrane, which form beautiful microscopical objects, being each made up of a central blood-vessel, clothed with thick layers of well-formed cells. These constitute what has been termed "villous cancer," which, however, has no claim to be regarded as a separate variety. Dr Moxon has described a case of this kind, in which the floor of the growth was formed by a large mass of soft carcinoma, growing directly into the substance of the liver. It is doubtful whether a simple papilloma occurs in the stomach, analogous to the well-known affection which affects the urinary bladder.

In a very few cases a cancerous growth in the stomach has presented the characters of a *cylinder-epithelioma*.

The surface of a carcinomatous growth in the stomach is generally ulcerated. Sometimes there are only a few superficial erosions, but very often a deep sore is formed with hard, raised, ragged edges and a sloughing base. It is possible that the digestive action of the gastric juice may be concerned in bringing about the detachment of large masses of the cancer tissue, which sometimes leads to the erosion of blood-vessels of considerable size. In such cases the ulcer is horribly foul and offensive.

Cancerous growths in the stomach, as in other parts, are liable to undergo *caseous degeneration*. Sometimes scarcely a trace of active growth is discernible; it might be said that the primary affection had undergone spontaneous cure; but the patient has, nevertheless, died of an extension of the disease to other parts.

Another change to which cancerous growths in the stomach are particularly subject is that known as *colloid degeneration*. For a long time pathologists supposed that there was a special form of cancer characterised by its gelatinous appearance. This they termed colloid cancer; but such a view was attended with great difficulties, and not the least satisfactory result of the microscopical researches of late years has been the solution of the doubts which prevailed as to the remarkable affection in question. The stomach has always been known to be one of its principal seats. Sometimes the whole thickness of the organ is infiltrated with a jelly-like material, there being nothing to suggest the presence of ordinary carcinoma. But, more frequently, while some parts of the growth have the character of colloid, others have those of scirrhus or of a form intermediate between it and soft cancer; and under the microscope it is not uncommon to find that more or less colloid change is present in cases in which it may not be observed by the naked eye. It is in such cases that the real nature of the affection is most apparent. For it is found that the alveoli are no longer filled with the characteristic epithelioid cells, but that towards their periphery they contain a greater or less quantity of a structureless translucent substance. As this increases, the cells become less and less marked and finally disappear. It is said by Rindfleisch that they individually swell out into colloid globes which ultimately blend with the rest of the structureless material that now distends the greatly enlarged alveoli; but he also thinks it probable that a part of this is the result of a chemical change in an albuminous substance secreted from the blood. As the alveoli increase in size, they become spherical and the septa between them break down; and thus large translucent globes are formed which under the microscope appear almost structureless. Cancerous growths which undergo extensive colloid degeneration are less apt to infect distant parts than other carcinomata; but on the other hand this form of the affection often spreads over the peritoneal surface, where it produces masses of enormous size. This will come under notice again among the diseases of the peritoneum. It is a remarkable fact that secondary nodules of colloid cancer reproduce, not the histological structure of the primary growth, but its degenerative features. Thus the lungs may be found studded with translucent gelatinous nodules of typical colloid character.

Anatomical results.—Certain consecutive changes in the form and situation of the stomach arise when the pylorus is the seat of cancer. The obstruction thus produced at the outlet of the cavity causes enormous dilatation, so that the stomach may become large enough to hold six or seven pints, and may fill the whole abdomen, while its greater curvature sweeps round just above the pubes.

Its walls may at the same time be greatly thickened by diffused hypertrophy of the muscular coat, but sometimes they are exceedingly thin. In some cases, however, cancerous disease of the pylorus fails to obstruct the outflow of the gastric contents; or vomiting is so frequent that no accumulation of them in the cavity of the stomach takes place; or, lastly, the patient's appetite is so bad that scarcely any food is swallowed; while in other cases the extension of the morbid growth along the lesser curvature and into the surfaces of the organ tethers it and prevents its dilatation. Indeed, when the lesser curvature is the original seat of the disease, the cardiac and pyloric orifices may be approximated by the contraction of the growth; and the anterior and posterior walls of the stomach may be so flattened against one another that scarcely any cavity is left.

Ætiology.—Of the causes of cancer of the stomach very little is known. It occurs chiefly in persons over forty years of age. Of forty-six cases at Guy's Hospital eleven patients only were under the age of 40; of the remaining thirty-five, there were sixteen between 41 and 50, eleven between 51 and 60, and eight between 61 and 70. These numbers correspond generally with those given by Brinton.

Among forty-three of our cases, in which the sex was noted, thirty occurred in males, thirteen in females. This also accords with Brinton's estimate; but certain other writers have said that the disease is more common in women than in men. Hereditary predisposition is said to be well marked in some cases, and the case of the Napoleon family is cited in proof of the fact.

In some cases cancer of the stomach has followed a blow upon the epigastrium. Andral related an instance in which it occurred in a patient who had taken nitric acid, and Dittrich one in which arsenic had been swallowed. Habitual spirit drinking has been mentioned as an antecedent by some writers; and by others the influence of depressing emotions.

Symptoms.—These differ so widely in different cases that it is difficult to give a good description of them. At first they are generally very indefinite; the patient, without apparent cause, begins to complain of a sense of discomfort in the epigastrium after his meals, he has a feeling of weight and pressure there, and he is perhaps troubled with acid eructations. He attributes these symptoms to dyspepsia; but his tongue remains clean, and yet he loses all appetite. Soon he becomes conscious that he is wasting. The uneasiness at the pit of the stomach passes into pain; this may be a dull aching referred to the epigastrium or to the back; or it may be exceedingly severe and of a burning or lancinating character. It is generally more or less increased by meals, but it is by no means limited to the periods at which the stomach contains food. Next the patient vomits. The times at which he is sick vary with the seat of the disease. When it is the middle of the stomach, vomiting may come on soon after meals; when it is the pylorus, the food is usually retained for three or four hours, *i.e.* until it should be passed on into the duodenum. The matters ejected consist at first of partially digested food or mucus, but soon these are streaked with altered blood, which is of a brown or black colour; or they may contain sufficient blood to resemble coffee grounds. Constipation and flatulence are also complained of. The aspect of the patient is altered; he acquires a pale, yellow, waxy look, or his complexion becomes earthy. He becomes depressed about his condition, irritable, and morose.

Jaundice, and ascites with moveable nodules in the omentum, are fre-

quently present; they point to secondary cancer of the liver or portal lymph-glands and of the peritoneum.

It is said that hydrochloric acid is absent from the gastric contents in carcinoma ventriculi.

These symptoms are by no means conclusive as to the existence of carcinoma of the stomach; they do not warrant more than a suspicion of its presence; a careful physical examination of the patient can alone enable one to speak positively as to the real nature of the case.

In some instances the greater number of these symptoms are absent. Cancer of the lower end of the œsophagus extending into the adjacent part of the stomach is often latent, and most of the cases in which gastric carcinoma has run its course without producing any marked symptoms have been instances of this kind, such as generally receive the designation of cancer of the cardia. But Sir Thomas Watson relates a very similar case, in which the disease occupied the greater curvature. A gentleman, between forty and fifty years of age, was on his way home from Scotland (where he had been deerstalking and shooting grouse) when he was seized one night in a London hotel with a deadly faintness, very rapid breathing and severe pain referred to the sternum. He had before been gradually losing flesh and strength, but the only definite symptoms of which he had complained were sour eructations, loss of appetite and repugnance to solid food. Sir Thomas Watson could detect no physical sign of disease. The next night the gentleman had a similar paroxysm and died. The greater curvature of the stomach presented throughout its whole extent a mass of scirrhus, while the cardiac and the pyloric orifices were free. In a case at Guy's Hospital of colloid cancer of the stomach there was scarcely any vomiting at all and no complaint of local pain during the three months before the patient's death. It was supposed during life to be cancer of the peritoneum, which was the case, but it was secondary.

The examination of the abdomen, when cancer of the stomach is suspected, should be made in a particular way. The patient must be made to lie down, and the front of the abdomen exposed. Its shape must then be observed, and particularly whether there is any fulness of the epigastrium or of the neighbouring parts; or whether, on the contrary, they are hollowed. Sometimes a tumour may be seen through the parietes, but most commonly it is to be detected only by manipulation. In some cases it can be felt as soon as the hand is laid upon the surface, but more frequently much care is required. The abdominal muscles are often very rigid, particularly those parts of them which overlie a deep-seated swelling, and careless handling may throw them into contraction, so that nothing can be felt. The patient should be made to draw up his knees and to breathe deeply, making the diaphragm descend freely at each inspiration. If his attention can be concentrated on the inspiratory movements his abdominal muscles will often relax. The palm of the physician's hand (which must not be cold) is then to be laid gently upon the abdomen, and allowed to rise and fall as the patient breathes; gradually slight pressure is to be made, which may be increased until the abdomen has been very thoroughly explored. During all this time the palm of the hand as well as the fingers should be kept evenly applied to the surface; and all sudden movements of the fingers' ends which might excite contraction in the muscles of the abdomen must be avoided. This was Sir William Gull's method. There is sometimes extreme tenderness when the stomach is affected with cancer; but even then it is generally possible to

make out the form and relations of any tumour that may be present, if only due pains be taken.

The position and form of the *tumour* produced by cancer of the stomach of course vary greatly in different cases; they are in fact determined by the seat of the growth. If this should occupy the middle of the organ, the epigastric region, or that a little to its left, will contain any mass that can be felt on manipulation. In one case two nodular ridges could be clearly made out, corresponding one with each curvature; while between them, and further back, lay an irregular mass which seemed to occupy the posterior wall. In other cases a more or less rounded prominent mass is felt, which is the thickened anterior surface of the stomach. Dr Cayley has related a case in the 'Pathological Transactions' in which the left hypochondrium contained a firm slightly moveable tumour which reached below the umbilicus and was supposed to be the spleen, but which proved to be a stomach indurated by a carcinomatous growth in its walls.

When the pylorus is the seat of the cancer, the tumour is usually much more definite. In some cases its character can be made out almost as plainly during life as when the interior of the abdomen is exposed in the dead-house. It forms a rounded mass, often somewhat lobulated, perfectly circumscribed on all sides except towards the left, where it can sometimes be felt to pass gradually into the wall of the stomach; it may vary in size from that of a walnut to that of a Tangerine orange. Its seat is usually a little above and to the right of the umbilicus; considerably lower than the position of the healthy pylorus, which, indeed, lies so completely under cover of the liver as to be inaccessible to palpation. By Brinton, the fact that a cancerous pylorus is sometimes felt in the umbilical region seems to have been recognised only in the case of female patients, and he attributed it to the alteration in the position of the viscera caused by the use of stays; but this explanation is clearly insufficient, since the same thing is frequently observed in males. It probably results from the traction exerted upon the lesser omentum by the weight of the tumour; for when that fold of peritoneum is thickened and involved in the growth, or when the pylorus is retained in its normal position by adhesions, no tumour can be discovered. On the other hand, it sometimes descends very much lower. Dr Wilson Fox says that it may be found in the right iliac fossa or even in the pelvis, adhering to the intestine, uterus, ovary, or bladder. In one case a tumour in the *left* hypochondrium, of which the exact situation varied at different times, according as the stomach was more or less distended, proved to be the pylorus, which had been dragged over to the left side and was firmly adherent to the parietes and to the edge of the liver. A scirrhous pylorus usually seems to move slightly downwards when the patient draws a deep breath; perhaps the liver having itself to descend, pushes it down in its turn. Some observers have thought that the movement is rather apparent than real, the truth being that the expansion of the ribs carries the abdominal walls upwards over the tumour.

The tumour caused by cancer of the pylorus often receives an impulse from the abdominal aorta.

It is said sometimes to disappear entirely for days together, as the result either of twisting of the stomach on its axis or of the tumour itself being over-ridden by a distended colon.

To percussion it should yield a dull note, but when it is of small size this may be masked by the resonance due to the adjacent intestine.

But the results of manipulation of the abdomen in cases of cancer of the

pylorus are by no means limited to the discovery of a tumour; one has also to ascertain, if possible, the position and size of the stomach itself. This organ is often greatly dilated, and descends much lower in the abdomen than under normal conditions; the greater curvature may be below the umbilicus, and may even reach down to the pubes. The stomach then resembles a large flaccid bag; the epigastric and left hypochondriac regions are deeply hollowed, while the lower part of the abdomen is protuberant. This, indeed, is not by itself proof of dilatation of the stomach, for it is common enough in persons whose small intestines are distended with flatus, if their abdominal walls are also loose and flaccid. What is conclusive is the detection of the peristaltic movements of its thickened walls. If the surface of the abdomen be attentively watched, a wave of contraction may often be seen to start from the left hypochondrium, descend below the umbilicus, and pass on to the right side, and then a little upwards towards the cartilages of the right ribs. Or a rounded protuberance, as large as an orange, may rise up on the left side and travel round to the right, in the same way as the wave. In one or two cases we have observed distinct antiperistaltic movements (from right to left) in a hypertrophied stomach. Another indication of enlargement of the stomach is the production of a splashing sound by manipulation of the lower part of the abdomen; but this is not a sign of much value by itself, for similar sounds may be produced from the presence of gas and fluid together in coils of the small intestine. If there should be occasion to pass an œsophageal tube down into the stomach, the end of it may sometimes be felt through the abdominal walls. According to Leube, it may, in health, reach as low as the umbilicus; but if it descends below the level of the *crista ilii* the stomach must be dilated.

It will be observed that the physical signs of this form of chronic dilatation of the stomach differ in some respects from those of the acute paralytic distension of the organ described at p. 322.

The presence of dilatation of the stomach modifies to a considerable extent some of the other symptoms. In ordinary cases of cancer of the pylorus the patient vomits about three or four hours after each meal, at the time when digestion is completed, and when the food ought to be passing on into the duodenum. But when the cavity of the stomach is enlarged, three or four meals may be retained in succession; and the patient, when he does vomit, may bring up surprising quantities of fluid, several pints at a time. In one instance vomiting never occurred except at night; and sometimes the stomach rejects its contents only at intervals of some days. The matter brought up in cases in which the stomach is dilated present characters in some respects peculiar. It generally consists of a thin, highly-acid fluid, of a dirty grey, brownish, or greenish colour, which, on standing, becomes covered with a thick, frothy, yeast-like scum, while it deposits at the bottom of the vessel a more or less abundant sediment. In the scum, as well as in the fluid, oval spores and beaded tubes of the *Torula cerevisiæ* (or yeast plant) are often found in large quantity; and also certain rectangular bodies, which belong to another microscopic plant (cf. vol. i, p. 23). They are often present in enormous numbers and have the remarkable peculiarity that they are divided by cross lines into smaller rectangles, some of them into four, others into sixteen, and some even into sixty-four, according to their size. They thus resemble packages tied across again and again by cords; and Goodsir, who in 1842 was the first to observe them, gave them the appropriate name of *Sarcinæ ventriculi*

(*sarcina* = woolpack). Dr Chambers has pointed out that they may be found in the dead body in the stringy masses adherent to the interior of the stomach, and therefore he regards this as their proper seat, and not the liquid which the organ may happen to contain.

Another circumstance that may considerably modify the symptoms of cancer of the stomach is the formation of a gastro-colic fistula. The best account of this affection is that which Dr Murchison gave in the 'Edinburgh Medical Journal' for 1857. In almost all such cases the patient vomits fæcal matter; it has been pointed out by Dr Gairdner that the only exceptions to this rule are cases in which there is at the same time narrowing of the pylorus, so that the stomach is constantly kept overloaded with its proper contents. The patient's breath often has a fæcal odour, or he has eructations of intolerable fœtor, or a horrible taste in his mouth. The effect of the fistula is sometimes to allow matters to pass from the stomach into the colon; but this appears to be much less frequent, for there are only seven out of the twenty-three cases collected by Dr Murchison in which undigested matters were recognised in the fæces. This constitutes what is termed *lientery.** It must not be supposed to be of itself a proof that a fistula exists. The attempt has been made to increase its significance by giving food coloured with cochineal to patients who pass undigested matters from the bowels, and by observing what length of time elapses before the colouring matter appears in the evacuations. In a case of Schönlein's this occurred only at the end of twelve hours, in the last of seven evacuations that took place during that period. He inferred that the case was not one of gastro-colic fistula; and (with less reason) that the lientery was due to widening of the pylorus. In patients who have an opening between the stomach and colon, the appetite is generally very bad, but in one instance there was craving for food. Pain is not invariably present. Indeed, the formation of the fistula sometimes leads to the relief of pain that had before existed.

Sometimes a gastric cancer opens into the third part of the duodenum. In a case where this occurred at Guy's Hospital, it gave rise to no symptoms.

Returning now to the consideration of the symptoms of cancer of the stomach in general, it is a remarkable fact that towards the fatal termination of the disease they often subside. In one case vomiting did not occur once during the last month of the patient's illness. Fresh symptoms, however, often appear; ascites, from extension of the cancerous growth to the peritoneum, or from compression of the portal vein; jaundice, from interference with the common bile-duct; œdema of one or both of the lower limbs, from thrombosis of the corresponding femoral or external iliac vein or veins. In some cases the patient lies for several days before his death, with cold blue extremities, and with a scarcely perceptible pulse, but suffering no pain.

Prognosis.—The duration of cancer of the stomach cannot be stated with precision, because we have no means of fixing the date of its commence-

* The term λειεντερία (λεῖος, levis, smooth; ἔντερον, intestine) was applied in Greek medicine to cases of diarrhœa in which food or drink, as soon as taken, seems "to run through the body" without being digested. In ordinary cases the stimulus of food in the stomach provokes peristalsis, but the stools consist of mucus or other intestinal contents.—Ἐπὶ διαῤῥοίῃ, δυσεντερία, ἐπὶ δυσεντερίῃ λειεντερίη ἐπιγίνεται (Hipp., 'Aphor.,' vii, 76).—Intestinorum levitas, quâ continere nihil possunt et quidquid assumptum est, imperfectum protinus reddunt (Cels., lib iv, cap. xvi).—Levitas intestinorum, Græce λειεντερία, est velox exitus eorum quæ comeduntur atque bibuntur, quæ talia dejiciuntur qualia fuerunt devorata (Stephani 'Vocab. med. expos.,' 1564).

ment. But it seldom fails to destroy life within a short time from the appearance of well-marked symptoms. Dr Brinton estimated this period as amounting on an average to twelve and a half months. Niemeyer says that the disease generally proves fatal in from five to fifteen months. Dr Wilson Fox says that the most rapid case he can find recorded is one by Valleix, in which death occurred in four months. But at Guy's Hospital three cases have occurred, in which the duration of the symptoms was stated at one month, eight or nine weeks, and three months respectively. The longest case mentioned by Dr Fox was one in which the patient lived three and a half years after the appearance of the first distinctive symptom; but he cites from Abercrombie the case of Napoleon, who had paroxysms of severe pain for nine years before his death in St Helena. One remarkable case is recorded at Guy's Hospital, in which the patient had suffered for seven years from symptoms of disease of the stomach.

Diagnosis.—That the detection of cancer of the stomach is often a very difficult matter must be sufficiently evident from what has been already stated. There are, indeed, some cases in which the most acute observer cannot do more than suspect the presence of the disease. The only rule is that whenever symptoms of dyspepsia come on without apparent cause in a patient over forty or fifty years of age, and are accompanied with great and rapid loss of strength and of flesh, the possibility that cancer of the stomach may be developing must always be borne in mind.

Even when the symptoms point clearly to the existence of serious organic disease of the stomach, there always remains the question whether this disease is simple chronic ulcer or cancer. Between these affections the diagnosis is often perfectly easy. In very young persons, malignant disease of the stomach may be dismissed from consideration. It scarcely ever occurs except in the form of a sarcomatous growth, attended with ascites, while it produces comparatively slight gastric symptoms. And in older patients there are many points of distinction. In cases of ulcer, the pain and sickness bear a much closer relation to the time at which food is taken than in those of cancer. Vomiting of blood in considerable quantity is much more apt to occur at an early stage of the disease, and life is prolonged to a much later period. Any case in which well-marked symptoms have existed eighteen months or longer may generally be pronounced one of simple ulcer and not of malignant disease.

On the other hand, cancer of the stomach may for the most part be diagnosed whenever a tumour is discovered having the characteristics above described. Cases of simple ulcer affecting the pylorus have indeed been placed on record in which this part has been so thickened and indurated that the presence of a scirrhous mass has been simulated. And when an ulcer occurs at this part of the stomach it tends, when it heals, to narrow the orifice, and so may give rise to further symptoms resembling those which result from cancer. Brinton says that he has met with one or two cases in which during the whole progress of the disease there was nothing to justify a positive diagnosis. The supervention of jaundice or ascites or enlargement of the liver would decide in favour of the disease being malignant.*

* The asserted fact that free hydrochloric acid is absent from the fluid removed by a siphon in cases of cancer of the stomach has been lately much discussed in Germany, and Dr Van der Welden, of Strassburg, has introduced an alcoholic solution of tropæolin as a test with this object (see the seventh number of the 'Berliner klinische Wochenschrift' for 1887).

In considering the diagnostic value of pyloric tumour, it must be remembered that it is sometimes present when there are no other symptoms of gastric disease. Sir Thomas Watson relates a case in point. A young woman had a pulsating tumour in the epigastrium which was at first supposed to be an aneurysm, and afterwards a mass of fæces in the colon. She had no sickness nor any gastric symptom. The tumour proved to be a cancerous growth in the stomach; it lay in front of the abdominal aorta.

On the other hand, it must not be forgotten that in many cases of cancer of the stomach, and even in some cases of cancer of the pylorus, no tumour is at any time to be discovered.

The diagnosis from cirrhosis will be best considered in the chapter on diseases of the liver.

Treatment.—With regard to the treatment of cancer of the stomach there is very little to be said. The patient's strength must of course be saved as much as possible; on this account the range of food should not be too strictly limited, and he must not be subjected to the starvation plan of treatment recommended for cases of simple gastric ulcer. Alcoholic stimulants may be allowed in moderation.

Medicines are required only for the relief of particular symptoms. The remedies available in the treatment of gastric pain, of vomiting, and of hæmorrhage, have already been fully discussed (*v.* pp. 336, 338, 343).

There is, however, another symptom, of the management of which nothing has yet been said: the vomiting of liquid which forms a yeast-like scum and contains sarcinæ. For this Sir Thomas Watson recommends common salt, creosote, or the sulphites or hyposulphites. The last-named remedies were first suggested by Sir William Jenner; the dose is fifteen or twenty grains, and they should be taken soon after meals.

But the presence of sarcinæ in the vomited fluids is in most cases an indication that the stomach is in a state of chronic dilatation, and of this condition there is still something to be said. We have seen that it is one of the effects of cancerous disease of the pylorus, and that it may also follow the cicatrisation of a simple ulcer affecting the same region. Those writers who admit that the pylorus is liable to simple hypertrophy regard this as another occasional cause of dilatation of the stomach. Paralysis of the muscular coat limited to the pyloric portion is also mentioned by Dr Wilson Fox, who quotes two cases from Andral in which the stomach was greatly enlarged without there being any actual narrowing of the pyloric orifice. But in one of these instances there was extensive ulceration of the pylorus; and in the other its walls were indurated, although the muscular coat was atrophied. And even in cases of cancer affecting the pyloric orifice, it sometimes happens that, after the ordinary symptoms have developed themselves and advanced to a fatal termination, it is found on *post-mortem* examination that the finger can still be readily passed into the duodenum. Evidently, then, the outflow of the contents of the stomach may be arrested, without there being an actual mechanical closure of the channel.

Nevertheless, it is doubtful whether this kind of chronic dilatation of the stomach ever occurs independently of obstruction of some kind at the pyloric orifice. Bamberger says that it may be due to dragging down of the stomach by an omentum adherent to a hernial sac. Other writers mention hysteria, hypochondriasis, and diseases of the nervous centres as possible causes. Hodgkin pointed out that the organ is often very large in Hindoos, who eat enormous quantities of vegetable food.

The exact determination of the various possible causes of chronic dilatation of the stomach is becoming a matter of considerable importance, in consequence of the success which has recently been attained in cases of this kind by the systematic use of the stomach-pump, or rather the stomach-siphon. This treatment was first proposed by Professor Kussmaul, of Freiburg (now of Strassburg), in 1868. A long flexible tube filled with water should be introduced into the stomach every day. On lowering the longer half, the water flows out and the contents of the stomach follow it. As much of the contents as will come away readily should first be withdrawn. Some tepid water is then to be injected and afterwards withdrawn again, and the process should be repeated two or three times until what returns is almost clear. Weak solutions of carbonate of soda, or of permanganate of potass, or even of creosote, may also be thrown into the stomach in some cases. Dr Schliep introduced the practice into England in 1872 (Clinical Society's 'Transactions,' vol. vi, p. 41). It has now been tried in numerous cases, both in this country and abroad, and with decided success. The first introduction of the tube is exceedingly disagreeable to the patient; but before long he becomes accustomed to it, and he is even glad to pass it himself, so great is the relief which he experiences from its use. The vomiting often ceases entirely; there is usually great diminution of pain; the appetite improves considerably; the patient becomes much more cheerful; he regains much of the flesh and strength that he had lost, and he is no longer troubled with constipation.

Such striking results are not, however, to be looked for in those cases in which dilatation of the stomach is the result of cancerous disease of the pylorus.

In recent days, surgeons have ventured to open the abdomen and excise a cancerous pylorus. The possibility of the operation was proved by Heidenhain in the course of his experiments on the gastric and pyloric secretion in dogs. It was first practised on a human being by Billroth, and was not followed by immediate death. Mr Maylard performed excision of the pylorus on a patient of Dr Coats at Glasgow ('Brit. Med. Journ.,' July 24th, 1886). Other surgeons have attempted mechanically to dilate the pylorus, and others have proposed to avoid the obstruction by making a communication between the stomach and the third part of the duodenum.

Gastric induration.—Another organic disease of the stomach is one in which its walls are uniformly thickened, without the development of any morbid growth, its cavity being at the same time greatly reduced in size. This affection is spoken of by systematic writers as "fibroid induration" or "cirrhosis" of the stomach. We shall see hereafter that it is occasionally the starting-point of a general chronic peritonitis. The coats of the organ may be from half an inch to an inch and a half thick, and only capable of containing four or five ounces of fluid.

The symptoms of this affection are exceedingly obscure. A tumour may be discoverable, and this may be more or less resonant on percussion. Probably it would be impossible to distinguish cases of this kind from those of diffused sarcoma of the stomach (p. 356).

This fibroid thickening of the coats of the stomach has probably its first stage in the thick, pigmented, almost warty condition (*état mamellonné*) of chronic gastritis. It chiefly affects the pyloric region, and occasionally forms what may be termed a non-malignant fibrous tumour of the pylorus. Some-

times, however, it extends to the whole of the stomach, which, to use Dr Bristowe's words, "retains its form like an india-rubber bottle."

Gastric concretions.—Brief mention must be made of certain very rare cases in which immense masses of hair and string, matted together and moulded to the shape of the stomach, have been found in its cavity, and in that of the upper part of the intestine. Sir William Gull brought a case of this kind before the notice of the Clinical Society in 1871, and another was related at a meeting of the Pathological Society by Mr Pollock. In the former case the mass, when dried, weighed five and three quarter ounces; it was composed of string, thread, cotton, wool, and hair of three colours, that of the patient herself (a woman aged thirty-two) and of her children. She had never been noticed to eat hair; but the person from whom Mr Pollock's specimen was taken, and who was a delicate girl aged eighteen, had been observed to put hairs into her mouth when only three or four years of age. In that case a projecting tumour, the size of a large orange, was felt in the epigastric region during life; it was apparently solid and very slightly moveable. A tumour was also felt in a third case, referred to by Sir William Gull. It occurred in a woman aged thirty, who for fifteen years had indulged in the habit of eating her hair, and who had suffered all the time from pain in the stomach, but had worked as a servant until six years before her death. In that case the mass weighed thirty ounces.

None of these patients were of unsound mind; in lunatics a similar condition is not infrequent. A fatal termination appears generally to occur sooner or later from perforation of the stomach, with consequent acute peritonitis.

In one case of the kind, Mr Knowsley Thornton removed from the stomach of a girl of eighteen by abdominal section a mass of hair weighing two pounds, and, notwithstanding subsequent parotitis and other mishaps, the patient made a good recovery. He refers to the only similar case also successfully operated on by Dr Schönborn, of Königsberg, and quotes several instances only discovered after death. In one of these the mass of hair when removed from the stomach was found to weigh four pounds and seven ounces.

FUNCTIONAL AND INFLAMMATORY DISEASES OF THE INTESTINES

COLIC.—*Onset—Causes—Diagnosis—Prognosis and treatment—Lead colic—History of its recognition—Modes of infection—Diagnostic characters—The blue line—Pathology and treatment—Other effects of lead.*
DIARRHŒA.—*Acute and epidemic form—Chronic form—Tubercular enteritis—Diarrhœa from lardaceous and malignant disease—Symptoms—Treatment.*
TYPHLITIS.—*Relation of inflammation of the cœcum to perityphlitis and disease of the appendix—Origin and course—Diagnosis—Treatment.*
DYSENTERY.—*Anatomy—Catarrhal and diphtheritic forms—Sporadic and epidemic dysentery—Ætiology—Symptoms, diagnosis, course and event—Prophylaxis—Treatment of acute and of chronic dysentery.*
CONSTIPATION.—*Origin—Effects—Treatment.*
Acute catarrhal colitis—Ulcerative colitis—Intestinal casts.

THAT part of the alimentary canal which lies below the stomach is liable, like almost all other organs of the body, both to disorders of function and to diseases of structure. As usual, we will begin with the former.

COLIC.*—Of this malady the main symptom is pain, of a twisting or dragging or wringing character, generally referred to the umbilicus or to some spot in the upper part of the abdomen. It comes on in paroxysms which are often of extreme severity; but during the intervals the patient may be perfectly easy, and there is frequently no tenderness on pressure. The patient rolls about in the hope of finding relief, or lies on his stomach with his hands clasped together beneath him, or leans with the whole weight of his body across the back of a chair. In exceptional cases, however, pressure increases the pain, especially when parts of the intestines are distended with gas.

An attack of colic is often attended with nausea and vomiting. Writers say that the skin is cool, and that the pulse is often slower than natural; but in a case of severe abdominal pain, admitted into Guy's Hospital one night, the fact that the temperature was two or three degrees higher than normal led to some doubt as to its real nature; yet next morning the patient was well, and a review of the symptoms seemed to prove that the attack had been one of colic. In another case of colic, which the writer watched for some hours with not a little uneasiness, the skin was covered with a profuse cold sweat, and the pulse was much quickened. The expression was anxious, but there was not the peculiar sunken look of the features which belongs to the more dangerous forms of abdominal disease.

The *immediate cause* of colic appears to be a spasmodic contraction of some part of the large (or perhaps of the small) intestine. Associated with this there may be an accumlation of gas in adjacent parts of the bowel; and the attacks of spasm are then attended with rumbling noises, "bor-

* *Synonyms.*—Passio colica—Colum (Pliny)—Enteralgia—The gripes.—*Fr.* La colique.

borygmi," which are audible to the patient and those about him. In such cases there may be partial distension of the abdomen; but in typical colic it is hollow and retracted, its parietes are hard, and the muscles feel as if drawn up into knots.

A very important cause of colic, and one that must never be forgotten, is the presence of lead; this form will be described separately.

In some cases colic is due to the ingestion of food which irritates the alimentary canal. Perhaps the most marked instances of this kind are those in which persons are attacked by it after eating meat or game which is "high." Unripe fruit, mushrooms, ices, are also mentioned as being capable of exciting it. The majority of purgative medicines give rise to intestinal pains, which are essentially the same as those which characterise colic. Under some of these conditions the complaint is associated with diarrhœa, or the patient's sufferings may go on increasing in severity until one or more loose evacuations are passed, whereupon the pain ceases for a time or even altogether. But in the more typical cases of colic constipation is a prominent symptom, the attack has no tendency to terminate of its own accord by the bowels acting, and the administration of medicine is necessary to bring about this result. In such instances the exciting cause is generally the presence of hard scybalous masses in some part of the large intestine; these can often be plainly felt through the abdominal walls.

From what has been said in the previous paragraph it must be evident that colic cannot be regarded as a true neuralgia in the sense in which that term is used in the present work; and it is quite independent of the neurotic temperament.

The *diagnosis* of colic is often easy, but always needs caution. For some of the most dangerous forms of inflammation to which the abdominal viscera are liable may for the first few hours present very similar symptoms, and a mistake may be attended with fatal consequences. Certain writers, indeed, define colic merely as a painful affection, dependent on spasm of the bowels, and they say that it is really present in cases of acute peritonitis and the like, but that it then constitutes a minor feature of the disease. But the more convenient plan is to limit the use of the term to those instances in which the pain is, so far as our knowledge carries us, the substantive complaint—as we do with such terms as neuralgia or dyspepsia. The rule must then be that no case should be set down as one of colic, and treated so, unless its characters conclusively prove that no organic mischief is present. The most important of these characters are a retracted, hard, knotted state of the abdomen, the fact that pressure relieves the pain, and the absence of anxiety of countenance and of constitutional disturbance. Sometimes important light is thrown on a case by the fact that the patient has had former attacks of exactly the same kind, which have passed off entirely within a few hours; or, again, by the confession that he has not long before eaten some particular article which he knows by previous experience to be capable of giving him severe griping pain.

But colic has also to be distinguished clinically from some other affections not more grave than itself. One of them is a form of gastric pain with distended stomach which has already been described (p. 333). As in that complaint the epigastrium is prominent, colic can be confounded with it only when the bowel is considerably distended with flatus. The real nature of the case, however, can generally be made out by gentle percussion; the note

being (as Dr Wilson Fox observes) less prolonged and higher pitched over the colon than over the stomach. Moreover, the pain has seldom exactly the same position in colic and in gastrodynia; in the former it often extends into the right hypochondrium or downwards into the left iliac fossa in the direction of the sigmoid flexure, whereas in the latter it is absent from these regions of the abdomen. The so-called "biliary colic" and "renal colic" have also to be distinguished from ordinary intestinal colic; and it must be admitted that there may be cases—particularly those which female patients commonly call "spasms"—of which it is very difficult to say that they are more likely to belong to one than to another of these affections.

An attack of colic aways terminates in the recovery of the patient, and that within a few hours, or a day or two at the outside. It is therefore not of the greatest importance to adopt active treatment, particularly at first. And since some of the drugs by which this complaint would be shortened are precisely those which would do the greatest possible harm if the disease should be commencing peritonitis, one cannot be too careful to avoid interfering in any case as to the nature of which it is possible that doubt can be entertained.

Colic, in fact, affords the only exceptions to the rule which Dr Wilks used to lay down, that whenever a pain in the abdomen is so severe as to cause the patient to send for a medical man, this *ipso facto* proves that the administration of a purgative is unjustifiable. The rule itself is of great value, and should be kept constantly before one's mind; but there are a few cases in which it ought to be infringed. The patient may clearly owe his attack to something which he has eaten, and which has disagreed with him; he may have suffered in the same way before, and have quickly got well after taking a purgative; his abdomen may be retracted and hard, the pain may be relieved by pressure, and it may be entirely paroxysmal, with complete intermissions. Under such circumstances one is fully justified in giving him at once an ounce of castor-oil with twenty or thirty minims of tincture of opium, and in directing that half as much should be taken again at the end of three hours if the bowels should not have acted in the meantime. Enemata of turpentine or assafœtida may be prescribed if there should be any accumulation of gas in the intestine. The abdomen may be rubbed with a stimulating embrocation, or a tin filled with hot water may be laid across it, or a cloth folded round a quantity of hot bran.

Lead-colic.—Of all the causes of colic the most remarkable is absorption of lead into the blood.

Long before this fact was ascertained the complaint itself was well known as of endemic occurrence in certain parts of England and of the Continent, Thus it prevailed in Poitou, and was called *Colica pictonum;* in Devonshire, so that within the five years ending in 1767 two hundred and eighty-five cases of it were said to have been admitted into the Devon and Exeter Hospital; and in the West Indies, where it received the very appropriate name of the "dry belly-ache." In each instance it was at an early date attributed to some beverage which the people habitually drank; in Poitou to wine; in Devonshire to cider; in the West Indies to rum. In the first half of the eighteenth century Huxham endeavoured to refer it more definitely to the "tartar" contained in each of these drinks.

The discovery that this form of colic is due to the action of lead was

first made by Sir George Baker, whose paper on the subject, read at the College of Physicians in 1767, is still quoted as a masterpiece of inductive logic. He showed that in the counties of Hereford, Gloucester, and Worcester persons who drank cider did not suffer from colic; and then that Devonshire cider contained lead, whereas Hereford cider was free from it. Next he traced the presence of the metal in the former to the circumstance that lead was used in the construction of the cider-presses, and that leaden weights were sometimes put into the casks to prevent its getting sour. Not long afterwards it was shown that preparations of lead were added to the wines made in Poitou with the very same object of neutralising acidity, and that in the West Indies the stills in which rum was made had leaden worms. One important link in the chain of evidence connecting these facts together was the circumstance that both in Poitou and in Devonshire a peculiar form of paralysis affecting the upper limbs was commonly associated with the colic. This also was at the same time traced to the poison; it was described in the first volume of this work (p. 505).

The recognition of the cause of the complaint of course led to the disuse of those particular methods which had been concerned in introducing the metal into the human body, and endemic lead-colic soon became a thing of the past. But the affection itself is still often met with. Persons engaged in the manufacture of white lead are very apt to be attacked by colic. This is believed to be mainly due to the diffusion of the carbonate in a pulverulent form throughout the workshops, so that, besides being inhaled in respiration, it collects upon the hands and is carried into the mouth with the food. It is doubtful whether lead is ever absorbed directly through the skin. Painters and plumbers often suffer from the disease. It is sometimes observed also in glassmakers, enamellers, shot makers, printers, and type-founders, but not so commonly as was at one time supposed. In all these occupations it is said to occur more seldom than formerly; and if the men were but sufficiently careful, it is probable that only those employed in making white lead, and perhaps those who make sugar of lead, would ever be atacked with lead-colic.

The directions given to workmen whose occupations bring them into contact with lead are chiefly that they should pay great attention to personal cleanliness, that they should prevent as much as possible the poison from entering their air-passages, and above all that they should not swallow any particles with their food. They should have an outer suit of linen clothing, worn only while they are at work, and washed at least once a week. They should never take their meals in the workroom. When there is much dust, masks or respirators would probably be useful, but the men can seldom be induced to wear them. Some years ago Liebig recommended the habitual use of "sulphuric acid lemonade"—a liquid containing a small quantity of sulphuric acid sweetened by sugar, which it was supposed would render any compounds of lead that might enter the stomach innocuous, by converting them into an insoluble sulphate. And Sir Thomas Watson states that in some works at Birmingham the addition of the acid in question to the treacle beer which the men drank caused the disappearance of colic from among them. It had before prevailed to a distressing extent; afterwards not a single case occurred for fifteen months.

Lead-poisoning, again, has been sometimes known to arise fom the use of snuff with which the red oxide, or the yellow chromate, had been mixed, or which had been fastened up in metallic foil made of this metal.

In other cases, however, the complaint results from the impregnation of drinking-water or of some article of diet with the poison, and this source of the disease is the more important because it is so liable to be overlooked. A well-known instance is that of the family of King Louis Philippe when living in exile at Claremont: several persons were attacked at the same time. The amount of lead in the water which they drank was seven tenths of a grain per gallon. Now, it is well known that water containing carbonic acid and certain salts of lime has less action on metallic lead than water which contains such ingredients only in minute proportions or from which they are altogether absent. Thus the distilled water sometimes used for drinking purposes on board ship is particularly liable to be impregnated with the metal; and even zinc vessels may contain enough lead to make distilled water which has stood in them injurious to health. It must be added, however, that the cause of colic occurring on board ship, especially in the French navy, has been a matter of much discussion; certain writers are still of opinion that it may be due to some climatic condition or dietetic cause of which the nature is as yet unknown.

As an example of the production of colic by lead contained in food Sir Thomas Watson quotes the case of the troops at a station in Ceylon in 1832. More than seven tenths of those who made up the force were attacked, and the cause was found to be the presence of lead in some coarse sugar which had been distributed among the soldiers from one particular estate.

Diagnosis.—Ordinary colic and the affection caused by lead do not differ in their symptoms. There is, however, this peculiarity, that whereas mild cases of the former scarcely ever occur, at least so as to come under medical observation, there are not a few instances of the latter in which the pain is of trifling severity and the abdomen soft and supple. Thus the patient may continue to suffer from the complaint for a considerable period, and yet go on with his work, absorbing all the time more and more of the poison.

What alone enables one to speak positively as to the real nature of such cases is the fact that the presence of lead in the body is revealed by a peculiar discolouration of the gums. This was first pointed out by Dr Burton in 1840, and it is commonly spoken of as the "blue line." The name, however, is unfortunate, and has no doubt often led to mistakes; for under conditions of irritation the margin of the gums is very apt to present a bluish-purple border, which has nothing whatever to do with the presence of lead. The line which is really characteristic presents peculiarities which were accurately noticed by Sir William Gull. It consists at first of a single row of black dots, corresponding with the vascular papillæ of the normal mucous membrane. Mr Tomes several years ago proved that it was caused by a chemical action between the lead and the "tartar," or deposit of calcareous salts from the saliva which forms upon the teeth. At the same time he pointed out that in all probability the constituents of the tartar itself are not concerned in its production, but rather animal matters which had penetrated into the pores of the tartar, and the decomposition of which would give sulphuretted hydrogen. He showed that where there is a gap between the teeth, so that tartar is absent, no "blue line" is formed. Further evidence of the same fact is afforded by cases (of which more than one has been noticed by the writer) in which persons who have kept the teeth very clean have failed to present the line, although they were indubitably affected by lead. In many cases, too, the line is exceedingly partial, even when it exists. There may be only two or three black dots on one or more of the processes of gum projecting

up between the teeth; and a lens may be necessary to enable their true nature to be determined. Some writers have supposed that the lead with which the sulphuretted hydrogen combines was previously in organic combination with the elements of the tissues. But more probably it really comes directly from the circulating blood, whence it is picked out and precipitated in an insoluble form. Thus only can the way in which the dots correspond with the vascular papillæ of the gum be accounted for. I have had one opportunity of examining microscopically the gum of a person who had died while affected by lead-poisoning; and I found that the colour was due to the presence of a multitude of minute black granules. Some of these were aggregated together in the interior of small blood-vessels, the ramifications of which were mapped out by their presence; others were arranged in double lines which probably corresponded with the exterior of other vessels. Thus it seems that the "blue line" is really due to an *excretion* of lead from the blood. And this accounts for the undoubted fact that when iodide of potassium is given to a patient suffering from the poisonous action of lead, but in whom the line happens to be ill developed or absent, the appearance in question often becomes well marked within a few days, just as the lead can then be found in the urine, although none was being excreted previously. I have myself seen at least three instances in which a blue line has thus been brought out by iodide of potassium while the patient was an in-patient in the hospital for symptoms due to lead-poisoning. Dr Frank Smith, of Sheffield, made the same observation independently.

These views appear to afford an explanation of a circumstance, noticed by Mr Tomes, which has given rise to some doubt in regard to the clinical value of the "blue line" as an indication of the presence of lead. This is the fact that such a line has sometimes been observed in persons who have not been known to be exposed to the influence of the poison, and who show none of its symptoms. Evidently, if the black granules consist of precipitated sulphuret of lead, the introduction of the smallest quantities of the metal into the blood from time to time might ultimately lead to the formation of a blue line. And there is no one to whose body such minute proportions of lead might not have access. It was at one time supposed that other metals, such as copper or bismuth, might be capable of producing similar appearances.* But there is no evidence that this is the case; and, as regards copper, Dr Clapton has shown that what it really causes is a bluish-green line on the teeth themselves. When the salts of lead are given medicinally in considerable doses, the line often makes its appearance very quickly. Dr Burton met with instances in which it was developed within two days—one within twenty-four hours—the quantity of acetate of lead taken by each patient having then been only from fifteen to twenty-four grains. When the blood is richly impregnated with lead, and when the teeth are so neglected that plenty of sulphuretted hydrogen is provided, the line may go far beyond what has just been described. The spaces between the dots may be filled up by a uniform black discolouration, which spreads over the gum for some distance from the teeth. The insides of the lips may also present a similar staining; in one patient at Guy's Hospital this was half an inch broad.

* Dr Bristowe believes that bismuth, and perhaps other metals, may sometimes produce a line on the gums resembling that caused by lead. But this seems very doubtful. His account of the appearance which he had observed in one or two instances is that the line was "bluish-red," "wider and redder" than the lead line.

It must be added that there are marked individual differences in susceptibility to the influence of lead. Sir Thomas Watson mentions persons in whom the colic was caused by their sleeping for a night or two in a freshly-painted room; and he contrasts with such cases that of a painter whose first attack occurred when he had followed his occupation for nineteen years.

There is no satisfactory explanation of the fact that the introduction of lead into the body causes colic. The metal is deposited in the tissues; but according to the analyses of Dr George Wilson, of Edinburgh, there is less of it in the intestines than in several of the other organs. In a case of marked plumbism in Guy's Hospital which proved fatal, with cerebral symptoms, Dr Stevenson discovered 3·5 grains of lead in the liver, which weighed 45 oz.; ·465 grain in the spleen, weighing 4 oz.; ·246 grain in a deeply pigmented part of the colon, weighing 6 oz.; ·054 grain in the heart, weighing 10 oz., and none at all in 8 oz. of cerebral matter.

Morbid anatomy.—At the present day lead-colic is seldom or never fatal, at least in England; but formerly patients seem not unfrequently to have died of it. In such cases, and in those in which during an attack death occurs from some other cause, the alimentary canal is generally said to present no morbid change of importance. Several *post-mortem* examinations were made by Andral and by Mérat ('Traité de la Colique métallique, vulgairement colique de Poitou,' 1810). The former found the intestines free from inflammation and neither dilated nor contracted; the latter, however, did find the large bowel contracted, and he also observed the same thing in rabbits which had died of lead-colic. It is true that contraction of the large intestine is not very uncommon in persons who have died from various causes, but if constantly found in those who had had lead-colic it would be significant. Some time ago a *post-mortem* examination was made upon a patient of Dr Moxon's who had died of heart disease, but who was a painter and had a well-marked blue line. Several years before he had had colic, and shortly before his death he complained greatly of pain about the splenic flexure of the colon. At this part of the bowel, and also in the transverse colon, there were scattered several slate-coloured patches which, although not indurated, looked puckered, and which, if they were not actual cicatrices, must have resulted from extravasations of blood at some former period. Dr Bristowe showed, at one of the meetings of the Pathological Society, the intestine from a man, also a painter, who had died in St Thomas's Hospital of what was supposed to be colic, and in whom the intestines were enormously dilated and some parts of them black from hæmorrhage into the mucous and submucous tissues. Whether the appearances in these cases had anything to do with the fact that the patient had been exposed to lead-poisoning it would be difficult to say.

The *treatment* of colic arising from lead is the same as for any other form of the complaint. Sometimes there is considerable difficulty in bringing about an action of the bowels, so that two or three successive doses of castor-oil with laudanum may be required, and it may even be necessary to add one or two drops of croton-oil; but when once a free evacuation has occurred, all the symptoms generally disappear. The patient should, however, be put through a course of iodide of potassium. This salt possesses the power of eliminating from the body the lead which had been deposited in the tissues, forming with it a soluble compound which is absorbed again into the blood and then excreted by the kidneys. This was long ago established by the observations of Nicholson and Parkes; and in marked case at Guy's

Hospital it was again demonstrated: the urine had contained no lead before the patient began to take the iodide, whereas the presence of the metal was afterwards detected without difficulty. Probably it is because the lead is apt to remain in the body all through an attack of colic and afterwards that, as is stated by Sir Thomas Watson, the complaint appears to have a special tendency to relapse.

Other effects of plumbism beside colic are a peculiar form of atrophic palsy chiefly affecting the extensors of the forearm, atrophy of the cerebral cortex, epilepsy or epileptiform convulsions, sometimes accompanied by mania, chronic atrophic nephritis, and saturnine gout. Some of these have been already described (vol. i, pp. 505, 673, 864), and the rest will be noticed in their place in the chapters on Bright's disease and on gout.

CONSTIPATION.*—This is the most frequent functional disorder of the intestines. It has already been mentioned as a symptom of cerebral disease, of dyspepsia and of colic; it is almost constant in cases of jaundice, and is one of the most obvious effects of mechanical obstruction of the bowels. But constipation has far more often to be dealt with as a primary complaint; the patient may show no further indication of being unwell, or if so, his other symptoms are mere results of the constipation.

Pathology.—There are great differences in the frequency with which fæcal evacuations are passed by healthy persons. In some an action of the bowels occurs only at intervals of three or four days, and yet they suffer not the slightest inconvenience. This is not a condition which calls for, or would justify, any medical interference. But in children and young persons, the fæces are often retained for several days at a time, merely because they are careless, or unwilling to face the outside air, or too modest to go to a water-closet to which the approaches are not altogether private. Great evil may result from this, and all the more because it is unforeseen.

Apart from such cases as those last mentioned, in which at first the will is alone concerned, the exciting cause of constipation is either that the peristaltic action of the bowel is too slow or deficient in force, or that the fæces are dried up and hardened to too great an extent, so that they do not readily pass down the intestinal canal. Some writers speak of a "sluggish state of the nervous system" as being the prime cause of constipation; arguing from the undoubted fact that a tendency to the complaint is often inherited and shows itself in many members of the same family. Among the more immediate causes may be mentioned excessive abstemiousness in eating and habitual restriction to a diet which is too exclusively animal. Sedentary habits may also play a part in preventing the bowels from acting properly, particularly in those whose work is mental. Again, affections of the abdominal viscera of various kinds may interfere mechanically with the passage of the intestinal contents, but mere constipation is then merged in a more serious condition—that of obstruction of the bowels—which will be described separately. Only a caution may be given that when a woman suffers from habitual torpidity of the intestines, the possibility must always be borne in mind that this may be due to the presence of a uterine or ovarian tumour, or of a prolapsed or displaced womb.

The evil effects of constipation are due in part to the retention within the large intestine of what should have been voided from the body. According to O'Beirne, the rectum in health is generally empty, and when a

* *Synonyms.*—Obstipatio—Alvus adstricta.—*Fr.* Constipation.—*Germ.* Hartleibigkeit.

fæcal mass, even of small size, has once entered this part of the bowel it ought at once to excite sensations which should bring about its expulsion. Among the discomforts caused by slight constipation, not the least is that which results from the passage into the rectum of isolated round pellets which had been moulded in the sacculi of the colon; these may excite a great desire to go to stool, and yet they are passed only after violent straining efforts. Patients in whom this occurs often speak of themselves as suffering from diarrhœa, and only a strict cross-examination can elicit the real state of the case. Dr Bright was once summoned into the country, in consultation with an eminent surgeon and a general practitioner, to see a lady who had been in vain treated with astringents, being supposed to suffer from a relaxed state of the bowels. He asked to see the evacuations, whereupon a single little hard pellet of fæcal matter was shown to him, and it was at once clear that a purgative was what alone would give relief.

Even when the colon or sigmoid flexure are the parts in which the fæcal masses accumulate, their presence often gives rise to a sense of weight and discomfort and to colicky pains. But in persons who are habitually constipated, the rectum itself loses its natural sensitiveness, and may then become obstructed by hard dry scybala of enormous size. Under such circumstances the bowel sometimes becomes irritated and pours out mucus, and this, or fluid fæcal matter, may pass down by the side of the retained masses, so that a condition of diarrhœa may be closely simulated.

Habitual constipation has a marked influence on the general health and spirits of the patient. It is well known to make the tongue furred and the breath foul. It causes a nasty taste in the mouth. It gives rise to feelings of languor and melancholy, and makes the countenance depressed and haggard.

Treatment.—One cannot wonder that those who suffer from such discomforts are ready to take purgatives almost every day and in constantly increasing doses. One hears of persons who for years have never had an action of the bowels except as the result of aperient drugs, and most quack medicines consist of aloes, gamboge, or purgative salts. Each time that they are taken they cause free evacuations, but they exhaust the susceptibility of the intestine and render it less capable than before of responding to natural stimuli.

The proper treatment of inveterate costiveness is the reverse of this. One may be obliged at first to prescribe a draught, or to clear out the lower part of the intestine by enemata. But from that time all ordinary purges should be scrupulously avoided. The following plan of treatment was suggested by Dr Spender in the 'Medical Times and Gazette' for 1870. It consists in the regular administration of a pill containing from one to three grains of sulphate of iron, and about a grain of the watery extract of aloes, the compound extract of colocynth or the compound rhubarb pill. A quarter of a grain of extract of nux vomica, or of extract of belladonna, may be added; but to the former Dr Spender ascribes no value at all, and he thinks that the latter is of comparatively little service. At first, the patient should take three pills a day, one after each meal. He should be told that for two or even three days he is not to expect an evacuation, but that when the bowels have once acted they will afterwards be moved more frequently. And now comes the point of importance, —that whenever there is a loose evacuation, he should instantly decrease the number of pills which he takes. Nothing approaching to a purgative effect should ever be permitted. Very soon two pills a day are sufficient; and a fortnight later, a single one perhaps produces the desired

effect. Within another month he is able to do with a pill once or twice a week. If the patient should make a difficulty about taking pills, the best substitute for them is a mixture of the compound decoction of aloes and Griffiths' steel mixture.

The cases which Dr Spender reports are very striking, and after repeatedly putting this plan in practice the author can confirm his testimony to its value. Sometimes patients may at the same time take a spoonful of olive oil once or twice a day, or eat brown bread, or take a glass of cold water on first rising in the morning. The diet indeed should always be carefully attended to, and regular bodily exercise must be taken. Another important point is that the patient should seek relief at a regular hour each day, and allow the necessary time for the bowels to act.

Trousseau insists on this, and rightly observes that without any other treatment it may be successful in overcoming habitual constipation. When it fails, he advises enemata of water, at first with the chill off, afterwards quite cold; or suppositories of cacao butter or soap. But it is surely unadvisable to employ measures of this kind, which must tend to render the bowel less susceptible to its natural stimulus. The medicine which Trousseau chiefly recommends is the extract of belladonna.

No plan, however, is always and in all cases the best, and one must adapt one's treatment more or less to the patient.

(*a*) In children constipation is seldom obstinate, and is usually the result of the acuter form of dyspepsia (cf. *supra*, p. 318): they are more inclined to looseness of the bowels.

Costiveness in infants often depends on too much starchy food being given, and is best treated by animal broths being added to their diet, or by substituting well-made oatmeal porridge for wheaten flour. Lactose, maltose, and manna are each useful in such cases. Castor-oil should only be used for a special occasion, not as a habitual aperient. Friction of the abdomen is useful, particularly when constipation is combined with colicky pains. A small piece of soap is a harmless and useful substitute for an enema.

With older children, constipation is often the mere result of carelessness, or of over-eating, and needs no drugs for its cure. As an occasional laxative castor-oil is the safest and most effectual. In the less frequent chronic cases, the combination of rhubarb and magnesia (or rhubarb and soda) with an aromatic, known as Gregory's powder, the Pulv. Scammonii Comp., or the Pulv. Glyzyrrhiziæ Comp., are each useful, and better than sulphur or senna, or saline laxatives. Cascara sagrada is also a harmless domestic medicine. But the diet to be presently described is as a rule sufficient.

(*b*) When constipation is associated with anæmia and amenorrhœa in young women it is necessary to give iron in addition to laxatives. Griffiths' mixture, and Blaud's pills, which are equally famous in Germany, consist of sulphate of iron and carbonate of potash; pilulæ Rufi (pil. aloes c. myrrha) are usually combined with these, or decoction of aloes is given separately.

(*c*) The most numerous cases of all, those of habitual idiopathic constipation in adults of both sexes, and particularly in young men, not infrequently disappears of itself during a busy man's holiday. Sedentary occupations favour it, and exercise is often a cure. Yet we find that the demand for patented purgative pills is scarcely less among Australian herdsmen, who live in the open air and are always in the saddle, in the patriarchal simplicity of Oriental life, or the ruder barbarism of Central Africa, than among the inhabitants of European capitals.

The first indication is to avoid habitual medicine if possible. Exercise and diet should be tried first. Oatmeal porridge at breakfast, treacle or honey, brown bread, chocolate, and fruit—particularly figs, stewed prunes, and baked apples—are all valuable. A larger proportion of vegetable to concentrated animal food and of liquid to solid food should be taken; a tumbler of water (cold or hot) while dressing, and another at bedtime after a baked apple for supper, is an excellent prescription for habitual costiveness. With many persons a pipe or cigarette after breakfast seems to act as an efficient stimulus to peristalsis, perhaps only by directing the thoughts and favouring regular periodicity.

When drugs are necessary, small doses of belladonna at night, and nux vomica and aloes or rhubarb before dinner, form the best laxative pills; while aperient salines should be added to the early draught of water.

Some persons are certainly better if they take a purge once a week, once a fortnight, or (as was once stipulated in the indentures of London apprentices) once a month. For this purpose colocynth, aloes, and rhubarb may be combined in a pill to be taken over night, or the compound gamboge pill may be resorted to, followed by a seidlitz powder or other saline draught the next morning.

(*d*) In patients who are subject to gout, and in those who habitually live too freely, a blue pill followed by a black draught is still the best occasional remedy; and in chronic cases of gouty dyspepsia with constipation a saline laxative before breakfast is often the best treatment for weeks or months together. Rochelle salts, Carlsbad salts, Friedrichshall, or Hungarian bitter waters (Hunyadi-Janos), are best adapted for this purpose.

(*e*) The constipation of women at the climacteric period of life is generally best treated by a combination of sulphate of iron, sulphate of magnesia, and aromatic sulphuric acid, attention to diet, and substitution of cocoa or coffee for tea.

(*f*) Lastly, the atonic costive state of the bowels which may be called senile constipation is perhaps the only one in which the habitual use of clysters is permissible. Of drugs, belladonna is in these cases the most valuable; nux vomica comes next, and may often be combined with a small quantity of aloes as a daily pill. Many cases of what looked like malignant disease of the rectum or sigmoid flexure have been cured by the persevering use of unmedicated enemata and belladonna. Of this we had lately (1887) a striking instance in Philip Ward, where a case of most obstinate constipation, with tympanites and visible peristalsis, was thus treated.

DIARRHŒA.*—A frequent result of intestinal disorder is *diarrhœa*—the discharge of the contents of the bowel in a fluid condition, and with excessive frequency.

This depends upon one or both of two causes: an increase in the peristaltic movements of the bowels, and an increase in their secretion. It is often difficult to distinguish between these two conditions; and no doubt, in many cases, both are in action simultaneously.

Subacute form.—The exciting cause of an attack of diarrhœa is often very definite. Sometimes mental influences give rise to it; it attacks the child

* Διαῤῥοία, *i.e.* a running through: διαῤῥοιας ἅμα ἀκράτου ἐπιπιπτούσης, οἱ πολλοὶ ὕστερον δι' αὐτὴν ἀσθενείᾳ ἀπεφθείροντο; of the plague of Athens, Thuc., ii, 49.—Alvus soluta—Looseness of the guts.—*Fr.* Diarrhée.—*Germ.* Durchfall.

who is in dread of being punished, or the man who is about to preach or to lecture. Exposure to cold—getting a chill—is in some persons its starting-point. It often follows quickly upon the ingestion of some irritating substance, medicinal or dietetic. In infants it is apt to be set up by amylaceous food, which at that time of life cannot be digested, since the secretions which convert starch into sugar are not formed during the first few months after birth. Some infants, however, have diarrhœa even if they are fed with the best cows' milk, or with anything but human milk.

Impure drinking-water is another frequent cause of diarrhœa. In Parkes's work on 'Hygiene' references will be found to numerous observations in which this effect has been produced by water containing suspended or dissolved mineral substances of various kinds, or suspended vegetable matters. Such water, however, would scarcely be used for drinking purposes except under special circumstances. As a rule, the danger arises rather from the presence of animal matters, especially those of fæcal origin. Thus diarrhœa has often been traced (as in Croydon in 1854 by Dr Carpenter) to sewage contained in suspension in the water. Probably dissolved solid substances of animal origin may have the same effect, but this does not appear to have been proved. Dissolved sewer gases, however, are certainly known to be capable of causing diarrhœa. A striking instance occurred in Salford Gaol in 1859. Within four days 266 out of 466 prisoners were attacked with the complaint, whereas none of the officers nor any members of their families suffered. The water which the prisoners drank was at once examined and found to have a yellowish colour and an insipid taste. The cause of this was that the overflow pipe from the cistern led directly into a sewer and conveyed a most foul stench to the cistern, which was covered in closely with boards. The water supplied to the officers, on the other hand, was clear and refreshing. Both waters came from the same source, being merely stored in separate cisterns.

It is well known that in late summer and early autumn diarrhœa is very apt to prevail *epidemically*, particularly in large manufacturing towns. In 1859 Dr Greenhow was investigating the causes of this form of the complaint. The general conclusion at which he arrived was that in those places in which it prevailed most severely one or other of the local causes could always be traced; either the air was tainted with the products of organic decomposition, especially of human excrement, or the water which the people habitually drank was impure. And the outbreak at Salford, which occurred in the end of September, was naturally regarded as a crucial instance, proving that such conditions are really the cause of epidemic diarrhœa.

Subsequent inquiries, however, have rather tended to throw doubt upon the validity of this explanation of epidemic diarrhœa in general. Dr Greenhow himself observes that as a fatal disease it is almost wholly confined to children under five years. This might, indeed, be attributed to the greater susceptibility of very young organisms, particularly as there is as yet no evidence to show in what proportion attacks of non-fatal diarrhœa occur in older subjects. But then it further appears that most of the cases of fatal diarrhœa are in children under one year of age. Dr Crane, of Leicester, investigated the conditions under which 283 children had been placed who died from this cause in the summer of 1873, with the result that a large majority lived in houses not in bad sanitary condition, and that 107 were wholly suckled, 98 partially suckled, and 78 fed by the bottle alone.

Impurity of the drinking-water clearly was not the direct cause of the disease in the 107.

Dr Buchanan, while not denying that summer diarrhœa has associations with filth, is evidently inclined to think that it is really due to a specific materies developed by the influence of heat at a particular season. One point on which he insists is that whatever may be the heat of the weather before July it does not cause epidemic diarrhœa. In May or June the temperature may average 60° or 62°, or be even higher, and yet no increase of diarrhœa will result. Yet the connection of the disease with autumnal heat is certain, for the mortality from this cause is much greater in hot than in cool summers. A curious circumstance, to which Dr Buchanan draws attention, is that the disease seems to be of modern introduction, so far as can be learnt from the old bills of mortality. At the beginning of the present century there is no direct mention of it, under whatever name, nor can any special mortality among infants be traced as having occurred in the summer or autumn months. But for many years past epidemic diarrhœa has been a very fatal disease, sweeping away thousands of lives annually.

The diarrhœa set up by the causes hitherto mentioned is an acute disease; one that may run its course in a few hours, and that perhaps never lasts more than a week or ten days—if we except those cases in which the complaint may be kept up by the repeated ingestion of irritating food or bad water. Another form is that which occurs as a complication of acute diseases, particularly of puerperal fever.

The *anatomical changes* discovered when acute diarrhœa proves fatal are generally very slight. The inner surface of the small intestine may be reddened and lined with mucus, or softened. But as in the case of other mucous membranes vascular injection probably often disappears after death so that it may not be seen at an autopsy, even though it existed during life; and, on the other hand, there are great doubts whether softening, on which French pathologists formerly laid great stress, is not a cadaveric change.

Sometimes, however, the mucous membrane of the small intestine is attacked with a much more marked and severe form of inflammation. A *diphtheritic* state, especially marked in the valvulæ conniventes, may be found throughout a large part of the jejunum. In all such cases relaxation of the bowels is a principal symptom, and it is doubtful whether they can be distinguished at the bedside from the more severe instances of ordinary diarrhœa, although the pathologist, finding diseased conditions sufficient to account for death, is naturally disposed to place them in a separate category.

Chronic diarrhœa.—Another and a very different kind of diarrhœa is that which runs a chronic course, lasting for months or even for years. The intestine then often presents appearances which are regarded as indications of chronic catarrhal inflammation. Thus, slaty or black patches may be seen in its mucous surface, or black dots and rings, corresponding with the solitary follicles. Its coats may be thickened and it may be lined with a viscid opaque mucus. These changes are particularly well marked when they are secondary to mechanical congestion, as in cases of disease of the heart, or hepatic cirrhosis, both of which are commonly complicated with diarrhœa.

Sometimes, however, one may be unable to detect any definite change in the intestine although the diarrhœa had been present for a long time before death. The author once made a *post-mortem* examination in the case of a

gentleman who had come home from China with what is termed "white flux," in which there is constant diarrhœa with discharge of matters devoid of bile. At the time of his death the complaint had lasted some years, and the only morbid appearance which he could discover was extreme thinning of the coats of the intestine.

In the bowels of infants, with whom chronic diarrhœa is a frequent cause of death, one seldom finds any pathological change. Dr Eustace Smith mentions that ulcers are sometimes present in the large intestine; but, as he says, these are probably secondary and the result of the irritation set up by acrid matters which had been secreted by the bowel higher up.

Tubercular ulcers.—But there are other forms of disease in which diarrhœa is a principal symptom, and in which changes in the bowel leading to ulceration constantly occur. Enteric fever might be cited as a case in point, but both its symptoms and the intestinal lesions which accompany it have been fully described already. Another form of ulceration of the bowels scarcely less important is of a *tuberculous* nature. The earliest stage in which this can be recognised is that in which opaque yellowish spots are seen in or beneath the mucous membrane. They doubtless are the result of caseation of the tissue of solitary follicles, or of the follicles of a Peyer's patch the lymphoid tissue of which had before undergone augmentation. And in all probability the augmentation arises by a formative process. One could, indeed, conceive that it might be due to one of simple inflammation. But after careful investigation of the question, the result to which the author has been led is, that in the *post-mortem* room one seldom or never finds caseation of solitary follicles in the intestine without tuberculous lesions being present in other parts. The next step in the development of the affection is that the mucous membrane covering the little yellow spots breaks through, and a small circular ulcer is formed. This almost at once acquires a smooth rounded edge, which is indurated, so that to the finger it feels almost like a rim of leather. The increase in size of the ulcer always takes place chiefly in a direction transverse to the axis of the bowel. Thus its form becomes elliptical, or even roughly quadrangular, and it may become so large as completely to encircle the bowel. Its floor is generally formed by the muscular coat, which is thickened by inflammatory products and may still have some yellow cheesy granules adherent to its surface, The subserous tissue and serous membrane also become thickened and opaque, and these changes, and the presence of an injected zone of blood-vessels round the ulcer, enable its seat to be clearly recognised on the outer surface of the intestine. A more important character still is the presence, in many cases, of distinct tuberculous granulations in clusters, or forming long ridges, which are believed to correspond with the sheaths of lymphatic vessels, or (according to Rindfleisch) with the smaller arteries. Tuberculous ulcers are more common in the lower part of the ileum than in any other part of the intestine; they are often very numerous, and just above the ileo-cæcal valve thay may form extensive patches of very irregular shapes. Sometimes, however, only one or two of them are present, and they may be confined to the upper part of the ileum, or even to the jejunum; or those which occur there have much more marked characters than any which can be found lower down; or, again, they may be seen only in the cæcum or the colon, which portions of the bowel are indeed very liable to be affected in common with the ileum. Tuberculous ulcers of the intestine are probably never seen in the *post-mortem* room without the lungs being likewise affected with active tuberculous disease. And their

clinical importance is generally altogether subordinate to that of the pulmonary phthisis, to which the patient in reality succumbs. They do, indeed, afford an explanation of diarrhœa, when it is present, but in many cases this and all other symptoms of intestinal lesions are wanting, so that the autopsy alone reveals the fact that such ulcers have been forming. Sometimes, however, before any symptom or auscultatory sign of phthisis is discoverable a patient suffers for a very long period from diarrhœa, and this is ultimately proved to have been due to a tuberculous affection of the intestine. Trousseau quotes Chomel as having especially insisted on the importance of fever and night-sweats as indications of the presence of such an affection.

It is said that tuberculous ulcers sometimes heal and that their cicatrices may produce stricture of the bowel; this will come under consideration in the next chapter. Acute peritonitis has been caused by ulcers of this kind giving way into the serous cavity. More commonly the affected coil of intestine becomes adherent to a neighbouring coil, and an opening forms between them; in this way a series of communications between one part of the bowel and another may be formed.

Lardaceous disease.—Another cause of chronic diarrhœa is the presence of lardaceous change in the intestinal mucous membrane. This, I believe, never occurs without other organs being affected, and in a marked degree. It may be caused either by syphilis or by protracted suppuration, of which one instance is that which accompanies chronic pulmonary phthisis. It may be worthy of note, so far as concerns syphilis, that Trousseau lays stress on it as an occasional cause of chronic diarrhœa, but in the particular case to which he refers the affection can hardly have been lardaceous, for the symptoms yielded to mercurial treatment. The peculiar change is said by Dr Moxon to begin in the walls of the minute arteries, and to spread from them into the tissues around. To the naked eye the mucous membrane presents an appearance which one can more easily recognise than describe; Dr Moxon compares it to wet washleather. Iodine stains it of the colour of dark walnut wood. Peyer's patches are generally less affected than the rest of the mucous membrane.

Malignant growths.—Yet another cause of chronic diarrhœa—comparatively a rare one—is the development of a new growth in the intestinal walls. A carcinomatous ulcer sometimes gives rise to this symptom, but very seldom, for such ulcers, as a rule, narrow the bowel and cause obstruction instead. But it is important to remember that cancer of the colon or rectum may produce, not obstruction, but diarrhœa, or obstruction with diarrhœa.

More frequently diarrhœa is set up by a form of lympho-sarcoma, the distinctive characters of which were pointed out by Dr Moxon. It may invade a large extent of the intestine and completely surround it at various points, but always with the effect of making it wider than natural. It constitutes a white, soft, medullary growth, and has little or no tendency to ulcerate. A marked instance of this affection occurred in a child who died under my care in the Evelina Hospital; the growth everywhere seemed to have entered the coats of the intestine along the line of its attachment to the mesentery.

Symptoms.—Of the symptoms of chronic diarrhœa there is little to be said. The discharges may consist of a fluid fæcal matter just like that which the small intestine normally contains at a certain period after diges-

tion of food. It may be of a bright yellow colour, or more or less brown. In infants, the evacuations of diarrhœa are often green. This was formerly supposed to be a result of the administration of calomel, but it is now known to be due to changes in the bile pigment which are independent of any such cause. We shall hereafter find that bilirubin (as it is called) is apt to turn green when acted on by an alkali; and the statement has been made that green diarrhœal matters are always alkaline, but, as Kühne pointed out, this is a mistake. In adults, the evacuations often look as if they consisted of pure bile, and are said to respond to the tests for the biliary constituents much better than ordinary fæces. In other cases diarrhœal discharges are pale and watery, and they may even approach in character the "rice-water stools" of cholera. Under the microscope crystals of triple phosphate can often be detected in the matters voided from the bowels in all forms of diarrhœa. Mucus is sometimes present in considerable quantity, but pus can seldom be identified, either with the naked eye or microscopically. Hæmorrhage forms no part of mere diarrhœa, and blood can seldom or never be detected, even when there is extensive tuberculous ulceration.

Acute diarrhœa is generally accompanied by some colicky pain, by sickness, and by slight tumefaction of the abdomen. In chronic diarrhœa these symptoms are commonly absent; the abdominal walls often become deeply sunken and retracted. In infants prolapse of the rectum is very apt to occur as a complication, and the anus generally becomes sore and excoriated.

Mild cases, in adults, are attended with little or no disturbance of the general health. But in young infants even very slight diarrhœa may give rise to great depression of the vital powers, indicated by coldness of the surface, by dark pigmentation and sinking in of the spaces round the eyes, and by depression of the fontanelles. This last is a most valuable sign, and must always be borne in remembrance; it often gives a warning of danger at a comparatively early period, when the child would otherwise seem to have but little the matter with it, and when neither the pulse nor the respiration is accelerated. As already mentioned, in infants the disease is very apt to terminate fatally. Death may also occur in old people and even in adults, if exhausted by previous disease or privation; the symptoms are then always those of collapse. Such cases, in which the evacuations are generally profuse and watery, commonly receive the designation of "choleraic diarrhœa" or of "English cholera," and these names regularly appear in the returns of the Registrar-General every autumn. The question whether English cholera ever proves fatal to a healthy grown-up person has come before us in discussing the relation of this form of diarrhœa to Asiatic or epidemic cholera (vol. i, p. 298).

The treatment of diarrhœa is a matter which requires judgment on the part of the practitioner, and for which detailed rules can hardly be laid down; but some general principles may be stated.

In the slighter forms of diarrhœa it is sufficient to see that the patient is *warm*, particularly his belly and his feet; to make him lie down and keep perfectly still, and to give him arrowroot. A flannel binding or a hot bran-poultice is often comforting.

In many cases the diarrhœa is the result of irritating food and then a dose of castor-oil is the appropriate remedy, just as an emetic may be the best cure for vomiting.

In acute diarrhœa of any severity, the patient must be kept in bed, and only a very bland diet should be allowed. Sometimes it is desirable to administer a dose of castor-oil with a little opium; or, what is perhaps better, a scruple of Gregory's powder, or some other preparation of rhubarb. But, as a rule, all that is in the bowels is being swept away by the diarrhœa itself. The best medicine is then a stomachic with a little alkali. A formula which is widely used, consists of a scruple of carbonate of soda, twenty minims of aromatic spirits of ammonia, and an ounce of peppermint water; this may be repeated every two or three hours. Another valuable remedy is the subnitrate of bismuth.

It is not advisable to prescribe opium, or even morphia, in acute diarrhœa, at least until other remedies have had a fair trial. Nor should astringents be given at the commencement of the attack. On the other hand, in chronic diarrhœa, astringents are often very valuable, and may suffice of themselves to cure the patient. Hæmatoxylum, krameria, kino, catechu; the extract of Indian bäel; the compound chalk powder and the aromatic confection; alum, permitrate of iron, nitrate of silver, and sulphate of copper; each of these may be used. Many medical men employ sulphuric acid largely, particularly in children; others believe it to be useless.

Sometimes, however, each astringent seems to lose its effect after the patient has been taking it for a few days, and one is then obliged to prescribe them all in turn. This is particularly apt to occur when there is tubercular ulceration or lardaceous disease of the intestines; but diarrhœa may last for a very long time without there being any evidence of organic change in the bowel. In some cases of this kind opium is very serviceable; and it may be continued in free doses for several months without appearing to affect the patient injuriously in any way. For cases of chronic intestinal catarrh, Trousseau speaks highly of the arseniate of soda, and one is prepared to believe that this may be useful from what we know of the influence of arsenic in other catarrhal affections.

A method of treating diarrhœa, which in infants often succeeds, consists in giving no food whatever except raw meat, finely grated into a pulp and mixed with powdered sugar or currant jelly to make it palatable. Trousseau calls this "conserve de Damas" for the sake of mystification. He relates the case of a young lady who had had intractable diarrhœa for six months, and who was quickly cured by raw meat. To remove any prejudice he gave directions that each slice should be exposed to a strong fire for a few minutes, but that the part of it which had been acted on by the heat should be entirely cut away before it was given to the patient.

ENTERITIS.—We have hitherto avoided the use of the term enteritis, for it appears to be no distinct affection which requires to be described under that name. Cases do, indeed, occur in which an intense inflammation extends from the mucous membrane of the intestine to the other coats, but at the bedside such cases fall either under the head of severe diarrhœa or under that of dysentery. On the other hand, it is not uncommon for a patient to present symptoms which clearly indicate that besides peritonitis he has inflammation of some part of the bowel, but after death, even if not during life, the affection seems always upon careful examination to be referable to some form of mechanical obstruction, perhaps to what will pre-

sently be described as volvulus. Thus to give an account of *enteritis* as a substantive disease would be to deal over again with cases which have been fully considered under other heads.

Typhlitis* and Perityphlitis.—The cæcum is very liable to be attacked by inflammation, and typhlitis, as the disease which results is called, requires a special description. Systematic writers mention typhlitis and perityphlitis as two distinct affections. The former, they say, is an inflammation of the connective tissue behind the cæcum. It runs a chronic course, and comparatively seldom destroys life, except as the result of protracted suppuration. The latter includes the rapidly fatal cases of perforation of the appendix vermiformis.

Dr Wilks, however, has repeatedly expressed the opinion that both in "typhlitis" and in "perityphlitis" the disease begins in the appendix, and that variations in the intensity of the morbid process there are the real cause of the supposed distinction between them. The evidence which morbid anatomy affords points strongly in this direction. Dr Theodore Williams has recorded in the 'Pathological Transactions' a case in point. A man who was being treated for pleurisy was attacked with pain in the right iliac fossa, and vomiting; the bowels were closed, and a tumour as large as an orange could be felt in the right iliac fossa. Some days elapsed, and all the symptoms were subsiding, when pneumothorax supervened and he died. He had passed several dark and offensive motions containing scybala, and in consequence the swelling had diminished in size until it could scarcely be detected. Thus the case was a perfect example of what would commonly be called perityphlitis and regarded as the result of the accumulation of fæcal matters in the cæcum. For it appears that in the common use of the term "perityphlitis" there is in reality no intention to limit it to cases in which the connective tissue behind the bowel is the exact seat of the disease, but rather an unacknowledged feeling that the term has a wider signification than typhlitis, and means that the disease is "about" or "in the neighbourhood of" the cæcum. However this may be, the real nature of Dr Williams's case would not have been cleared up if the patient had not died accidentally from another cause. Then it was found that the cæcum was surrounded by adhesions, but that there was a small collection of purulent matter round the appendix vermiformis. Here Dr Powell found a minute perforation, and outside the aperture lay a small mass of hardened fæcal matter. Two cases which came under Dr Wilks's observation tend to establish the same conclusion. In each of them the patient had a comparatively mild attack and recovered; afterwards he was seized a second time with the disease and died, when a *post-mortem* examination showed that the disease had originated in the cæcal appendix, and that this was perforated. The name typhlitis therefore seems preferable to perityphlitis.

Origin and course.—The affection spreads from the mucous to the serous coat of the appendix, either by a process of ulceration or by one of sloughing; in the latter case the end of the tube, or one side of it, may be found in a gangrenous state. The cause of the inflammation is generally the presence of

* Typhlit's, from τυφλόν, cæcum sc. intestinum, the blind gut, or caput coli. It is perhaps well to remind the reader that the cæcum of human anatomy is a mere dilatation of the colon found only in man and certain apes. The homologue of the greatly developed true cæcum of many of the lower animals (*e.g.* the horse, the rabbit and other rodents) is the atrophied *appendix cæci*, which only occurs thus reduced in man and the anthropoid apes, and in the wombat.

a concretion. This may be the size of a pea, or as large as a plum-stone. It sometimes consists of a substance like wax, but usually is composed merely of hard, dry fæcal matter, mixed with mucus, and containing a large proportion of earthy salts. A mass of this kind may look very like the stone of a cherry or some other fruit, and has often been mistaken for such a substance. Indeed, supposed "foreign bodies" from the appendix have so frequently been found on examination to be of fæcal origin that many pathologists are disposed to doubt whether typhlitis is ever set up by such a thing as a fruit-stone. It is, however, certain that seeds, pills, bristles, pins, pieces of bone, and shot, have all been found in the appendix, and that some at least have led to its perforation.

In certain cases ulceration of this part of the bowel, penetrating its serous coat, has been tuberculous; and in yet other cases no exciting cause for the inflammation can be discovered. It is possible that the walls of the appendix may have given way as the result of its distension with fluid, for such a condition is now and then met with, the opening into the cæcum being closed. Dr Wilks saw a case in which the appendix was dilated to the size of the ileum, and distended with three or four ounces of white odourless mucus.

The extent to which the inflammatory process spreads over the peritoneal surface varies greatly in different cases of typhlitis. Sometimes it lights up almost instantaneously throughout the whole serous cavity. There is then during the early part of the case no possibility of determining the fact that ulceration or sloughing of the appendix formed the starting-point of the disease; one can only set it down as an instance of acute peritonitis, the cause of which is unknown. Such cases will be again considered further on. When they terminate favourably it becomes possible after a time to detect a hard swelling in the right iliac fossa, and this clears up their nature.

In other instances the symptoms of acute peritonitis are wanting, or but little marked. The main clinical feature depends upon the inability of the intestinal contents to pass through the affected part of the bowel. They are sometimes undistinguishable at the bedside from cases of primary mechanical obstruction, and will be referred to again in the next chapter.

Diagnosis.—The local symptoms, together with the age of the patient, and the way in which the case began, help us in most cases to distinguish typhlitis from ileus; but in cases only seen after peritonitis has developed the diagnosis may be difficult or perhaps impossible. From tubercular peritonitis the distinction is usually easy; but here also it is sometimes impossible to make, and what began as typhlitis not infrequently ends as tabes mesenterica.

Cases of typhlitis have been mistaken for enteric fever; in the former diarrhœa may take the place of constipation, and in the latter it is far from common for diarrhœa to be entirely absent. The rose-rash is often absent in children, and the course of the temperature will not always be distinctive, or at least not at first.

Lastly, the existence of inflammation or suppuration in the right iliac region may be the result, not of typhlitis, but of ovaritis, of caries of the ilium, or of post-peritoneal abscesses, originating in the kidney or the vertebræ.

Thus the cases of typhlitis that can be satisfactorily diagnosed form only a moiety of the whole, and, as a rule, they are the milder ones,—those in which the inflammation does not spread beyond the serous covering of the

cæcum and the parts immediately adjacent. Among the symptoms is pain, referred mainly to the right iliac fossa. This is generally in part paroxysmal; it is often of extreme severity; and it is associated with more or less tenderness, the latter being sometimes so marked that the patient cannot bear even the slightest touch. Nausea and vomiting are generally present, and also constipation. The amount of constitutional disturbance is very variable. But the principal symptom is the presence of an ill-defined rounded swelling extending upwards from the iliac region towards the right loin. This is doubtless formed in part by the thickened coats of the affected portion of bowel, but in much greater part it is due to the accumulation of fæcal matters within it. The size and form of this swelling may vary from day to day. If the disease subsides it gradually disappears.

Sex and age.—Typhlitis is much more common in males than in females. Out of ten consecutive cases of the author's, eight occurred in boys or men; out of fourteen patients under the editor's care, nine were males. It is a disease of an early period of life; in seven of the ten cases the patients were between thirteen and twenty-one years old; in eight of the fourteen cases the patients were between ten and twenty, in one six, and in four between twenty and forty.

Treatment and prognosis.—The course and event of typhlitis depend very much upon the treatment which is adopted. Few cases in which the disease can be diagnosed as seated in the cæcum terminate fatally if judiciously managed; and fortunately, the treatment to be adopted in a doubtful case scarcely differs from that suitable to one in which we are satisfied that typhlitis is present. The essential points are that the patient should be kept perfectly at rest, that he should be strictly confined to liquid diet, that he should not be allowed to take a single dose of aperient medicine, and that opium should be given freely. The treatment should in fact be exactly that for acute peritonitis. When the attack subsides, the greatest possible care must be taken to prevent a relapse. Even then the action of the bowels should be solicited by enemata only, and never by medicines taken into the stomach; and the restriction to fluid food should be continued for several days longer than seems at first sight to be necessary. One repeatedly sees relapses occur from disregard of precautions that seemed to have been enforced with sufficient emphasis to ensure their being attended to. The disease, indeed, is one which is very apt to recur, even at considerable intervals of time. In more than one instance repeated attacks have taken place, with a few weeks or months between them, until at length there has been one so severe as to place life in imminent danger. The patient has then at last submitted to being kept in bed for a considerable time, and has observed the greatest possible care during convalescence; probably for this reason this alarming attack has often been the last.

When typhlitis ends in suppuration, the pus forms in the loose connective tissue, between the cæcum and fascia iliaca, and forms an external abscess, marked first by œdema and then by fluctuation. Sometimes it passes out of the pelvis through the sciatic foramen, and points below the fold of the buttock; sometimes it passes under Poupart's ligament; it very rarely reaches the rectum, but may open into the bladder, as in the case of a boy under the editor's care some years ago, who discharged the pus *per urethram* and made a good recovery.

When suppuration has occurred the abscess should be opened by Hilton's method and drained. In one case lately in Mary Ward, the pus was allowed

to find a way for itself, a fæcal fistula formed externally, and this ended in lardaceous disease.

In a remarkable case of the late Dr Mahomet, Mr Symonds cut down upon the appendix cæci and removed the concretion which had caused the mischief ('Trans. Clin. Soc.,' 1885, p. 285). The result was successful.

Even when typhlitis presents itself clinically under the guise of intestinal obstruction or of diffused acute peritonitis, it seldom destroys life if judiciously treated. There may be cases in which a considerable part of the appendix sloughs away, and in which death is inevitable; but only one fatal case occurred during five consecutive years at Guy's Hospital, if we exclude those in which death took place a few hours after admission and in which purgatives had been given before.

DYSENTERY.*—The symptoms of this disease are very different from any yet mentioned. The patient has frequent desire to go to stool, but passes little except mucus or blood; at the same time he has a severe burning sensation at the anus, and complains of griping pains in the abdomen, in the course of the large intestine.

But the symptoms just enumerated are not by themselves sufficient to characterise dysentery, for they may be caused by other affections of the large bowel. Hence it will be convenient to describe first the morbid appearances which belong to the disease.

Anatomy.—The structural change which characterises dysentery consists in an inflammatory process which has its principal seat in the mucous membrane, and which is not limited to any one part, but spreads more or less widely throughout the whole length of the large intestine, and even into the lower part of the ileum. Cases have occurred in Guy's Hospital in which the last few feet of the ileum have been diseased, in conjunction with the whole length of the large intestine, but in which no indications of scurvy have been present. So that extension to the small intestine may occur in severe cases of dysentery.

In some instances all the large bowel, from the rectum to the cæcum, shows morbid changes of the same kind and in the same stage. But in others the disease is more advanced, or more severe in one part than in another. Commonly the rectum is the seat of the most intense changes, and these gradually diminish towards the cæcum; but sometimes the reverse is the case, as was noted by Sydenham. In other cases the flexures are said to suffer more than the intervening parts of the bowel.

The appearances presented by the affected parts in dysentery are exceedingly varied, but most writers are now agreed that the processes concerned in their production may be reduced to two. Virchow designates these respectively "catarrhal" and "diphtheritic." Excellent descriptions of them have been given by Heubner, of Leipzig, and by Woodward, of the American army.

The catarrhal form.†—The mucous membrane at first shows lines and patches of a dark red colour, with points which are almost black. The

* *Synonyms.*—Bloody flux.—*Fr.* Dysentérie.—*Germ.* Ruhr, blutige Ruhr. The Greek word is classical: a passage in Herodotus clearly alludes to the dysentery of camps:—(of the army of Xerxes during its retreat from the invasion of Greece) ἐπιλαβὼν δὲ λοιμός τε τὸν στρατὸν καὶ δυσεντερίη κατ'ὁδὸν διέφθειρε, lib. viii, cap. 115, and the term is common in Hippocrates, who correctly refers it to ulceration of the intestine. It is also used by S. Luke, himself a physician: πυρετοῖς καὶ δυσεντερίᾳ, συνεχόμενον (Acts xxviii, 8).

† *Synonyms.*—White dysentery.—*Germ.* Katarrhalische Ruhr—ῥεῦμα γάστρος (Galen).—Intestinorum rheumatismus (Cœl. Aurel.)—Coryza ventris—Dysenteria fiens (Sennert), a translation of Galen's Δυσεντερία γινομένη.

summits of any ridges or folds projecting into the interior of the bowel are more injected than other parts of the surface. The mucous membrane is lined with a rather thick layer of mucus streaked with blood. It is itself swollen, and so is the submucous tissue. In the earliest stage of the disease, all that the microscope reveals is a dilatation of the minute blood-vessels, which are gorged with blood. Soon, however, inflammatory products are poured out. The mucous membrane is now still more swollen; it becomes less uniformly reddened; the solitary follicles are enlarged and appear as white points with red rings round them. The submucous tissue is increased from three to five times in thickness; even the muscular coat is swollen and thrown into folds.

Under the microscope all the tissues are seen to be infiltrated with pus-cells, which are also present in large numbers in the mucus lining the interior of the intestine. In the submucous tissue the pus-cells occur chiefly in the spaces round the blood-vessels. The solitary follicles are markedly increased in size; the lymph sinus which surrounds each of them is wide, but does not contain pus-cells; leucocytes, however, are collected in large numbers under the thin mucous membrane which covers the glands, and which evidently was about to give way and rupture.

In former days there have been vehement discussions as to the importance of these changes in the solitary follicles, in relation to the changes in the rest of the mucous membrane. In 1843–44 Dr Parkes examined the intestine very carefully in numerous fatal cases of dysentery, and stated that the earliest lesion was the alteration in the follicles; and Dr Baly arrived at the same conclusion from his investigations at Millbank Prison. Even before this the question had been mooted; Cruveilhier had insisted that the disease was not a follicular inflammation, and had gone so far as to say that the solitary glands had no share in it. Since the publication of Dr Parkes's observations, the objectors to his views have rather taken up the ground that the white granules visible in the early stage of dysentery are not really solitary follicles, but new formations. The right opinion, however, appears undoubtedly to be that which has been stated in the previous paragraph; namely, that the solitary glands are enlarged in the early stage of the catarrhal form of dysentery, but that this is, after all, only part of a general process of inflammation which affects the whole mucous membrane, and even the deeper parts of the intestine.

A little later, the mucosa softens down with the increasing infiltration of pus-cells, and ulcers are formed. A peculiar appearance is now produced by the changes round the solitary follicles. Their roofs give way, and minute round holes are produced, each of which leads into a small cavity having in its interior the substance of the follicle, which is isolated from its attachments and in fact forms a small slough. The destruction of the mucous membrane, however, is by no means confined to those parts which surround the follicles. It also takes place between them, so that for a time each orifice is surrounded by a little ring, which appears to be raised, and looks like a deposit upon the surface instead of being a remnant of the original tissue. Even when the ulcers have increased in size, and run together so as to form large patches, there remain irregular islands of still undestroyed mucous membrane, which are of a bluish-red colour and covered with grey or greenish layers of tough mucus. The ulcerated surfaces themselves have a yellow or yellowish-red colour; their floor is formed by the submucous tissue.

*The sloughing or necrotic form.**—Widely different are the appearances in the other form of dysentery, which, in obedience to the teaching of Virchow, all modern German writers call *diphtheritic*. To digress for a moment—this term, diphtheritic has now two meanings, which must be carefully distinguished from one another. In the first volume *diphtheria* was described as a specific disease, generally affecting the throat. But, in Virchow's sense, "diphtheritic" inflammation of a mucous membrane or of the skin has not necessarily anything to do with that disease; it is a morbid change of a particular kind, probably dependent upon intense irritation of the part. This pathological condition requires to be distinguished by a name of its own, but the ambiguity of the term "diphtheritic" is undoubtedly apt to create confusion in the mind of an English reader. Diphtheritic in Virchow's sense does not imply the presence of a false membrane,† but it does imply a putrid, destructive, more or less necrotic process. See the exposition of this point in the chapter on Inflammation (vol. i, p. 62), and in that on Diphtheria (vol. i, p. 260).

In this form of dysentery the whole thickness of the intestine is from the first affected in a marked degree. Even the serous surface is intensely injected, so that it is of a dark bluish-red colour. The bowel feels hard as well as massive. In its interior there is a thin reddish fluid, or, in some parts, a little fæcal matter. Its lining is of a greyish-red colour and here and there exhibits what looks like a raised deposit on its surface. This, in its earliest stage, is present only on the summits of the ridges formed by the mucous membrane. It may be seen forming transverse lines in the ileum, the lower part of which is commonly affected in this form of the disease. Lower down, in the cæcum, the seeming deposit becomes more extensive; and in the colon it occurs in large patches, or may even occupy the whole surface; being, however, broken up into plates by deep grooves or fissures. The parts affected in this way look dry and granular; to the touch they feel rough and hard. Their colour varies to some extent with that of the intestinal contents, which possess the power of staining them. Thus they may be yellowish, greenish, dark red, or even black. On making a section through the intestine one finds that it is enormously thickened; the muscular layer is much thicker than natural and folded in and out. But the most striking change is in the internal coat. Instead of the dry rough substance above described being a deposit on the surface of the mucous membrane, it is now seen to take the place of that structure and perhaps even of the submucous tissue also. The whole thickness of the intestinal wall within the muscular coat may thus be made up of a tough, homogeneous, yellowish-red material, which offers considerable resistance to the knife, and in which the natural strata can no longer be recognised. Even under the microscope one can hardly make out the original elements of the tissues. One sees nothing but a mass of extravasated blood, of hard amorphous fibrinous exudation, and of pus-cells in greater or less number. But Heubner states that in very thin sections, lines of epithelium arranged in double rows may be identified as the remains of Lieberkühn's tubules.

According to recent observers, however, the fibrinous material, described as amorphous, contains elements of great importance. It is highly granular,

* *Synonyms.*—Red dysentery—*Germ.* Diphtheritishe Ruhr—Dysenteria facta (Sennert), a translation of Δυσεντερία ἤδη γεγενημένη.

† "Muco penitus absterso, nulla membrana superficiem quæ tegeret inventa est" (Hewson).

and these granules appear, with the aid of high powers, good illumination, and appropriate staining, to be really bacteria.

It is evident that the apparent "deposit" or "membrane" in the diphtheritic form of dysentery is really formed by the exudation of fibrin and the extravasation of blood into the tissues themselves, not upon the surface of the mucous membrane. This process can have but one termination—the death of the affected structures. Accordingly, whenever there has been time for the occurrence of further changes, eschars are found; and at a still later period these break down into shreds or detritus and are cast off, exposing deep and ragged ulcers of dark green or brown colour. For detailed information of the morbid anatomy of dysentery, illustrated by photographs and coloured plates, the reader is referred to the valuable monograph on the diarrhœa and dysentery observed during the war of the Rebellion in 1861–66, by Surgeon-Major Woodward, U.S.A. Among early accounts of autopsies may be mentioned one by Hewson, and two others (likewise communicated to Sir Geo. Baker) by Dr Wollaston.

The changes presented by the intestine in the two forms of dysentery are so different in appearance that one would at first sight be disposed to regard them as belonging to different diseases. It is, however, certain that they merely indicate different degrees of severity in the morbid process. For they are very frequently found side by side in the same intestine, the more intense inflammation being present in those parts (commonly the rectum or cæcum) which were earliest attacked. And this being so, the fact that the solitary follicles appear not to be specially affected in even the earliest stage of the diphtheritic form affords strong corroborative evidence in favour of the view that these glands are not the seat of any primary or special change, even in the catarrhal form of dysentery.

Later anatomical course.—In severe cases, abscesses also arise in the submucous tissue, and these may burrow, so that undestroyed parts of the mucous membrane over them are detached in the form of bridges, and when pressure is made upon these, the pus exudes at several distant spots. The inflammation extends at one or more points through the muscular coat, and perhaps penetrates to the serous membrane, so that perforation of the bowel occurs, with consequent peritonitis; or the connective tissue at the back of the bowel may be reached by the ulcerative process, the result being that a fæcal abscess is formed. In one case at Guy's Hospital such an abscess formed a large tumour filling the left side of the abdomen, extending into the psoas muscle and the spleen (which was sloughing), and denuding the ilium of its periosteum over a considerable space.

It is probable that the most intense form of dysentery, in which the whole large intestine and the last few feet of the ileum are uniformly affected, is necessarily a fatal disease. But if the morbid changes in the bowel be not too extensive and severe, recovery may take place, even though in some parts they may have assumed a diphtheritic character. In the catarrhal form, the inflammation probably often subsides before any breach of surface has occurred. When ulceration takes place, and the ulcers subsequently heal, a thin membrane is formed over their surface, which is at first depressed below the level of the parts that had been unaffected; but this difference gradually becomes less obvious, and ultimately disappears. In the diphtheritic form of dysentery the ulcers left by the separation of the sloughs become covered with granulations; their undermined edges adhere to the submucous tissue, and thickened and

irregular cicatrices gradually develop themselves. The cicatrices which follow dysentery are always of a dark grey or even black colour, which probably results from a chemical reaction between the colouring matter of blood extravasated during the course of the disease, and the sulphuretted hydrogen, which is one of the gases contained in the interior of the bowel.

In many cases, however, there is no definite termination of the morbid process, either in the death of the patient, or in his recovery. The disease passes into a chronic form, and may continue for months or even years. The ulcers remain unhealed, or fresh ones may form in succession as others cicatrise over. But, as Dr Maclean points out, it is a mistake to suppose that unhealed ulcers necessarily remain present so long as symptoms of dysentery in the chronic form persist. Cases are often observed in which not a single breach of surface is discoverable after death. Numerous black cicatrices may be seen, but the essential pathological change is an atrophy of the coats of the bowel, the glandular structures having disappeared, and the wall being so attenuated as to be transparent.

Sporadic, endemic, and epidemic forms.—Probably there is no part of the world in which dysentery does not sometimes occur *sporadically*, but in London it is now decidedly a rare disease. Formerly it was common there and in many other parts of England, and has become rare along with ague and other results of malaria. Cardinal Wolsey died of English dysentery. In 1762 it occurred epidemically in London, and was described by Sir George Baker. The account of it by the late Dr Baly, as a local disorder in Millbank Prison, is, we may hope, the last. At present the cases met with by London physicians are imported, and are mostly confined to soldiers and sailors. At the Seamen's Hospital, Greenwich, dysentery may still be studied in its less acute forms.

There are, however, countries in which it is exceedingly prevalent, so that it may be said to be *endemic* there. These are Greece and other coasts of the Mediterranean sea, many parts of India, the coast of China, almost all tropical Africa, Madagascar and many islands, and Central America.

Dysentery sometimes affects large numbers of persons as an *epidemic*, and then is apt to assume a severe type. Heubner, indeed, is disposed to think that a primarily "diphtheritic" dysentery never occurs sporadically. But among fourteen or fifteen cases of acute and rapidly fatal dysentery that in the course of the last twenty years have from time to time occurred at Guy's Hospital, there have been several in which the inflammation showed the most marked pseudo-diphtheritic character. It is therefore evident that, so far as concerns the anatomical changes in the intestine, no absolute distinction exists between sporadic and epidemic dysentery.

As a rule, the disease is much milder when sporadic than when endemic or epidemic.

Ætiology.—The origin of *sporadic dysentery* is commonly attributed to the ingestion of irritating articles of diet, such as unripe fruit, decomposing meat, or bad water. But Trousseau and others have disputed the correctness of this opinion. It has been urged that when anything which disturbs the intestine is swallowed, active peristaltic movements are excited which expel it from the body, and that ingesta are not likely to leave the small intestine unaffected and to exert an irritant action first upon the cæcum and colon. But it is well known that this very thing occurs in cases of poisoning by bichloride of mercury, in which violent inflammation and even ulceration of the cæcum and colon have repeatedly been observed although the small

bowel has escaped entirely. Moreover, there is some reason for thinking that one factor in the causation of dysentery may be a habitual torpidity of the large intestine. This is a point on which Virchow lays stress; he remarks that the cæcum and the flexures of the colon, which are particularly liable to be affected by the disease, are also especially apt to become loaded with fæcal masses. And it is evident that if there is any irritant substance among the intestinal contents its action must be favoured by their retention in the bowel as the result of imperfect peristalsis. Now, Annesley long ago pointed out that in India the disease often commences with the characteristic signs of morbid accumulation in the large bowel. And in connection with this, the suggestion of Dr Dickinson may be remembered, that certain transverse ulcers in the colon, which are not uncommonly found in the bodies of those who have died as inmates of London hospitals, and which have been known to perforate the bowel or to cause a fæcal abscess, are due to irritation from the fæces. Another cause to which sporadic dysentery has been attributed is cold. Heubner alludes to a case which occurred in a washer-woman who had been standing for a long time with the clothes about her body wet through.

As regards the causation of *endemic and epidemic dysentery* very little is as yet certainly known. On each side of the equator, to about 35° or 40° of south or north latitude, there are in all parts of the globe territories in which it prevails, but it is a great mistake to suppose that it is endemic in every country with a hot climate. Hirsch mentions Gujerat in India (particularly the peninsula of Kathiawar), and Senegal in Africa, as regions in which the heat is intense, but in which there is no dysentery. So, again, Singapore is said to be free from the disease, which yet exists in all other parts of the peninsula of Malacca. The rainy season is generally the period of the year at which dysentery is most apt to prevail. The alternation of hot days and cold nights has been supposed to have a special influence in producing it.

In temperate climates, epidemic dysentery occurs, at the present day, chiefly in camps and armies. It was very fatal in 1854 among the British troops engaged in the Crimean war, as it had been among their forefathers in 1415 before the battle of Agincourt. It raged terribly in the armies of the United States in 1862–65, and in the camps of the Northern prisoners. It was again prevalent in 1870, during the campaign between France and Germany, particularly among the miserable fugitives who sought refuge in Switzerland after the dispersal of Bourbaki's army.

Even in time of peace, the large cities of Europe were formerly liable to epidemic dysentery, and Paris suffered severely from it as lately as 1859, after having been free for a hundred years. In London, in the seventeenth century, it is believed to have caused from 1000 to 4000 deaths annually; in the following century it gradually disappeared, the last general epidemic being that of 1762. In Millbank prison, however, small outbreaks of the disease were of frequent occurrence until a few years ago.

While this second edition is passing through the press, we have accounts of a severe local epidemic of dysentery in a lunatic asylum in Ireland. At a meeting of the Royal Irish Academy of Medicine (December 2nd, 1887), Dr Conolly Norman reported that of the 1100 inmates 120 were attacked, and 22 died. In two of the latter cases there was perforation of the colon, and in a third multiple abscesses were found in the liver.

As in the tropics, so in temperate climates, the autumn is the season at

which the disease is most apt to break out. The years of its prevalence have sometimes been exceptionally hot.

Dysentery occurs in persons of all ages. In this country the sporadic form, or a closely related disorder, is not uncommon in infants who are brought up by hand. When it is epidemic, it attacks especially those who are weak or old, or whose health is impaired by intemperance.

The conditions which are concerned in the production of endemic dysentery have been studied with the utmost care and attention; but, as one might have anticipated, it is scarcely possible to make out which of them is really the exciting cause of the disease.

Certain facts appeared at one time to point strongly to the conclusion that it was a telluric poison, like that which generates intermittent fever. Thus it has long been known that even in temperate climates, and still more in the tropics, the countries in which dysentery prevails are also those in which ague is common. Indeed, the two diseases frequently occur together, in the same patient and at the same time; and Dr Aitken remarks that if a boat's crew be sent ashore in a tropical climate, and exposed to paludal miasmata, the probabilities are that of the men returning on board some will be seized with dysentery and others with remittent fever. So again, the gradual extinction of dysentery within the last two centuries in England has coincided with a very marked decrease in the amount of ague throughout the country, and with its complete disappearance from certain parts. It was, indeed, known that the very same miasm could not be the cause of both diseases, for dysentery prevails in many places in which there is no intermittent fever. Still it appeared probable, until quite recently, that the real cause of the former disease was some poison allied to that which produces the latter.

From his investigations at Millbank prison, Dr Baly came to the conclusion that the epidemics of dysentery there were due to a malaria arising from the soil; and Dr Maclean subsequently endorsed this opinion, and further attributed the poison to the decomposition of organic matter in the ground. In the meantime, however, the course of events at Millbank has proved conclusively that Dr Baly was wrong. In the year 1854 the prisoners ceased to be liable to dysentery; and during the next eighteen years (up to 1872) one death only occurred from that disease or from diarrhœa. Now, as Mr de Renzy has shown, one, and one only, change in its hygienic arrangements has coincided in time with this improvement in the sanitary state of the prison. Formerly, the water which the convicts drank was taken directly from the Thames as it ebbed and flowed beneath the walls. But on August 10th, 1854, the artesian well in Trafalgar Square was made the source of water supply to the prison, and has since been exclusively used. The change was effected in the middle of a cholera epidemic; six days afterwards the disease suddenly ceased. Enteric fever, too, no longer attacks the convicts, and the death-rate has declined to an extraordinary extent. It seems impossible to avoid the conclusion that the exciting cause of dysentery in Millbank prison was the Thames water; and in all probability the noxious ingredient was derived from the sewage it contained.

Another series of epidemics, which have been traced to a somewhat similar origin, occurred in the Cumberland and Westmoreland Asylum. In 1864, chiefly between May and August, twenty-six persons were attacked with dysentery, and in March, 1865, five others. For a long time Dr Clouston, the medical superintendent, was completely at a loss to account for the disease. It had often occurred to him that the cause might be connected

with the distribution of the sewage of the asylum, which, after being thrown into a large tank, was allowed to flow over a field about 300 yards distant. But it was not until August, 1864, that an offensive smell was noticed at the asylum during several hot and sultry evenings; and Dr Clouston then had the sewage carried away in a covered drain to a distance; from that time no fresh cases of dysentery occurred. An investigation was then made as to the exact meteorological conditions which had existed during the prevalence of the epidemic, and it was found that within a week before the day on which each patient fell ill there had always been either hot sultry evenings with no wind in the night, or northerly winds which blew from the direction of the field which was being irrigated. Male and female patients, too, were attacked at different times, according as the exact direction of the wind was such as to carry the sewage emanations either to one or to the other of the parts of the building which the two sexes severally occupied.

The probability that these observations pointed to the real cause of the dysentery was greatly increased by the fact that the five cases of dysentery in March, 1865, all occurred within a week after the sewage was again allowed to flow over the field, during one calm night, when the direction of the wind was towards the asylum. The evidence may be said to have amounted to proof in the year 1868. There had then been two years' immunity from dysentery, and, the most approved precautions having been taken, it was determined to run the sewage over another field. Two months later six patients were attacked with dysentery and diarrhœa within a few days of each other; they were all in that part of the asylum nearest the field, and the wind had been blowing towards it continuously for eight days before the outbreak occurred. It must be mentioned that the subsoil was a stiff clay, through which water could not penetrate, and which therefore was entirely unfit for irrigation purposes.

It must be admitted that these interesting observations do not appear to be supported by the recorded experience of military surgeons. But, on the other hand, their experience is not in any way adverse; and in time of war, as well as in tropical epidemics, the conditions are generally so complicated as to defy analysis. Dr Chevers, indeed, has expressed the belief that much of the dysentery (as well as cholera) occurring on board vessels in the port of Calcutta is caused by men drinking the water taken up in buckets over the ship's side, this water being loaded with sewage and the products of putrefaction. And Heubner was told by several military surgeons, on whom reliance could be placed, that where many severe cases of dysentery were crowded together the disease was often spread by the latrines, and ceased when the proper precautions were taken with regard to them. Prof. Maclean says that in India the barrack-rooms most exposed to the effluvia from latrines always furnish the largest number of dysenteric cases.

We must believe, then, that the cause of dysentery may be, and probably always is, the entrance into the body of some organic matter conveyed either by drinking-water or through the air. So far dysentery resembles enteric fever and cholera.

Contagion.—The next questions are whether, as in these diseases, the organic matter is a specific poison, and whether this poison undergoes a process of self-multiplication in the human body, so that the discharges possess special powers of infecting others with dysentery. Probably these questions cannot yet be finally answered. All observers, however, are agreed that the disease seldom or never passes from the sick to those who are

attending on them. And Heubner lays stress on the fact that it has hardly ever been known to spread from the military to the civil population in time of war.

In 'Ziemssen's Handbuch,' however, dysentery is placed among the infective diseases, and Liebermeister explains that he regards it as belonging to the same class of maladies as cholera and enteric fever. He goes on to admit that it is a local disease, and that all its symptoms are dependent upon the intestinal inflammation; but, as he remarks, gonorrhœa and the soft chancre are *local*, and yet *infective* in the fullest sense of the term. Still the presumption is surely in favour of the view that no specific contagion is concerned in the production of a disease which is admitted to be a local inflammation; and this presumption is greatly strengthened by the fact that epidemic and sporadic dysentery cannot be separated by any strict boundary-line. It has indeed been urged that between these two forms of the disease the same relation exists as between Asiatic and English cholera; and since the occurrence of a simple variety of cholera does not prevent the acceptance of the view that, when introduced from the East, this malady is due to a specific contagion, it is argued that the existence of sporadic dysentery is no bar to the infective theory of epidemic dysentery.

It would appear, too, that strong evidence in this direction has been afforded by an epidemic which occurred in Norway in 1859. Dysentery had not prevailed there for half a century, and as the population was scattered, and the ground very broken, there were unusual opportunities of tracking its course. The disease was studied with great care by Homan and Hertwig, and they believed that they established the fact of its spreading by a definite contagion, which was even conveyed by healthy persons from infected places to other spots where the disease had not before appeared. It does not seem that they traced the poison to any particular source, but the analogies of enteric fever and cholera evidently render it in the highest degree probable that, if there really is a contagion which multiplies itself in the human body, this reaches the outer world in the alvine evacuations.

With regard to the period of *incubation* in dysentery, Dr Clouston states that one of his patients fell ill within three days after first inhaling the poison; but others were attacked as long as three, four, or five days after the cessation of opportunities for its entrance into the body. Homan and Hertwig state that in Norway in 1859 the period of incubation was from two to eleven days.

Symptoms.—Before the characteristic features of dysentery develop themselves, there is generally a period in which the patient suffers from simple diarrhœa, with more or less griping pain in the abdomen. This may last from three to five days, or even a fortnight. He then becomes worse and perhaps he shivers, or at least complains of greatly increased malaise and weakness. The abdominal pain is more severe and paroxysmal. The desire to go to stool becomes more and more frequent, and, instead of abundant liquid fæces, he begins to pass only very small quantities at a time, with violent straining, and burning pain in the rectum. He is then said to suffer from "tenesmus," but although this term is universally employed there are hardly two writers who are agreed as to its exact signification. Some mean by it the peculiar sensation that there is "something in the rectum that wants to come away," others the straining, and others the burning pain; while others again include two or all in the connotation of the word. But as all these symptoms are constantly associated together it is really not a

matter of importance whether tenesmus means one or another of them.* It is taken by Dr Woodward as the most characteristic of the symptoms of dysentery. So incessant may be the call for relief that the patient goes to stool twenty or thirty or even as many as two hundred times in the twenty-four hours. Or he may sit there for half an hour at a time, straining violently, but passing little or nothing. In a number of cases of dysentery occurring in camp, Heubner had the total daily evacuations collected. The quantity passed by each patient was found to be only from 28 oz. to 42 oz.

The evacuations in dysentery often contain no fæcal matter whatever. Small hard scybalous masses may be voided occasionally, but the most careful recent observers are agreed that these are comparatively seldom seen. Thus the paradoxical expression of Stoll is justified, who described dysentery as *morbus alvum occludens.* But in some cases fluid fæcal matter is passed from time to time throughout the whole course of the disease, and this is said to be rather an unfavourable sign, as showing that the upper part of the large intestine, or even the small intestine, is involved. In most cases, however, the only result of the straining efforts of the patient is that he passes two or three drachms of gelatinous mucus, colourless, or more or less deeply stained with blood. Or there may be membranous shreds of mucus, and small clots, mixed with more or less of the fluid fæcal matter. Or, again, pure blood may come; or a thin reddish fluid, which has floating in it a number of little yellow or red masses, soft, and looking like pieces of sodden meat. This kind of discharge is often described as resembling the washings of meat, and was formerly called *lotura carnea.* The masses suspended in it were supposed to be cast-off portions of the intestinal mucous membrane, but it is now known that this is not the case. Dr Goodeve, in Calcutta, is, indeed, said to have found membranous sloughs from half to one inch or more in diameter, in the sediment obtained by washing the evacuations after the eighth to twelfth day of the disease. But, according to Heubner, it very rarely happens that masses are passed the nature of which can be recognised. Another dysenteric product is pus, which may occasionally be discharged pure and odourless, and just as if it had come from an ordinary abscess, but which is much more commonly mixed with fluid fæcal matter and blood. In some cases, again, a substance is voided which looks like frogs' spawn or boiled sago. This consists of rounded bodies which were once thought to be sloughs derived from the solitary follicles. Heubner, however, says that they are too large for this, and that they consist of mucus. Lastly, the discharge may be a brownish-red, or blackish fluid, of a most horribly offensive odour; this is an indication that sloughing is going on in the intestine. It is to be noted that in all but the earliest stages of dysentery the matters passed from the bowel have an exceedingly disagreeable odour, which Parkes regards as peculiar to the disease.

The abdomen is not at first distended, but it may become so as the disease advances. Tenderness may be altogether absent, or pressure over different parts of the large intestine may give more or less pain. In some cases one can obscurely feel an induration in the course of the bowel, and it has even been suggested that the extent to which the upper part of the colon is affected may be determined in this way.

Another symptom which is sometimes present in severe dysentery is

* Tenesmus (τεινεσμός, from τείνω): "Est autem affectus hic continua et implacabilis desidendi cupiditas (Paulus Ægineta apud Stephanum)—Quod τεινεσμόν Græci vocant: in hoc frequens desidendi cupiditas est, æque dolor ubi aliquid excernitur" (Cels., lib. iv, cap. 18).

strangury. It may be necessary to have the urine drawn off by a catheter.

The most marked general symptom is the great prostration. The patient not uncommonly faints while he is at stool. Anæmia is very rapidly produced, and the face assumes a pale, waxy look.

The febrile disturbance is generally slight. Even in severe cases the temperature may be normal or below normal, but sometimes it rises to 100° or 101°. The pulse is not much quickened, except in certain epidemics formerly characterised as "inflammatory dysentery." The patient often makes great complaint of thirst, and although his appetite is not always lost, griping pain is frequently brought on by food, so that he is unwilling to take any but the blandest nourishment. Cold liquids frequently have the same effect, and therefore it is well that what the patient drinks should be lukewarm.

Diagnosis.—A question of great importance is whether dysentery is always attended by the very striking train of symptoms which have just been described. Writers mention that some patients pass no blood, and it is said that certain epidemics are characterised by the absence of this symptom. Again, it is well known that tenesmus and the other symptoms which are constantly associated with it depend upon disease of the lower part of the large intestine, and that they are absent when the inflammation is limited to the cæcum and the upper part of the colon. Dr Clouston says that in the epidemic which he observed at the asylum near Carlisle, some patients experienced scarcely any pain throughout the whole course of the disease, and at first, having no fever or want of appetite, they refused to believe that they were ill, although they were passing glairy mucus mixed with blood. It does not appear that tropical dysentery is ever latent when the disease is epidemic. We have, however, had at least two well-marked instances of fatal sporadic dysentery, in each of which there was very extensive diffused ulceration of the large intestine, the presence of which had been entirely unsuspected during life. Both patients died in Guy's Hospital, the one of an enormous hepatic abscess, the other of a large abscess in the left iliac fossa, resulting from extension of ulceration from the bowel into the retro-peritoneal connective tissue. Dr Dickinson has recorded a similar case, that of a woman who died in St George's Hospital of abscess of the liver, and who, during the week which she passed as an inmate of the hospital, was so constipated as to require aperient medicines. She would not allow that she had ever had any looseness of the bowels, but after death the upper part of the large intestine was in a state of ragged ulceration.

In at least three cases of acute dysentery that have proved fatal in Guy's Hospital the disease was supposed during life to be *enteric fever;* the characteristic symptoms were either absent or passed under the name of diarrhœa. It is worthy of inquiry whether a similar latency or obscurity of symptoms may not sometimes occur in epidemic dysentery, both in India or in temperate climates. We shall hereafter see that the question is one of considerable interest in reference to the causation of abscess of the liver.

On the other hand, even when all the symptoms of dysentery are present, the diagnosis should be made with more caution than would appear necessary from the statements of writers who speak of it as presenting no difficulty whatever.

In young persons, and particularly in male children, one must always

bear in mind that *intussusception* causes very similar symptoms, although they no doubt begin in a different way. In this country more than one case of intussusception has been mistaken for dysentery, and this very serious error must often have been committed in those parts of the world in which the latter disease, being endemic, is likely to have its presence taken for granted.

Again, in persons more advanced in years, it very frequently happens in England that *cancer of the rectum* is overlooked, and that its symptoms are attributed to dysentery. I know of so many examples of this that I would almost lay it down as a rule that a supposed case of chronic dysentery in a middle-aged or old person, who has not been out of England, is really one of local disease of the rectum, most probably cancer. A digital examination generally clears up all doubt as to the nature of the disease; and very likely it would be possible in most cases to determine by careful investigation that the blood which is passed during defæcation is not intimately blended with the motion, but comes before or after it.

Progress and event.—The course taken by dysentery varies much in different epidemics, as well as in individual cases; we shall presently see that it may to a very great extent be modified by treatment.

In favourable cases, the symptoms quickly begin to decline. First the pain and tenesmus pass off, and then some of the evacuations begin to contain fæcal matter. Formed fæces may for a time be passed alternately with blood and mucus.

An occasional *sequela* is an affection which somewhat resembles acute rheumatism. According to Homan and Hertwig, it attacks several joints at the same time, but these do not become very tender, nor is there much swelling. The febrile disturbance is moderate, and the affection subsides in three or four weeks. Parotitis is also said to occur sometimes during convalescence from dysentery.

In cases which are about to terminate fatally, the prostration passes into collapse. The features become sunken, while the body is covered with cold sweat, and exhales a fœtid odour. The urine may be suppressed. The tongue and lips are covered with sordes, hiccup sets in, and a painful sense of constriction of the epigastrium is complained of. The fæces are passed involuntarily, the anus and penis become excoriated, and the lower part of the rectum is often prolapsed. Bedsores are formed, if the patient should live long enough. Consciousness is often retained to the end, but in some cases the mind wanders; and during the last few hours all pain may cease, so that the patient fancies he is doing well, or his delirium assumes pleasing forms. Before death the temperature of the body often rises considerably.

Prognosis.—With regard to the grounds on which a forecast must be based in a case of dysentery, there is little to say that has not been implied in preceding paragraphs. The severity of the disease is proportional to the extent and intensity of the local inflammation, but the latter are nevertheless to be measured during life by the gravity of the constitutional rather than of the local symptoms. Tenesmus and pain may be slight or even absent, and yet the patient may be in very great danger; or his sufferings may be extreme, and yet there may be no grounds for alarm if the affection is limited to the rectum. But the appearance of his countenance, the state of the circulation, and the presence or absence of symptoms of collapse, generally allow a correct judgment to be formed as to the probable issue of the case. The

rate of mortality varies greatly in different epidemics. In Dr Clouston's Asylum it was 64·5 per cent. This is enormous, for even in Western Africa the proportion of deaths to admissions appears from a table drawn up by Sir Alexander Tulloch to be only 14·2 per cent.; and the death-rate was higher than in any other of the intertropical stations for British troops.

In epidemic dysentery the prognosis must also be greatly influenced by the presence of *scurvy* as a complication. This is exceedingly frequent, so that many observers speak of scorbutic dysentery as a distinct variety of the disease. But it is needless to describe the symptoms of such a form in detail, because they are in fact a combination of those which belong to the two maladies separately. Only it may be worthy of mention that when scurvy is present the course of the disease is slower than in other equally severe cases in which it is absent. The patient almost always lives three weeks, and often as long as three months.

Even the most severe form of uncomplicated dysentery is seldom rapidly fatal. Death rarely takes place within the first week, or before the ninth or tenth day. At Guy's Hospital, however, some years ago a case occurred in a child who died after five days' illness. She was in the hospital for chorea when she was attacked, and it was a question whether the sulphate of zinc which she was taking could have been concerned in causing the intestinal inflammation. The symptoms were so severe that the case was mistaken for one of Asiatic cholera, that disease being prevalent at the time.

In the later stages of dysentery, perforation of the intestines sometimes occurs, setting up fatal *peritonitis*. In one case this was observed in a patient at Guy's Hospital. He was in a surgical ward for disease of the knee, when in the month of August he was attacked with "severe diarrhœa." This continued, and he died at the end of a fortnight. The large intestine was ulcerated and sloughing in its whole length, and at one spot the transverse colon was perforated. But in three other cases of sporadic dysentery there has been acute peritonitis without any perforation being discovered to account for it. This seems not to have been hitherto observed in epidemics of the disease.

In other cases, as already mentioned, acute dysentery passes into a *chronic form* of the disease, which may last for months, or even years. It is not stated by writers of experience that chronic dysentery may arise without being preceded by any acute stage; but, if not, this disease is an exception to a rule which seems to obtain in the case of all other maladies. In chronic dysentery the discharges still have to some extent the peculiar odour. They are for the most part liquid, but they vary in character from day to day. Sometimes they may contain tolerably natural fæcal matter, at other times they consist of a blood-stained mucus or serous fluid. The patient does not gain flesh, but becomes more and more emaciated. The appetite is generally capricious. The tongue is red and glazed. Abscess of the liver not infrequently forms in these cases; or death may arise from pneumonia, phthisis, Bright's disease, or lardaceous disease of the viscera; or, again, a fæcal abscess may make its appearance in the iliac fossa, or elsewhere. Peritonitis from perforation may also occur, even at this period.

Chronic dysentery is by most writers regarded as an exceedingly intractable disease, and one which generally proves fatal at last, the patient dying by exhaustion, with febrile symptoms of hectic type, even if no complication should arise to cut short his existence. Dr Ward, however, says,

from his experience at the "Dreadnought," that in the majority of cases recovery at length takes place, if they are judiciously treated. Even then, however, the bowels often remain irritable and easily disturbed for a very long period afterwards, it may be for the rest of the patient's life.

Prophylaxis.—Before entering on the treatment of dysentery, we may ask whether any preventive measures can be adopted when the disease prevails, to protect those who have hitherto escaped. The answer to this question is that all general hygienic measures must be strictly attended to, overcrowding being particularly avoided. As a precaution, it is proper that the evacuations of those who are sick should be disinfected with carbolic acid, and removed without delay, although, as we have seen, it has not yet been proved that they contain a specific poison. Those who are still well must clothe warmly, wear a flannel binder, and be careful to avoid chills; they should eat and drink moderately, taking no strong wine or spirits, and they should not allow the bowels to become constipated. Ripe fruit need not, as a matter of course, be excluded from the dietary.

Treatment.—The therapeutics of dysentery must be based upon a knowledge of the natural course of the disease. Now, Dr Austin Flint once observed ten cases in succession in which no medicine was administered except a little tincture of cinchona as a placebo; and he found that the mean duration was eleven days and one fifth; the most protracted case lasting twenty-one days, the shortest six. Or, reckoning from the first dysenteric evacuation (instead of from the commencement of illness) he obtained a mean duration of eight and a half days, the maximum and minimum being twelve and five days respectively. Previously the same observer had analysed forty-nine cases, which had been treated, some with calomel and opium, some with opium alone, some with castor-oil, and others with astringents. The mean duration was almost exactly the same in these cases as in those in which the disease ran its natural course.

These particular figures are evidently applicable only to dysentery as it occurs in the city of New York, and in strictness only to the actual period within which the observations were made. It is clear that Dr Flint's cases were mild ones; one point on which he lays stress is that the complaint showed no tendency to become chronic and that relapses never occurred, although he allowed his patients to eat solid food as soon as they chose to do so. Very different is Prof. Maclean's account of dysentery as he saw it in India. "Speaking from large experience," he says, "I affirm that complete restoration to health, by the unaided efforts of nature, is of extremely rare occurrence; the disease either destroys the patient or it passes into a chronic form."

But it would seem that Dr Flint's observations fairly establish the uselessness of the various remedies employed in his earlier series of cases. And it is therefore a gratifying circumstance that we have for dysentery another medicine of which he does not seem to have made trial, but which is proved by the concurrent testimony of a large number of observers to possess the power of cutting short the disease and even of curing cases that would otherwise have terminated fatally. The medicine is ipecacuanha. The root of this plant was first employed as a remedy for dysentery in Brazil, where it is indigenous. Towards the end of the seventeenth century it was introduced into France, it was successfully given to the Dauphin, acquired a great reputation, and was known as the *radix antidysenterica*. In India it was used even before mercury came into vogue, and of

late years it has become the staple remedy.* The method of administering it which is now generally adopted was introduced by Surgeon Docker, of the 7th Royal Fusiliers in 1858, and first tried by him in the Mauritius.

The patient having been put to bed, twenty-five to thirty grains of powdered ipecacuanha are given to him in as small a quantity of fluid as possible; a little syrup of orange-peel covers the taste as well as anything. Some surgeons think it of importance that thirty minims of laudanum should be given half an hour before, in order to make the stomach tolerant of the ipecacuanha; but Dr Maclean says that he has seen the latter drug well borne without any such precaution having been taken. After the dose, the patient should keep perfectly still, and abstain from drinking for at least three hours. If thirsty, he may suck a little ice, or have a teaspoonful of cold water at a time. Under this management he seldom complains of excessive nausea, and vomiting rarely sets in within two hours. A poultice is in the meantime placed over the abdomen, or a piece of spongiopiline, wrung out of hot water with a little turpentine sprinkled over it. Afterwards some bland nourishment is given. In from eight to ten hours, according to the urgency of the symptoms and the effect of the first dose, the ipecacuanha is repeated, its quantity being somewhat reduced, but with the same precautions as before.

"All who have had opportunities of trying this mode of treating dysentery," says Dr Maclean, "can bear testimony to the surprising effects that often follow the administration of one or two doses of ipecacuanha. The tormina and tenesmus subside, the motions quickly become purulent, blood and slime disappear, and often after profuse action of the skin the patient falls into a tranquil sleep, and awakens refreshed." The remedy may, however, require to be continued in diminished doses for some days; and even after the stools have regained a healthy appearance, it is well to administer ten or twelve grains at bedtime for a night or two.

Even when the powers of life are very low, this remedy may sometimes be given with safety and success. Dr Maclean mentions the case of a lady who landed at Calcutta, having come from Madras, and who was so exhausted that her voice was scarcely audible. With some misgiving he gave twenty-grain doses at intervals of eight hours; after the third dose she was out of danger.

When severe vomiting follows the administration of ipecacuanha, Dr Maclean says that coexistent liver disease may be suspected, or complication with malarious fever. In the latter case he advises that quinine in ample doses should be alternated with the ipecacuanha.

In mild cases, he recommends that the treatment should be commenced with a hot bath, which must be brought to the patient's bedside. He is to be kept in it until he feels faint, and after being rapidly but carefully dried he is to be put to bed, and to have a dose of fifteen to twenty grains of ipecacuanha. In some cases a few drachms of castor-oil, with a little tincture of opium, may be afterwards prescribed. A turpentine stupe should be applied to the abdomen, and repeated from time to time. According to Heubner, enemata of starch and opium often give great relief to the tenesmus in these mild cases.

The action of ipecacuanha in acute dysentery is as yet unex-

* "Dehinc ad radicem Ipecacuanha confugiendum, qua nullum præstantius aut tutius, cum vel sine sanguine, ad fluxus compescendos natura excogitavit remedium," 'Gul. Pisonis de Medicina Brasiliensi,' lib. ii, cap. xi, Lugd. Batav., 1648: quoted by Woodward.

plained; it must be regarded as a "specific." Dr Maclean supposes that it is an "evacuant," increasing the secretions of the alimentary canal. Its administration renders unnecessary the use of castor-oil, tamarinds, rhubarb, or the sulphates of potass or soda, which are recommended by different French and German writers. It is curious that even Heubner, writing in 1874, seems to know scarcely anything of the value of ipecacuanha, except as an emetic. But he has heard that it was used by some English surgeons with good results in the war between France and Germany.

Calomel has been definitely abandoned in the treatment of dysentery. Opium is believed by all experienced observers to be injurious, if systematically given. Venesection has been laid aside, as tending to exhaust the patient's strength to no purpose.

The value of ipecacuanha in treating dysentery is attested not only by individual experience, but by statistical results, which are most striking. In Bengal, under "the old system," the average mortality among Europeans during forty-two years was 88·2 per thousand; in 1860, under ipecacuanha, it was 28·87 per thousand. In Madras, the corresponding numbers were respectively 71 and 13·5. Surgeon Mee, at Madras, treated sixty-eight cases from the 44th Regiment "in the ordinary way," with a mortality of 6 (or 88 per thousand); afterwards he treated fifty-nine cases with large doses of ipecacuanha, and these all recovered. It is also asserted by the advocates of this remedy that, as its administration becomes more general, the number of chronic cases of dysentery diminishes year by year, and the development of hepatic abscess, as a complication, becomes less frequent. Hygienic improvements, however, may have had a share in this result.

Against these statements it is proper to set Dr Clouston's experience in the epidemic at the Cumberland and Westmoreland Asylum. He found the ipecacuanha treatment useless, even if it did not (as he appears to think) take away the last chance from one or two of the patients by causing vomiting that could not be stopped and prostration that was never rallied from. But, as already stated, this epidemic was one of a type far more severe than ordinary tropical dysentery.

Dr Baly says that ipecacuanha wholly failed in his hands. But it is a cardinal rule—although one which is too apt to be forgotten—that in all the worst cases of a disease a medicine may fail to produce any appreciable benefit, and yet it may be capable of curing those which tend but a little less surely towards a fatal termination.

In severe and malignant forms of dysentery, Dr Maclean recommends the solution of pernitrate of iron; he says that he has sometimes prescribed ten drops every hour with advantage. The patient must of course be sustained with nourishment, and stimulants may often be freely given. Dr Clouston found that milk boiled with a little flour, and allowed to cool, was taken more readily than anything else, and kept up the patient's strength. He also gave strong beef-tea, jelly and eggs, and wine and water.

In *chronic dysentery* an essential part of the treatment is removal to a better climate. In India it sometimes suffices to send the patient to the seaside; but more often a voyage to Europe is necessary. Persons invalided home on account of chronic tropical dysentery are occasionally seen in the London hospitals. In such cases rest in bed is a very important part of the treatment. Dr Ward has especially insisted on this fact, and on the necessity that the diet should be very carefully restricted, milk and farinaceous food

being the best that can be given. His experience at the "Dreadnought" has led him to believe that ipecacuanha is useless in chronic cases. But Dr Maclean says that exacerbations of a subacute character are of frequent occurrence in chronic dysentery, and that at Netley he has often given the ipecacuanha in suitable doses with the happiest effect. Our experience at Guy's Hospital has certainly been that this medicine is of great value long after dysentery has been brought home from the East. In at least one instance it did good, even in out-patient practice.

Dr Ward's patients were probably sailors, whose food on board ship had been such as must necessarily aggravate the disease to the highest point; and it is not surprising that they derived so much benefit from rest in bed and a milk diet, as to throw the effect of medicines into the background. Even when a person affected with dysentery is sent home as an invalid, with nothing to do but to take care of himself, the disease is very apt to become worse on board ship, unless special care is taken that he has proper food and puts on warmer clothing as he passes into a more temperate climate.

In some cases of chronic dysentery, astringents are of great value. Dr Maclean recommends the solution of pernitrate of iron, which, besides checking the discharges, removes anæmia. Krameria, catechu, hæmatoxylum, even tannic and gallic acids, may each be useful in turn. Marked benefit sometimes results from the administration of drachm doses of the extract of Indian bael, though this hardly accords with the opinion of Dr Maclean that even the fresh bael fruit is only efficacious in dysentery when a tendency to scorbutus is also present. Acetate of lead and sulphate of copper are sometimes useful. Dr Ward mentions one case in which enemata of nitrate of silver (gr. iv ad aq. ʒij) were repeated every night with good effects.

The experience of the American surgeons of ipecacuanha in the treatment of acute dysentery during the Civil War was not so favourable as might have been expected. It appears, however, to have been little tried. Dr King reported good results in the Confederate army at Richmond, and several other favourable results are referred to in the official report already quoted, but on the other hand its trial at Washington ended in disappointment, as was the case in an epidemic in South Carolina in 1868.

Acute catarrhal colitis.—There is a somewhat rare affection of the bowels, which is inflammatory in its character and acute in its course, yet distinct from ordinary acute diarrhœa on the one hand and from dysentery or any form of ulcerative enteritis on the other.

The following is an illustrative case. A previously healthy man, thirty-four years old, after exposure to cold and fatigue, was attacked with shivering and diarrhœa, accompanied by severe abdominal pain and tenesmus. The temperature rose to 103° F., and there was complete anorexia, scanty, high-coloured urine, a thickly-coated tongue, pains in the back and limbs, and slight febrile delirium at night. The symptoms were more severe than those of acute catarrhal dyspepsia (p. 318), and vomiting and nausea were absent. There was no source of irritation from food or other ingesta, nor from arsenical poisoning. Moreover, after the bowels had been emptied, there remained tenesmus, with passage of abundant clear, colourless, and inodorous mucus, entirely free from any trace of fæcal matter or bile, of blood, or of pus—a lientery, in the ancient meaning of the term. In rather less than a week

acute symptoms had subsided, but a somewhat tedious convalescence, with great muscular weakness, followed.

Such cases in a less marked degree are not very uncommon both in children and adults. They often appear to be due to direct chill, and though seated in the colon, correspond pathologically to acute gastric catarrh (p. 319) and acute muco-enteritis of the small intestine (p. 379). Warmth, fomentations, diluents and opiates seem to be the treatment indicated.

Ulcerative colitis.—A more common disorder, chiefly met with among women and children, is ulceration of the colon and rectum, running a subacute or chronic course, and resembling dysentery in the passage of blood and purulent or muco-purulent matter with the stools, in the tenesmus and most other symptoms. But it does not appear to depend upon any of the causes of true dysentery as above described, and does not occur epidemically. Accordingly Dr Eustace Smith and Dr Goodhart admit as dysentery in children only chronic cases which have been acquired in the tropics.

Some cases of severe and even fatal ulcerative colitis in adults are very difficult to explain. When cases dependent on typhoid fever and tubercle, from syphilis and dysentery have been excluded, a certain number still remain, which must at present be classified rather by their anatomy than their origin and pathology. Dr Hale White showed a good specimen of this morbid condition at the Pathological Society on December 6th, 1887. Another was recorded in the 'Guy's Hospital Gazette' for the 24th of the same month, and a third (which was diagnosed as general tuberculosis following acute catarrhal pneumonia) died in Miriam Ward about the same time.

Membranous colitis, sometimes called "diphtheritic" or "croupous," is a very rare affection. It is not to be distinguished during life from the so-called infantile dysentery or other sporadic cases of ulcerative colitis, unless casts of the intestine should be voided. Dr Goodhart narrates a marked case of this condition in a girl of eleven. There was high temperature, a purpuric rash, and excessive anæmia. After death the rectum and lower parts of the colon were found covered with a thick adherent membrane.

The true pathology of the *intestinal casts* which are sometimes met with is very obscure, and probably they are not all produced under the same conditions. Sometimes they are associated, not with diarrhœa or dysenteric symptoms, but with constipation, and may be passed for long periods of time. In 1857 Mr Hutchinson showed several specimens of this kind at one of the meetings of the Pathological Society ('Path. Trans.,' ix, 188); they were cylinders several inches or even feet long. Their walls were from one eighth to a quarter of an inch in diameter, yellowish brown, transparent and gelatinous. Under the microscope their surface showed a regular arrangement of round or oval pits, which had evidently corresponded with the mouths of the tubular glands of the intestine. Their substance was almost structureless, but embedded in it were large numbers of epithelial cells. When they had been retained in the rectum, the casts were apt to be changed into hard, white, round masses, about the size of nutmegs.

More recently Dr Goodhart exhibited some specimens to the same Society ('Path. Trans.,' xxiii, 98) which were almost exactly similar, except that they were solid. Many of them shaded off at their ends into a clear

colourless jelly. Both in Mr Hutchinson's case and in Dr Goodhart's there was much complaint of abdominal pain. It does not appear that medicinal or other treatment led to any definite good result.

Occasionally, as the result of severe ulcerative colitis, fragments of mucous membrane are passed. These are not mere casts, like those just mentioned, nor dysenteric sloughs, nor portions of the entire gut thrown off as in cases of invagination (*infra*, p. 409). At the present time a patient is lying in John Ward who had at first an enlarged liver with jaundice and pyrexia, but afterwards passed, on several occasions, fragments of mucous membrane apparently from the colon, one of which formed a complete ring and showed the tubules of Lieberkühn clearly. There was very little hæmorrhage, and none of the distinctive characters of dysentery. After being extremely ill, the patient, a man of about forty, is now gradually recovering.

INTESTINAL OBSTRUCTION

INVAGINATION.—*Anatomy—Obstruction and subsequent strangulation—Ætiology and pathology—Symptoms: pain, tumour, hæmorrhage—Duration of first and second stages—Diagnosis—Treatment.*

IMPACTION.—*Gall-stones—Concretions—Fæcal masses in the rectum or colon.*

STRICTURE AND CONTRACTION.—*Cicatrices—Adhesions and contractions affecting the ileum—Simple and malignant stricture of colon and rectum.*

STRANGULATION.—*Bands—Apertures in omentum or mesentery, &c.—Internal hernia—Volvulus—its seats, mechanism, and effects.*

General results of obstruction and strangulation—Anatomy—General symptoms and diagnosis: constipation, pain, vomiting—Special symptoms of chronic obstruction—diagnosis of cause—treatment—colotomy—Special symptoms of acute obstruction—diagnosis of cause—prognosis—treatment: expectant—by abdominal section—by other mechanical methods.

WE now pass to the consideration of a group of affections which differ widely from one another in their anatomical characters, but which agree in this, that they tend to cause mechanical obstruction of the contents of the bowels.

INVAGINATION.*—Of these affections there is no one more interesting, or more important, than that which is termed intussusception or invagination: one part of the bowel passes into another part, just as the finger of a glove can be made to slip into itself. The direction in which this occurs is invariably downwards (or "forwards"); *i. e.* the sheath, *la gaîne*, the portion of the gut (*recipiens*) which receives the other portion (*receptum*) is always on its anal side. Out of 500 fatal cases of intestinal obstruction collected by Brinton, 215 were due to invagination.

Anatomy.—A moment's consideration will show that an intussusception must consist of three parts, or, as they are often called, "layers." Of these we may term the outermost the "receiving," the middle the "returning," and the innermost the "entering" layer. The two together are called *le boudin* by French writers. The returning layer, unlike the others, has its mucous outside its serous coat; it is in fact turned inside out. The bend which connects the receiving and the returning layers is situated at the upper part and its convexity is formed by the peritoneal coat of the intestine; that which connects the returning and the entering layers is at the lowest point of the intussusception, and the mucous membrane covers it.

Probably almost every portion of the bowel is liable to intussusception. Sometimes one part of the small intestine enters another part, or one part of the colon another part. A case has occurred at Guy's Hospital in which the rectum with just the lower end of the sigmoid flexure passed down through the anus; there was some difficulty in distinguishing it from a mere prolapsus of the mucous membrane. There is, however, one point at which

* *Synonym.*—Intussusception.—*Germ.*—Einstülpung.

invagination occurs much oftener than anywhere else, namely, at the junction of the small with the large intestine. Such cases are generally distinguished as *ileo-cæcal* intussusceptions, and make up nearly half of Leichtenstern's 442 cases. It is often supposed that they arise by a gradual protrusion of the ileum through the ileo-cæcal valve into the cæcum. But in reality this rarely occurs; perhaps not more than a dozen instances have been recorded of "valvular" or *ileo-colic* intussusception. Next in frequency is invagination of the ileum, and next that of the colon.

An invagination at its commencement affects only a very small part of the bowel. Gradually more and more of this is involved, and always by the inclusion of one part after another of what had been the receiving layer. Thus the upper bend of the intussusception is constantly shifting, but the lower bend, on the contrary, remains stationary from beginning to end. In an ileo-cæcal case, for instance, the entering layer is the ileum; the cæcum usually constitutes both the returning and the receiving layers; the lower bend is situated exactly at the ileo-cæcal valve. As the affection advances, the whole of the cæcum, the ascending, transverse, and descending colon may become included; but the ileo-cæcal valve always remains at the lowest point of the mass. Extraordinary as it appears, an invagination of this kind may pass through the anus, and even project for some inches; and the valve may actually be seen and felt in this position, as well as the orifice leading into the vermiform appendix. The peritoneal layers fixing the intestine, and the vessels which pass to it, must indeed be stretched to an extent which it is difficult to conceive; but the protrusion of an ileo-cæcal intussusception from the rectum has repeatedly been observed, and may be regarded as the natural course of the affection. On the other hand, when the small intestine is invaginated, the mesentery tethers it much more closely. It is drawn in, and forming a wedge-shaped mass on one side of the gut, pulls on it and drags its lower end to one side, so as to make it assume a contorted form.

The next step is that the circulation of blood in the invaginated mass is interfered with. Sometimes, it seems, the veins alone are compressed; blood can then no longer return from the affected part of the bowel, which becomes enormously swollen, with hæmorrhage and exudation of serum into its tissues. This is particularly apt to occur in ileo-cæcal intussusceptions; but it is often delayed until an advanced period of the case. The swelling is always more marked at the lower bend than anywhere else. Dr Moxon mentions an instance in which the coats of the bowel were three quarters of an inch thick in this position. The included part of the mesentery likewise becomes dark and thickened by effused blood.

Thus to *obstruction* is now added *strangulation* of the intestine. The one condition prevents the passage of fæces and acts in a way comparable to stricture of the urethra, stricture of the œsophagus or stenosis of the pylorus. The other condition interferes with the nutrition of the affected tissues, and causes congestion, inflammation, and at last gangrene. Both obstruction and strangulation exist in an ordinary case of hernia when left to itself, and we shall have to observe their effects in all forms of ileus. Obstruction alone is caused by impacted fæces or calculi. Strangulation alone is caused by a part of a coil of intestine, or a piece of omentum, being caught in a tight band. Mr Bryant has particularly insisted on this important distinction.

In other cases the influx of blood through the arteries appears to be

arrested as well as its escape by the veins. This takes place especially in intussusception of the small intestine, in consequence of the comparatively narrow diameter of the receiving layer in that form of the disease.

The inevitable result is gangrene of the invaginated mass. And strange as it may appear, this does not always lead to the death of the patient. The sloughing part of the bowel may be cast off, and may pass down the large intestine, and be discharged per rectum. It generally appears as a single tube, with its mucous surface outwards; this perhaps includes both the entering and the returning layers, the former having undergone inversion during the process of detachment; or it may be that the entering layer is cast off separately in the form of soft shreds, so that the inverted mass is constituted by what had been the returning layer only. Some years ago a specimen of this kind was sent up to Guy's Hospital by Mr Higginbotham, of Bruton; it consisted of twelve inches of intestine, and within its channel lay the appendix vermiformis, which opened on to its outer or mucous surface. Only an inch of it was small intestine. Consequently, if the intussusception was of the ordinary ileo-cæcal variety, the cast-off mass must have been derived mainly from the returning layer, the entering layer having doubtless broken down and been discharged separately. Several instances have been recorded in which portions of bowel from twenty to forty inches long have been shed; and in one extraordinary case related by Carswell eight distinct pieces, amounting altogether to fully twelve inches in length, were passed through the anus at considerable intervals of time. In different cases there are great variations in the extent of the inflammation that occurs at the line of separation, which of course corresponds with what was the upper bend of the intussusception. In a case which Dr Hare brought before the Pathological Society in 1862, and in which the patient died of phthisis three months after passing some inches of bowel, the line of union could only just be detected on the mucous surface by its shining glazed appearance. Even on the serous surface there was only what is described as "considerable puckering," but below the cicatrix a small pouch existed into which projected a curious little hollow cylinder, evidently a relic of the invaginated part. In other instances, however, inflammatory products have been formed in large quantity at the line of separation; and thus the new channel has been reduced to a very small calibre. Or suppuration has occurred at the same spot, and an abscess has developed itself containing pus alone, or pus mixed with fæcal matter. Cases of this kind probably always terminate fatally. And even when the expulsion of the cast-off bowel is followed by the recovery of the patient there is always a risk that the cicatrix may gradually contract. Dr Moxon has twice seen a fatal annular stricture of the ileum, with puckering of the mesentery, which he believed to have arisen from a former intussusception.

Age and sex.—Intussusception may occur at any age. In adults, however, it is far more rare than might be supposed from the comparatively numerous cases that Hutchinson, Peacock, and others have collected from different medical works. Dr Wilks used to say that he had only seen one case in a grown-up person. On the other hand, in young children and infants the disease is very frequent; and probably very many cases are overlooked, so that it really occurs oftener than would appear from the published statistics. In fact, with rare exceptions, intestinal obstruction in a child is due to invagination.

Among children, males are far more liable to intussusception than females.

Rilliet and Barthez met with twenty-two cases in boys to only three cases in girls. In Leichtenstern's collected cases of all ages, 285 occurred in males and 157 in females. In adults the difference between the sexes in this respect is far less marked. Thus in one year, the Registrar-General's returns (according to Mr Gay) gave altogether 163 male to 93 female patients, while between thirty-five and forty-five the numbers were 55 men to 74 women. Mr Treves has shown that the acute cases belong chiefly to childhood, while the chronic ones are more common in adult life.

Exciting causes.—There is reason to believe that sudden movements of the body may cause intussusception. Rilliet and Barthez mention two instances, in each of which it came on suddenly in a child who was being jumped in its father's arms. Violent muscular efforts to raise a burden have also been assigned as causes of it; and likewise direct injuries.

It probably suffices for the production of an intussusception that the contraction of the transverse muscular fibres of one portion of the bowel should lengthen it, and push it into the interior of the succeeding portion.

Everyone who is accustomed to make *post-mortem* examinations knows that in children, particularly after death from cerebral disease, it is common to find two or three, or even several, short intussusceptions at different parts of the small intestine. They are sometimes reversed or retrograde. The affected parts are not reddened in such cases, and there have been no intestinal symptoms; hence some writers have supposed that the invaginations have arisen after death as a result of rigor mortis. But it appears more probable that they really occurred towards the end of life, and that they would have undergone spontaneous reduction. The same thing is constantly to be seen in opening the abdomen of rabbits and other animals.

One distinction between such intussusceptions and those which give rise to symptoms is that the former do not occur at the junction of the ileum with the cæcum, which we have seen to be the favourite seat of the latter. The special liability of this part of the bowel to the disease is probably the combined result of two different circumstances: one, that the axis of the large bowel is nearly at right angles with that of the small intestine; the other, that the cæcum is much more fixed as well as much larger than the ileum. In two recorded cases, invagination of the ileum appeared to have been favoured by the presence of cancerous induration of the cæcum; this may be supposed to have kept it more widely open than in its normal condition. On the other hand, many instances have been recorded in which the starting-point of an intussusception has been a polypus hanging from the mucous membrane. This seems to have been caught by the contraction of the intestine below, and to have dragged downward in its turn the part of the bowel to which it was attached. Dr Moxon met with a case in which an intussusception appeared to have been caused in a similar way by a diverticulum of the ileum; this must itself have been first inverted into the gut. The presence of the *Ascaris lumbricoides* in cases of intussusception has been noticed by several observers, and it seems not impossible that a worm might have its body grasped by the peristaltic movements of the intestine, so as to invert the part to which its head was attached.

Symptoms.—As a rule, the effects of invagination of the bowel are very different from those of obstruction from other causes. In the progress of many cases two distinct periods can be recognised, each attended with its own symptoms.

In the *first period* the intestinal contents pass freely through the intussuscepted part. The bowels may act every day and the evacuations are natural. The chief complaint is of a paroxysmal *pain* in the abdomen, the first sudden onset of which forms in fact the starting-point of the case. This pain is generally referred to the neighbourhood of the umbilicus. It is often extremely violent, so that the patient rolls about the bed or the floor of the room in agony. It is sometimes accompanied by vomiting, but rigors seldom occur. It usually passes off and may return only after a considerable interval In a case that came under the author's observation, the patient, a boy five years old, had for four months only two or three attacks of pain during each twenty-four hours ; between them he appeared perfectly well.

There is another symptom of intussusception which can almost always be detected by manipulation of the abdomen—the presence of a *tumour*. This is in fact a most important feature of the disease, even from its very commencement. To detect it one may sometimes have to place the patient under chloroform so as to relax the abdominal walls, and if there be a great quantity of fat in them and in the mesentery, the examination may fail to yield a definite result. But with these limitations, doubt as to the existence of an intussusception may generally be cleared up by thorough palpation of the abdomen. The tumour which occurs in this disease is felt as a sausage-like mass of greater or less length. The note yielded by percussion over its surface may be dull, or partially resonant, or scarcely distinguishable from that given by other parts of the abdomen. Its seat varies with the part of the bowel which is concerned. In the common ileo-cæcal variety it originally occupies the right iliac fossa. As more and more of the intestine becomes involved, the tumour gradually changes its position. It moves across the abdomen, either at the level of the umbilicus or a little higher ; having reached the left side it passes downwards into the left iliac fossa, and ultimately into the pelvis. Brinton says that it often forms an elongated mass, which lies horizontally just above the pubes. Another peculiarity about this kind of tumour is that its size and form are in many cases liable to frequent changes. It may be hardly perceptible when one first places one's hand upon the abdomen, and may harden and become prominent under manipulation, particularly if a paroxysm of pain should come on. In one case so positive a diagnosis was founded upon the presence of these two symptoms only—pain and tumour—that Mr Howse was asked to open the patient's abdomen and to search for and pull out the intussusception, with the result that the patient made a good recovery. The case is recorded in the 'Medico-Chirurgical Transactions' for 1876.

As the disease advances, exploration of the rectum affords further aid in diagnosis. The end of the invaginated intestine can often be felt with the finger, and ultimately it may even protrude from the anus. Before this occurs, however, fresh symptoms commonly develop themselves, belonging to the *second stage* of the disease. As already remarked, the veins of the intussuscepted part of the bowel become obstructed, and it consequently becomes intensely congested and swollen. Another result of this change is that *hæmorrhage* occurs from its surface, and that blood is discharged by stool, or a mixture of mucus and blood. Some *tenesmus* may also occur if the affection extends into the lower part of the large bowel. Or blood may be vomited, in the rare event of the small intestine near the stomach being the seat of the disease. At the same time or somewhat later, the passage of fæcal matter through the invaginated part is obstructed. *Sickness* sets in,

there are no longer any remissions in the pain, the vomited matters become stercoraceous, the abdomen now becomes distended, the patient is collapsed, and before long he expires. In the case already referred to of a boy who for four months had no symptoms but paroxysmal pain and tumour, death occurred within three or four days from the time when he first began to have tenesmus and to pass blood and mucus. And in infants such symptoms generally set in at the very commencement of the disease, which commonly proves fatal by the second or third day.

It has generally been supposed that the cases attended with these symptoms are those in which the invaginated mass is apt to slough away and to be discharged per anum, but Mr Hutchinson showed that in chronic cases of intussusception this very rarely occurs. Moreover, the changes which lead to the gangrene and shedding of the whole of the invaginated part of the bowel involve the complete arrest of the circulation of blood in it. Consequently, one would not expect hæmorrhage from the bowels to occur while these changes are in progress, although it is true that the ulceration at the neck of the intussusception may lead to hæmorrhage at the time when the sloughing part is being detached. Out of twenty cases collected by Dr Peacock, in all of which the invaginated parts were shed and passed per anum, there was only one in which bleeding is said to have occurred, and in that one it ceased twelve or fourteen days before the expulsion of the gangrenous mass. In fact the symptoms of those cases in which the affected part of the bowel sloughs can seldom be clearly divided into two stages; and very often they are undistinguishable from those of other forms of intestinal obstruction, until the portion of gangrenous intestine is unexpectedly voided from the rectum.

Among those cases which present the more characteristic symptoms of intussusception the duration of the first period varies greatly. The above-cited case lasted four months, and similar instances have been recorded by others. A patient of Dr Brinton's died of cancer of lungs during this stage of an intussusception, which had lasted four months and a half, and it remained uncertain whether the abdominal affection had any share in determining the time of the man's death. Such protracted cases appear always to be examples of the ileo-cæcal variety of the affection. On the other hand, many cases, even of this variety, pass into the second stage from their very commencement; and when the small intestine is alone concerned, the disease appears generally to take this course. Sometimes hæmorrhage and the other symptoms belonging to the second period set in and afterwards subside. Mr Sydney Jones met with a case in which they lasted for three days and then passed off, returning again seventeen days later and then leading to a fatal termination.

Brinton says that when the invaginated mass sloughs off and is discharged, the date at which it commonly separates is the eighth day, in cases limited to the small intestine; and that it is expelled on the tenth day. In cases belonging to the ileo-cæcal variety the corresponding dates are, according to him, the fifteenth and twenty-second days respectively. But these last figures are of little value, because of the variable duration of the first stage in ileo-cæcal intussusceptions.

Diagnosis.—This may either be perfectly easy, or exceedingly difficult. It should be laid down as a rule that the abdomen is always to be carefully explored, by the hand laid upon its surface, whenever a patient (particularly a child) complains of paroxysmal pain there, recurring without obvious

cause. A tumour may perhaps be discovered, the form of which, and its seat, would be consistent with its being an intussusception. The alternative diagnosis is generally that the tumour is an impacted mass of fæces. An enema (repeated, if necessary) generally solves all doubt upon this score; or the suspicion may be confirmed by finding the tumour harden under manipulation, or by detecting peristaltic movements in it. In one instance a rounded swelling, of somewhat cylindrical form, and only partially dull on percussion, was felt crossing the abdomen just above the umbilicus, and was at first thought to be due to intussusception. But the fixity and unchanged position of the tumour, and the absence of other symptoms, soon showed that it was not. The man died some months afterwards, and the case proved to be one of tubercular peritonitis, the mass that had been felt being the omentum indurated with caseous tubercular matter.

In those cases which are attended at an early stage with hæmorrhage from the bowels, there is danger of mistaking the disease for dysentery. All doubt may then sometimes be removed by digital examination of the rectum. But even when there is protrusion of the invaginated mass from the anus, it has sometimes been taken for a mere prolapsus. This occurred in a case which has been recorded by Mr Hutchinson; a practitioner, called in to see the patient, returned the bowel into the rectum, and fitted a cork pad to the anus to prevent its coming down again.

Treatment.—Our object in a case of intussusception is to replace the bowel in its natural position. This may be effected by several different methods. In at least two cases introduction of a gum-elastic bougie into the rectum has succeeded, but it is applicable only when the invaginated mass lies in the rectum, and probably only when the part of the intestine concerned is limited to the lower part of the colon.

Copious injections of warm water have been known to cure the disease. But a much more effectual measure than this appears to be inflation of the intestine with air. This procedure was many years ago (1838) recommended by Mr Gorham in the 'Guy's Hospital Reports' (1st series, vol. iii, p. 345); and he quoted three cases which had been successfully treated in this way in America. It has now been frequently employed, and sometimes with the result of completely curing the disease. More often, perhaps, its success has been partial. The tumour has been reduced in size; or it has changed its position, returning towards the seat which it had occupied at an earlier period; or it has been made to disappear for a time, and all the other symptoms have subsided, but only to recur a few days later, with a fatal termination.

The method of injecting air into the bowel is very simple. An ordinary pair of bellows is employed. Round the pipe, at a little distance from the nozzle, a plug of lint is firmly fixed with adhesive plaster. The pipe is then introduced into the rectum, the plug being kept firmly pressed against the anus. Air is then forced in until the abdomen becomes tense, or until the patient can no longer bear the pain. Sometimes it has appeared to be advantageous to place the patient head downwards while either air or water is being injected into the bowel; and the inhalation of chloroform is recommended by many authorities. Care must be taken that too much force is not used, for in the case of an infant, aged five months, which was treated by inflation in Guy's Hospital in 1873, the bowel gave way and a large quantity of air passed into the peritoneal cavity. Some of it was let out through a puncture in the abdominal walls, but the child did not rally and died in a few hours. In

another instance the serous covering in the bowel was cracked in several places and the muscular fibres torn; in that case both air and water were injected.

It is easy to see that air, as a highly compressible fluid, is far more likely to effect the reduction of an invaginated mass than an incompressible fluid like water; and inflation should be practised in every case of intussusception in which the diagnosis is made at a sufficiently early period; but it is evident that if the process of sloughing of the included bowel has once set in, the procedure would not only be useless but would take away the last chance of the patient's recovery. Unfortunately, there are no definite symptoms which indicate that the invaginated mass is passing into a state of gangrene. Still, as Mr Hutchinson has remarked, the fact that in a particular case the tumour is advancing lower and lower in the large intestine is always proof that this is not the case, and that the upper bend is not yet fixed.

If it should be decided that such methods of treatment are inapplicable, and indeed in every case at first, the patient must be kept well under the influence of opium. Ice may be given him to suck in small quantities, but he should be allowed to drink as little food as possible. The treatment in fact should be the same as in a case of acute peritonitis or of ordinary intestinal obstruction.

Inflation, however, may fail, even if performed early, and repeated two or three times at intervals of a few hours. When this has occurred, the operation for opening the abdomen should as a rule be performed without further delay. As far back as the year 1784 this was done successfully in Paris, in the case of a woman aged fifty. In 1873 Mr Hutchinson advocated it in a paper read before the Royal Medical and Chirurgical Society. He had in 1871 performed the operation on a child, aged two years, who had an ileo-cæcal intussusception for a month, which was protruding from the anus. He opened the abdomen in the median line, introduced two or three fingers, and quickly drew out the invaginated mass at the wound. He then easily effected its reduction and returned it into the abdominal cavity. This was all done in two or three minutes, and the child recovered well.

Mr Hutchinson remarks that the cases most suitable for this operation are those ileo-cæcal intussusceptions, in which the symptoms come on slowly. In such cases there is but little tendency to sloughing and detachment of the invaginated part, so that on the one hand there is scarcely any prospect of a spontaneous cure, and on the other hand the surgeon is very unlikely to find the bowel in such a state as would render it impossible for him to proceed with the operation. But the included portion of intestine is often highly congested, and this may seriously interfere with the complete reduction of the invagination. Dr Goodhart made an autopsy in one case, on a child, aged six months, who had died in less than twenty-four hours. and in that instance he could neither by traction nor by squeezing succeed in replacing the whole of the cæcum; the end of it still remained inverted.

The occurrence of hæmorrhage, with or without tenesmus, is at least suggestive of the probability that the included part of the bowel is in a highly congested if not sloughing condition. It is therefore a matter of importance that the disease should if possible be diagnosed before this symptom shows itself. In 1874 Dr Adcock, of Bermondsey, asked the author to see a woman who had suffered for a fortnight from paroxysmal attacks of pain in the abdomen. A tumour was readily detected which

presented all the characters of an intussusception. She had but little sickness, and no constipation. Inflation with air was at once practised, and the swelling then receded from the left iliac fossa towards the right side of the abdomen, which had been its original seat. This clenched the diagnosis. We therefore asked Mr Howse to see the patient. He took the same view of its nature, and after repeating the injection of air performed the operation of abdominal section. There was no difficulty whatever in reducing the invagination, and the patient recovered without a single bad symptom ('Med.-Chir. Trans.,' vol. lix).

Out of 33 cases collected by Peyrot and Treves in which laparotomy was performed for intussusception, there were 9 deaths and 24 recoveries. In 10 of these cases (with 7 successful) the reduction was easily accomplished after the abdomen was opened; in the remaining 23 (with only 2 successful) it was difficult or impossible.

We now enter upon a subject—mechanical obstruction of the bowels—of which the pathological aspect is exceedingly complicated. It will therefore be convenient to approach it from this side.

Of the various conditions that may occlude the gut, some have their seat in its interior, others in its coats, while a third set affect it from without. And each of these will have to be considered separately as they affect the small and the large intestine.

1. *Impaction*—the bowel being blocked by a mass occupying its lumen.

a. In the *small intestine* this scarcely ever occurs, except from the presence of a large gall-stone. It either escapes by gradual dilatation of the duct, or more frequently by ulceration into the colon or the duodenum. Forty-one cases are recorded by Leichtenstern, but they are, nevertheless, of rare occurrence. The seat of impaction is usually in the duodenum or upper jejunum, or else at the ileo-colic valve. This accident is most frequent in women, and after middle life (40–70). The symptoms are not very severe: vomiting is early; there is no distension, but considerable pain. Two cases were published by the editor in the 'Path. Trans.' for 1887, in one of which the calculus was felt (though not recognised) per rectum; the patient, a woman of seventy, passed it safely, as did the second patient. One stone weighed 270 grains, the other 323.

In idiots the ileum has sometimes been found obstructed by balls made up of fibrous materials that had been swallowed. Dr Langdon Down in 1866 showed to the Pathological Society a mass of interlaced cocoa-nut fibres the size of a hen's egg, which caused death by occluding this part of the intestine; the patient, a boy aged sixteen, had a habit of putting a shred of cocoa-nut fibre into his mouth, and playing with it between his teeth. A similar case occurred to the late Dr Mackenzie Bacon.

Obstruction of the smaller gut by concretions of magnesia is extremely rare. One example is preserved in the museum of St Thomas's Hospital.

It very rarely happens that ordinary articles of food, in passing along the healthy bowel, cause anything approaching to obstruction. An instance is related by Brinton, in which an abdominal tumour, the size of a pullet's egg, was believed to be formed of a mass of half-chewed filberts. Having been first detected in the right hypochondrium, it in two days moved almost entirely downwards into the left iliac fossa, and then disappeared; a few hours afterwards the bowels acted for the first time.

β. In the *large intestine* obstruction has occasionally been caused by concretions. Of these one variety (less rare than in the ileum) consists chiefly of magnesia. A case in point was brought before the Pathological Society in 1855 by Mr Hutchinson. It was that of a lady who had the rectum blocked by a rough hard body, at least fifteen inches in circumference, which had to be broken down before it could be removed. It was made up partly of strawberry and other seeds, partly of concentric layers of what looked like a red stone, but was found to be a mixture of iron and magnesia. The patient had formerly taken sesquioxide of iron and also carbonate of magnesia in large doses, but not for twelve years before the detection of the mass in her rectum.

In persons who eat largely of oatmeal another kind of concretion is sometimes met with, consisting of a felted mass of hairs derived from the grain. Such masses have a soft, velvety feel. Several specimens of them are preserved in the museum of the College of Surgeons; their nature was first suspected by Mr Clift, and afterwards demonstrated by Dr Wollaston. As might be expected, they have been found in Scotland more often than elsewhere. They have not generally caused insuperable obstruction of the bowels, but have been passed after giving rise to more or less distress. In one case, that of a man named Gordon, quoted by Sir Thomas Watson, no less than thirty-two such concretions, varying from a hen's egg to a filbert in size, were voided at different times.

Fæcal matter in the large intestine sometimes accumulates to such an extent as to cause symptoms of obstruction of the bowels. A remarkable case of this kind occurred to Dr Peacock, and was recorded by him in the 'Pathological Transactions' for 1872. A man aged twenty-eight died in St Thomas's Hospital after an illness of six weeks, during which he had had obstinate constipation. The bowel, from the cæcum to the rectum, was found loaded with semi-solid greenish fæces, to the amount of fifteen quarts; it measured from six to eight inches in circumference. The patient had been subject to constipation from childhood, and for twelve years before his death his bowels had never acted without an enema, aperients having ceased to produce any effect. Another case which seemed to be of this kind was remarkable, because the patient, a woman aged twenty-two, was attacked with abdominal pain and other symptoms of obstruction twenty-four days before her death, and yet at the autopsy no cause could be discovered for her illness, unless it were the presence of rounded hard fæces in considerable quantity in the sigmoid flexure and rectum. This, however, was not all that had accumulated, for much had been removed during life by a large enema.

2. *Narrowed calibre.*—Obstruction may be caused by changes in the coats of the bowel itself, causing *stricture*, or otherwise narrowing its lumen.

α. *Stricture and constriction.*—In the small intestine one scarcely ever meets with anything comparable to a stricture of the urethra, or of the œsophagus. In one instance, after an *operation for strangulated hernia*, the patient suffered from continual vomiting, and died in two months; about an inch and a half of the small intestine was found to be narrowed, its coats thickened by hard, white cicatricial tissue, and its mucous membrane almost devoid of villi; evidently this was the part which had been in the hernial sac. Dr Moxon saw two cases in which narrowing of the small bowel was believed to have resulted from the sloughing of an *intussusception* (*supra*, p. 409), and similar instances have occurred to other observers. In

the 'Pathological Transactions' for 1869 a remarkable case is related by Dr Wickham Legg, in which the opening from the ileum into the cæcum was only just large enough to admit a No. 9 catheter; and this writer refers to similar instances recorded. He supposed that in his case the stricture was *congenital*. May it have resulted from the shedding of an intussusception? The patient, a woman aged thirty-two, had nearly all her life been liable to attacks of what was termed colic, and six years before her death she was in University College Hospital under Dr Walshe. It was then noticed that manipulation of the abdomen produced a peculiar crackling sensation, which could be both felt and heard. That this was due to the presence of cherry stones in the interior of the intestine was tolerably evident, for on one occasion she passed some. After death the intestine was found to contain almost enough fruit stones to fill a pint measure; most of them lay in the jejunum or ileum at a distance from the strictured valve, but a few in a dilated pouch measuring seven inches in circumference, which was situated immediately above it.

Most writers state that the cicatrisation of tuberculous ulcers is a pretty frequent cause of stricture of the small intestine; and, by way of contrast, that such a condition never follows the healing of the ulcers which occur in cases of enteric fever. But no case of either kind is recorded in the 'Pathological Transactions,' and none has been met with at Guy's Hospital within the last twenty-five years. The nearest approach to it is a case which occurred at the hospital in 1858. A child died of phthisis after an illness of three or four months' duration, of which diarrhœa had been a principal symptom. There were numerous large tuberculous ulcers in the bowel, some of them extending all round it. A remarkable degree of narrowing, apparently from contraction of the peritoneal coat of the intestine, was found at several parts, and through them the blade of an enterotome could hardly be passed. In this instance, however, the symptoms of intestinal obstruction seem to have been altogether absent.*

But if the small intestine is thus scarcely liable to true stricture, it is very apt to be affected with another condition, which I endeavoured, in the 'Guy's Hospital Reports,' ser. 3, vol. xiv, to distinguish by the name of "*contraction*."† In this the morbid appearances are of a much less striking kind than in other forms of obstruction of the bowels, and they are but little susceptible of illustration by drawings, or of preservation in pathological museums. Consequently I believe that it had remained almost unrecognised before the publication of my paper, although it is not really by any means of infrequent occurrence. The cases which I recorded affecting the small intestine were twelve in number. In four of them the affection was the consequence of chronic peritonitis, by which more or less of the small intestine was bound down to some part of the abdominal wall, or by which its coils were made to adhere among themselves. In two it resulted from the puckering caused by cancer affecting the serous covering of the bowel, and from adhesions which had formed. In one instance, a somewhat similar condition of the intestine arose as a result of tubercular peritonitis; and in

* Mr Treves figures one case of double stricture of the jejunum from tubercular ulcers, and quotes a unique case, reported by Klob, of stenosis after typhoid ulceration. He remarks that stricture in the small bowel is often double or multiple. Of 26 cases collected by him from various sources 10 were cancerous, and the rest were cicatricial; 10 after ulceration, 4 after hernia, and 2 after injury.

† Dr Bristowe calls it "compression and traction." It includes cases otherwise described as obstruction by adhesions, causing traction, compression, shrinking or bending of the gut.

three it was due to contraction associated with chronic disease of glands in the mesentery. The remaining two cases were of a less definite nature. The great peculiarity in this form of illness is that, instead of there being one particular spot beyond which the contents of the bowel cannot pass, the impediment is generally continued through a greater or less length of it. The whole of the small intestine may be matted up, so that one is unable to say that one spot more than another was the seat of the obstruction. Or there may be one or more sharp bends or twists or "kinks;" and sometimes it is clear how the part of the bowel above a bend, becoming distended, pressed on that below, and occluded it. Or, one portion of the intestine being fixed by adhesions, that above it may be stretched by accumulated fæcal matter, and hang down into the pelvis, so as to drag on the attached portion, and prevent anything passing through it. Two striking instances of the latter kind have occurred in Guy's Hospital since the publication of my paper. One of the former kind came under the observation of Dr Bristowe and has been recorded by him in the 'Pathological Transactions' (vol. xxi, p. 185). In it, the intestinal coils from the middle of the ileum to within a foot of the cæcum, were adherent to one another and to the brim of the pelvis by bands and filaments of false membrane, and were so entangled that their direction was traceable with difficulty; but there was no part of the bowel through which the finger failed to pass.

The origin of the adhesions and contractions is often the presence of an old hernial sac, or the injury inflicted by a strangulation which was relieved by herniotomy. Often it is pelvic cellulitis, with adhesions of the gut to the ovary, uterus, or broad ligament; often former suppuration of mesenteric glands, past typhlitis, and chronic tubercular or cancerous peritonitis. As Mr Treves remarks, the fact that femoral hernia, pelvic peritonitis, and malignant peritonitis are more common in women than in men explains the greater frequency of female cases.

β. In the large intestine, obstruction sometimes arises from its being bound down or adherent to the adjacent structures; in other words, there is an affection analogous to "contraction" of the small intestine. I related three such cases in my paper; in one each end of the loop formed by the sigmoid flexure was bound down to the spine by firm fibrous tissue; in another the impediment resulted from adhesion of the transverse colon to the neck of an umbilical hernia; and in a third its cause was that the same part of the bowel had been dragged down and fixed to the mesentery over the lumbar vertebræ.

But below the ileo-cæcal valve, an affection of this kind is exceedingly rare in comparison with *true stricture*. This last is, indeed, the commonest of all the lesions that give rise to intestinal obstruction, being present (according to observations made at Guy's Hospital) in seventeen out of fifty-one cases, or as nearly as possible in 34 per cent. Its seat is, in the majority of cases, in the rectum; next in the sigmoid flexure. Out of 100 collected cases from Guy's Hospital, and from the lists of Mr Treves and of Dr Coupland and Mr Morris, 58 have been in the sigmoid flexure; while, of the remainder, 11 were in the descending colon, 8 in the splenic flexure, 7 in the transverse colon, 10 in the hepatic flexure, 2 in the ascending colon, and 4 in the cæcum. Thus it may almost be said that the liability of the large intestine to stricture increases regularly from its upper to its lower end. It must be borne in mind, too, that a large number of cases of disease of the rectum, of precisely the same pathological characters, are excluded

from these figures because in them obstruction of the bowels was not present during life.

The affections which cause stricture of the large intestine vary greatly in their nature in different cases. Occasionally the disease consists of a simple overgrowth of connective tissue, contracting the bowel and puckering up its muscular coats. More often it can be traced to the cicatrization of a *dysenteric* ulcer. Frequently the stricture (particularly in women) is *syphilitic* in origin, usually rectal in position, and annular in shape. But the most common form of stricture of all is some form of *malignant* growth. This often has more or less distinctly a villous character. In some cases it forms a raised ring of a bright crimson colour and projecting with a smooth velvety surface into the cavity of the bowel. In other cases it is excavated by ulceration.

Histologically it may occasionally be an ordinary glandiform carcinoma, but it is far more frequently a cylinder epithelioma. Indeed, Mr Harrison Cripps finds all reported cases of scirrhus or encephaloid cancer of the rectum to be on examination cylindroma; and so M. Hausmann (1882) and Mr Treves (1884). Not infrequently the growth is found to have undergone extensive colloid degeneration. Secondary growths in the lymph-glands or in other viscera are often absent; the reason being, without doubt (as in the cases of cancer of the stomach and of the uterus), that the death of the patient occurs at no distant period from the commencement of the disease.

3. *Strangulation.*—The remaining forms of intestinal obstruction differ from those hitherto considered in the circumstance that the constricting agent has no structural connection with the coats of the affected part of the bowel, and is invested with a distinct peritoneal covering.

a. In the small intestine the cases which come under this head are chiefly such as would be described as examples of *internal strangulation*, in the narrower sense of that term. These form a considerable proportion among all cases of intestinal obstruction. Thus, in Guy's Hospital, between the years 1854 and 1869, there were thirteen of them among fifty-one cases of all kinds. The exact nature of the constricting agent varies widely.

In many it is a *fibrous band* of greater or less length, attached at each end, but free in the rest of its course. This was described by Mr Gay as the "solitary band," on account of there being only a single one present in each case, or at most two or three. Such a band may be attached to any part of the mesentery or intestine, or may pass from the mesentery or intestine to the neck of an old hernial sac, or to the uterus, ovary, or Fallopian tube. Or it may consist of a process derived from the omentum, and pass to any of the structures that have been mentioned. Its mode of origin is not always to be ascertained, but some observations have been made which show that it may be the direct result of the process by which injury of the intestine is repaired. Mr Gay records a case in which the transverse colon was punctured by a trocar in the operation of paracentesis abdominis. The patient recovered, but died many months afterwards of fever. A firm and thick band, two inches long, was then found, passing from the seat of the wound to the parietal peritoneum. And this writer quotes a case of Jobert de Lamballe's, in which a man who had been stabbed in the abdomen by a stiletto died some time afterwards from stangulation of the intestine by a band, which extended from the abdominal wall to the spot in the bowel that had been injured.

But in many cases the constricting agent is not a mere fibrous band, but a cord connected with a *diverticulum ilei*. The latter, indeed, is the cause of fatal obstruction of the bowels in a proportion of cases which is remarkable, if we consider how seldom it is found in those who die from other causes. Among fifteen cases of internal strangulation at Guy's Hospital five resulted from the presence of a diverticulum. This is always situated on the side of the bowel furthest from the mesentery, and near the lower end of the ileum. According to Wilkinson King it is never more than from ten to twenty inches above the cæcum; but in the museum of Guy's Hospital we have a specimen which was fifty-four inches from the valve. As Meckel long ago showed, this form of diverticulum is a relic of a fœtal structure, the omphalo-mesenteric duct, which passes in the embryo from the umbilical vesicle to the ileum. Before birth it ought to waste away entirely; but its intestinal end may remain pervious and acquire adhesion to the mesentery or to some other part. It is remarkable that this abnormality scarcely ever occurs except in males. Out of ten cases, in which a diverticulum was found at Guy's Hospital, only one occurred in a female patient.

Strangulation by a cord attached to the end of the *vermiform appendix* appears to be relatively much less common. Duchaussoy states that females are more liable to it than males; but Leichtenstern's cases were 27 men to 13 women, while those of ileus from a diverticulum were 52 men to 14 women.

A portion of the small intestine may pass through an *aperture* in the mesentery or in the omentum, and then swell and become irreducible, exactly as it would under the femoral arch. Or it may be strangulated by the pedicle of an ovarian tumour, or by the edge of the mesentery of another coil of small intestine, which is hanging down into the pelvis.

Internal hernia is another cause of strangulation of the small intestine. A most remarkable and interesting form of this was first described by Treitz under the name of retroperitoneal hernia ('Guy's Hosp. Rep.,' 1871, p. 131); in it a pouch is formed at the back of the abdomen, a little to the left of the spine, passing backwards and downwards behind the curve formed by the inferior mesenteric artery and its left colic branch. A specimen of this kind, in which fatal strangulation occurred, was shown at one of the early meetings of the Pathological Society by Dr Peacock; and it is probable that another case was one in which Hilton performed an exploratory operation, and drew out a coil of intestine from an opening, apparently in the mesentery, just at the point where the jejunum became free from the spine. A different form of internal hernia was observed some years ago by Mr Cooper Forster; a knuckle of intestine became strangulated in a sac which lay close to the upper border of the obturator membrane, without passing through it.

It must also be borne in mind that in any case of what appears to be internal strangulation, the cause may be an external hernia, too small to be discovered by manipulation. Hilton once opened the abdomen during life, and found an obturator hernia, the presence of which could not even then be detected in the thigh; and in a case at Guy's Hospital, in which an exploratory operation was performed, a very small knuckle of intestine was engaged in one femoral ring. Several of us had previously examined the groins most carefully, but had failed to detect any hernia.

β. The large intestine scarcely ever becomes constricted by a band, or strangulated in any of the ways described in the foregoing paragraphs.

There are two cases recorded in which the sigmoid flexure was compressed by the mesentery of a coil of small intestine hanging into the pelvis, one in which the ascending colon is said to have been strangulated by a similar cause, one in which the same part of the bowel is stated to have been constricted by the vermiform appendix, and a fifth in which the cæcum was strangulated by a diverticulum.

4. *Volvulus.*—*a.* Not infrequently, when a knuckle of small intestine is strangulated by a band, or is "kinked" by adhesions, the condition is further complicated by the gut becoming twisted on its axis. Or, when a loop has passed through an aperture or into a hernial sac, it may twist round the long diameter of the loop. Either condition may be called volvulus. A primary "twist" of any kind is rare in the small intestine.

β. The more loosely attached parts of the large intestine—the cæcum and the sigmoid flexure—are most liable to primary volvulus. This usually consists in the twisting of the loop formed by the affected part of the bowel, so that each of its ends may be said to be strangulated by the other one being wound round it.* The ascending and descending parts of the loop are in fact screwed up into a narrow cord where they cross, their calibre is obstructed, and the circulation of blood in the walls of the bowel is arrested. In such cases the affected portion of the intestine is always enormously distended. Thus we have more than once found the cæcum filling nearly half the abdomen, and reaching up into the left hypochondrium; while in another instance the sigmoid flexure extended upwards in a similar way, so as to come into contact with the diaphragm, and push it and the thoracic viscera upwards. When an attempt is made to untwist the coil, it may spring back into its abnormal position with considerable force. There is some difficulty in understanding how such an affection is brought about. Both the cæcum and the sigmoid flexure are not uncommonly found floating freely and considerably enlarged in persons who have passed middle age, but who may have suffered from no marked abdominal symptoms, except perhaps constipation. Probably such a condition is a necessary antecedent to the formation of a volvulus. But when it has been the cause of death, the loop is always found full of fluid and intensely inflamed. Much of the contents are doubtless the product of secretion from its mucous surface, and this must evidently have been poured out at an early period of the case, and before the arrest of the circulation through the vessels of the affected part. Dr Bristowe has recently suggested that enteritis is in fact the primary condition, and that the twisting occurs secondarily. He supposes that the portion of intestine becomes first inflamed and paralysed, and that, being heavy with accumulated contents, it is then pushed aside by the pressure of the active parts around it. But this explanation is insufficient to explain the way in which the neck of the volvulus is screwed up; and it is disproved by the exact limitation of the inflammation to the part of the bowel which is twisted. Volvulus is most frequent in adult males.

A typical case occurred at Guy's Hospital in 1886. A young man, aged twenty-three, was admitted with a history of previous attacks of the same kind, and with recent symptoms of acute obstruction. The pain was intense, the vomiting severe, and it soon became stercoraceous; the distension was enormous. The abdomen was opened on the fifth day, and the distended colon was reached, but the source of the obstruction could neither

* Occasionally a loose cæcum or sigmoid is found twisted round a coil of small intestine.

be reached nor redressed. After death the sigmoid flexure almost filled the abdomen, twisted on itself, and turned up, so that the returning end of the loop touched the liver. Even when the other viscera had been removed, it was found impossible to return it into its natural position.

5. *Compression from outside.*—This may be by a solid tumour, an abscess, an aneurysm or a cyst. It naturally is a rare cause of obstruction in the looser parts of the bowel, and is by far most common in the rectum, confined within the true pelvis. The symptoms are those of chronic obstruction; they are ingravescent and often intermittent. In two cases under the editor's care, the compressing tumour was a pregnant retroverted uterus, which was redressed, with removal of the obstruction; in another, it was an ovarian cyst which had fallen into the pelvis, and was tapped per rectum with relief to the symptoms.

General anatomy of the obstructed gut.—Whatever the cause of obstruction may be, the bowel below the seat of disease is pale, empty, and contracted, that above it is distended, often in the most extreme degree. The jejunum or ileum may become dilated until it is at least as big as the healthy colon; while the colon may reach a size which can only be described as enormous. Dr Moxon met with a case in which, after removal with its contents, the large intestine weighed nearly eight pounds; and some fæcal matter had previously escaped. One effect of the distension is that in the small intestine the mucous membrane becomes forced out between the layers of the mesentery, forming rounded pouches (as in two specimens shown to the Pathological Society in 1875), for which the author proposed the name of "distension-diverticula." In the small intestine the dilatation of the gut diminishes more or less rapidly as one passes upwards above the seat of disease; but the large intestine may be distended pretty uniformly in its whole length; or there may be a great accumulation of fæcal matter in the cæcum, even when the obstruction is situated far below the arch of the colon, this being evidently the result of reversed peristaltic movements on the part of the bowel, by which its contents are forced backwards upon the ileo-cæcal valve.

In other respects the state of the bowel above the obstruction differs in different forms of the disease. In those in which the course is chronic, the muscular coat becomes greatly hypertrophied, forming a translucent grey layer which gradually increases in thickness towards the affected spot. In the acute forms this is wanting, but all the coats may be swollen and injected, so that the bowel feels unnaturally thick and massive. The mucous membrane is very apt to be *ulcerated*, especially in chronic cases; so that nearly the whole lining of the colon is sometimes found to be destroyed. Perforation is a not uncommon consequence of this; and we have seen the greater part of the large intestine to be in a sloughing state, so that its contents were escaping from all parts of it. Peritonitis necessarily results from such a condition as this, unless the patient should die before there is time for it to be developed. But inflammation of the serous covering of the bowel is also apt to occur at an early stage in the more acute forms of the disease, quite independently of rupture, and even of ulceration.

General symptoms and diagnosis.—In their clinical history, cases of intestinal obstruction divide themselves into two groups, in which the symptoms are to a great extent different, and of which the treatment has to be regulated

by considerations which are distinct for each group. These are respectively *acute* and *chronic* in their course. The most convenient plan will be to take in succession all the points which concern one of these groups before entering upon those which belong to the other. But first there are three symptoms common to both, which require a brief notice, namely, constipation, pain, and vomiting which after a time becomes fæcal.

Constipation may be said to be the fundamental symptom of obstruction of the bowels. It is generally absolute and immoveable, whether by purgatives or injections, so long as the disease remains unrelieved.

A first enema may indeed bring away fæces which had lain in the part of the bowel below the seat of the disease, but subsequent ones almost invariably return uncoloured; and even below the obstruction the bowel is paralysed as a rule. In most cases, not even flatus can be passed per anum. In this, as in many respects, invagination is peculiar, for constipation, as we saw, is not always the first or even a constant symptom of that condition.

It must, however, be remembered that liquid fæces may pass through a stricture which is capable of causing great distension, ulceration, and perforation of the part above it. In a case in which we had diagnosed annular stricture of the ascending colon and had arranged an operation, the passage of a large liquid motion of healthy fæces made us postpone interference. The patient afterwards died, and the cæcum was found enormously distended, ulcerated, and perforated.

Pain is perhaps always present, and it is often of extreme severity. Brinton distinguished two kinds of it, which are not met with alike in all forms of the disease. One is a constant pain, the seat of which generally corresponds more or less closely with the position of the obstructed part of the bowel, and which consequently is very often referred to the right iliac fossa. There may be some tenderness with it, but this is not usually marked. Probably the pain itself is always traceable either to disorder of the circulation in the part of the intestine immediately affected, or to paralytic distension of that which lies above the obstruction; in the latter case, it may spread over the whole abdomen. The other kind of pain comes on in paroxysms. It evidently results from spasmodic contraction of some part of the bowel above the seat of disease, but, according to Brinton, only indirectly so; he thinks that its immediate cause is the increase of pressure in the distended coils of intestine close to the point of obstruction, towards which the waves of peristalsis set.

Vomiting occurs sooner or later in all forms of intestinal obstruction and in all cases. Its severity depends partly upon the seat and nature of the affection, but it is also liable to be greatly increased if the patient takes much liquid into his stomach. Brinton found that in animals in which he ligatured the intestine, the quantity of fluid which they drank had more influence than anything else in determining not only the amount of sickness, but also the rapidity with which death ensued. The matters rejected in cases of obstruction of the bowels consist at first of the gastric contents, then of bilious fluid from the duodenum, and afterwards of matters derived from the small intestine down to the obstructed part, and possibly sometimes from the colon. These almost always constitute a thin yellow liquid, which, if the seat of disease be high in the jejunum, may have merely a disgusting mawkish odour, but when the obstruction is lower the smell becomes decidedly fæcal at last. The stench is often so powerful as to fill the room in which the patient lies, and to be almost insupportable

by those about him. The cause of this "stercoraceous vomiting" has been matter of some discussion. At first it was universally believed to result from a reversal of the peristaltic movements of the intestine, the waves of which were supposed to travel upwards instead of downwards when obstruction existed. By Brinton, however, the occurrence of antiperistalsis was denied; he showed that even though the muscular coats of the bowel should continue to contract in the ordinary way, there would be a tendency to the formation of a double current in its interior, one downwards in the outer part of its channel, and another upwards in its central part, and he conceived that by this the contents of the whole alimentary canal above the seat of disease must soon be so completely mixed up that the fluid in the duodenum and even the stomach would acquire fæcal characters. Since that time, however, Engelmann has demonstrated the occurrence of reverse contractions in the intestines of animals, in which he had opened the peritoneal cavity; and in all probability antiperistalsis does occur in the human subject also, under conditions of disease. Indeed, it is difficult to account in any other way for the excessive distension of the cæcum in cases of stricture of the lower part of the large intestine. It is, however, certain that the direction of the waves of muscular contraction in the bowel is not entirely reversed; for, if it were, the part immediately above the seat of obstruction would not become distended; and such substances as mercury or castor-oil, when given by the mouth shortly before the patient's death, would not be found to have passed down to the stricture.

One result of stercoraceous vomiting is that, during the distressing efforts which accompany it, some of the fæcal matter may be sucked into the air-passages. This occurrence has been more than once noticed in the *post-mortem* room; when pressure was made towards the cut surfaces of the bronchial tubes, these gave exit to little yellow cylinders which certainly must have entered them during life; and in one case of strangulated hernia, in which death had arisen from peritonitis after relief of the obstruction, the lungs contained patches of gangrenous pneumonia, which had a yellow colour in the centre due, probably, to fæcal staining.

So much for the symptoms which are common to both acute and chronic cases of obstruction of the bowels. We now enter upon other points on which these two groups of cases present wide differences; and it will be convenient to deal first with the chronic.

Chronic obstruction of the bowels—of which rectal stricture is the type—is in the first place characterised by the slow or imperfect development of the symptoms already mentioned. Thus the *constipation* is sometimes incomplete, scanty fæcal evacuations occurring from time to time. Indeed, for several weeks or even months before obstruction definitely sets in the patient often has considerable and increasing difficulty in procuring an action of the bowels; or he may have repeated attacks of partial obstruction before the one which at length completely closes the intestine. Even when the constipation is absolute, it is wonderful how life is sometimes prolonged. The late Mr Cooper Forster recorded an instance in which there was not any action of the bowels for eighty-eight days. Again, *vomiting* is often absent in cases of this kind for some days or even weeks after the cessation of fæcal evacuations. And constant *pain* may be altogether wanting, the only pain being paroxysmal.

Visible peristalsis.—If in such a case the abdomen be examined during a

paroxysm of pain, it will often be found to present appearances which are almost decisive of the obstruction being chronic. The writhing movements of the intestine can be seen through the parietes; irregular elevations arise here and there, and are succeeded by depressions, or appear to travel from one part of the surface to another. For the production of these appearances to any marked extent, it seems to be essential that the coats of the bowel should have undergone hypertrophy; at any rate it is seldom or never observed in cases of acute intestinal obstruction. Peristaltic movements are much more often seen in the small than in the large intestine, but they may undoubtedly occur in the latter. Even during the intervals between the paroxysms of pain, the position of the different parts of the bowel is often distinctly visible through the abdominal parietes; and it is to be particularly noted that the transverse colon, when distended, does not continue to lie horizontally across the upper part of the abdomen, but bends downwards, so as to form a broad loop, lying vertically and (with the dilated ascending and descending colon) filling the whole front of the abdomen. On the other hand, the coils of the ileum, under similar circumstances, are generally arranged transversely. Now, as these coils are often quite as broad as the transverse colon would be, at least under normal conditions, the uppermost one, lying horizontally just below the ribs, may be mistaken for that part of the large intestine, if the facts just mentioned be not borne in mind. This error has been repeatedly committed. On the other hand, in the case of volvulus of the sigmoid flexure referred to above, we mistook the huge parallel and vertical limbs of the C for the loop of the transverse colon just mentioned.

The general symptoms presented by a patient suffering from chronic obstruction of the bowels are sometimes exceedingly slight, particularly when the treatment is judiciously managed. His pulse may be quite natural; there may be no fever; he may sleep well at night. The tongue may be clean, and food may be relished.

Such a patient, however, is always on the brink of a precipice. At any moment acute symptoms may supervene which may destroy life in a day or two, or still more rapidly. Probably in many cases these symptoms depend upon the occurrence of paralytic distension in the part of the bowel above the obstruction; for this condition is sufficient to account for them. It must also be remembered that ulceration is very apt to take place in the same part of the bowel; and this is unattended with symptoms. Thus one can never say when perforation may not take place.*

Thus it would be difficult to fix an average duration for cases of chronic obstruction, and if one could be fixed it would be of no practical value.

Diagnosis.—Chronic obstruction is sometimes caused by impaction of the large bowel with indurated masses of fæces; but among the diseased conditions that may affect the coats of the intestines there are only two—contraction and stricture—which run a chronic course; so that any case presenting the characters described in the last two paragraphs must belong to

* It once happened to me to send up from my out-patient room into the medical ward a woman who had cancer of the lower part of the sigmoid flexure. She had been ill for three months. I went up to see her later in the afternoon and she appeared to be perfectly comfortable, had a quiet pulse, and presented no urgent symptoms whatever. I therefore thought that the question of colotomy might be safely deferred until the following day. In the evening the nurse was turning her over to give her an enema when she suddenly expired. About a pint of liquid fæces was found in the abdominal cavity, which had escaped from an opening in the sigmoid flexure. There were also several large sloughing patches in the peritoneal covering of the cæcum, as well as its mucous lining—C. H. F.

either the one or the other. Now, as we have seen, contractions occur chiefly in the small, and strictures in the large, intestine. The distinction between them is therefore to be based mainly upon the points already mentioned as respectively indicating distension of the small or of the large bowel. There is also a difference in the shape of the abdomen when distended, according as the arch of the colon is below or above the seat of obstruction. In the former case the belly is rounded, projecting well forwards, but with comparatively little fulness of the lateral and lumbar regions. In the latter case it is more broad, and if the hand be placed on the patient's loins as he lies in bed, a feeling of resistance is experienced which is wanting when the small intestine is alone distended.

In some instances a tumour can be discovered on palpation; and this, or the fact that the pain is referred definitely to one particular spot, may suggest the exact seat of the mischief. Moreover, all the signs which show that disease of the large bowel is the cause of intestinal obstruction in reality indicate more than this fact, and point to the conclusion that the affected spot is situated below, or to the left of, the hepatic flexure of the colon. So far as these signs are concerned, chronic obstruction of the cæcum or even of the ascending colon is undistinguishable clinically from that of the lower part of the small intestine; for the transverse colon does not in either case become distended. We have seen, however, that the right side of the colon is but very little liable to disease in comparison with the descending colon, sigmoid flexure, and rectum.

With a view to treatment, it is of great importance to make out whether the seat of obstruction is below the middle of the descending colon or above it. In some cases one may perhaps determine this by placing each hand under one of the patient's loins, and by raising them so as to poise the two sides of the abdomen. If a fulness is felt in the right loin which is wanting in the left, it may perhaps be inferred that the ascending colon is distended. Another procedure which may throw light upon the question at issue is the slow injection of a large quantity of fluid into the rectum. Brinton believed that this was capable of yielding trustworthy conclusions as to the seat of the disease. According to this writer, when a pint is the most that can be thrown up, the obstruction is at the upper part of the rectum; a pint and a half, two pints, three pints, correspond respectively with different segments of the sigmoid flexure. The descending and transverse colon can be made to receive larger but more irregular quantities. In one case, in which it was evident that a stricture occupied the upper part of the ascending colon, nine pints of fluid were always found to be the most that could be injected. But it is to be borne in mind that a stricture may be pervious to fluid injected from below, although fæcal matter may be unable to pass through it from above. Thus, in one of the cases recorded in the 'Guy's Hospital Reports' (ser. 3, v. xiv), in which there was a mass of disease in the sigmoid flexure, just above the pelvis, four pints of warm water were injected, of which only a small portion returned, the rest having doubtless passed upwards through the affected part of the bowel.

It is well known that often in cases of cancer of the lower part of the rectum the fæces are narrow; and one patient with cancer of the upper part of the sigmoid flexure, declared that for three months he had noticed his motions to be smaller than natural. But even if this sign is constantly present when the fæces are hard, one must not forget that under conditions of temporary irritation of the colon, formed evacuations of soft consistence

may be much narrower than usual without there being any permanent affection of the bowel. Moreover, there must be a point above which disease of the colon would not give rise to any change in the size of the fæces, for they would be moulded into shape lower down.

Digital examination of the rectum is not to be omitted in any case of chronic (nor indeed of acute) obstruction; and if there is reason to believe that the seat of mischief is higher than can thus be reached, it may be well, when the finger fails to reach the diseased spot, to pass the whole hand into the bowel, the patient being under the influence of chloroform.

All appear to agree that introducing a long tube into the rectum is useless for purposes of diagnosis; it may catch against a fold of mucous membrane, or against the seat of obstruction, and in either case it may bend upon itself so as to appear to pass up much higher than it really does.

Beside the determination of the seat of obstruction, the pathological character of the disease is also a question for diagnosis, and one which it would in some cases be extremely important to settle, if only this were possible. We have seen that both strictures and "contractions" may be either simple or cancerous, and sometimes the discovery of a definite tumour shows that the latter is the case in a particular instance. But it is doubtful whether there is any other way of proving it. Cancer affecting the bowel is by no means confined to persons of advanced age. Among nineteen cases collected for the paper above quoted in the 'Guy's Hospital Reports,' six occurred in patients less than thirty-five years old. Moreover, malignant disease may be present in a person who looks well and has a florid countenance. Cancer of the bowel perhaps destroys life at too early a period of its growth to give rise to a proper cachexia. On the other hand, if the patient's health is broken down, one can seldom say that this malignant look may not have been the result of pain and sickness.

Treatment.—In cases of chronic obstruction the patient has often been taking powerful purgatives for a considerable time before he seeks medical advice. When this is the case it is generally advisable to abstain from the use of such drugs, and to employ enemata only. The latter are frequently of great service. In almost every instance there is at first a possibility that the disease may be merely an impaction of the contents of the intestine; and even when organic disease of the coats of the bowel is present, the systematic administration of enemata, with or without the use of gentle laxatives, not infrequently removes all the symptoms for a time. The constipation is, indeed, almost certain to return after a shorter or longer interval, and even if it should once more yield to similar treatment, the period at length arrives when the bowels remain occluded in spite of all that can be done. Then, if not before, the patient must cease to take purgative medicines. It is wrong to prescribe them when peristaltic movements can be felt or seen, when the abdomen is becoming rapidly distended, or when symptoms of collapse or of severe constitutional disturbance appear. But small doses of olive oil, as in our Mistura Olei,* are admissible when ordinary aperient drugs ought not to be given. Enemata may be continued somewhat longer still, but at last they too must cease to be administered.

The remedy which should now be prescribed is opium, and that not sparingly; from half a grain to a grain may be given every four hours, or even every two hours, according to circumstances. Some add to it a quarter

* ℞ Ol. Olivæ ʒj, Liq. Potassæ ♏iv, Aq. dist. ad ʒj. Misce.

of a grain of extract of belladonna, believing it to quiet, instead of exciting, the peristaltic movements.

If the patient should suffer greatly from flatulence, the application of turpentine stupes to the abdomen may now be useful. Another measure to which recourse may be had for the relief of this symptom is puncturing the intestine with a fine trocar. The late Mr Stocker long ago performed this operation, and Dr Braxton Hicks once let out a large quantity of flatus by this means. Moreover, the introduction of a sharp instrument into the bowel appears to act as a very powerful stimulus, for fæcal evacuations have often been passed soon afterwards, and that this is not due to the escape of the gas is evident from a remarkable case of the editor's in which puncturing the abdomen in five places with a grooved needle, although neither fluid nor gas was withdrawn, led to complete subsidence of all the symptoms of intestinal obstruction and to the prolongation of the patient's life for several months. In chronic cases there is little or no fear of the escape of the contents of the bowel into the peritoneal cavity, for the mucous membrane protrudes into the minute aperture so as to close it; but when the intestines are inflamed, the procedure can no longer be regarded as safe.

As soon as it is evident that a case of chronic obstruction will not yield to medical treatment, the question arises whether the bowel should not be opened at some point above the obstruction, so as to establish a fæcal fistula. Now, if one can clearly make out that the seat of disease is below the descending colon, the splenic flexure should be selected for the operation. On the other hand, if there be a doubt whether the obstacle lies below the descending colon, but none that it lies below the ascending colon, the latter must of course be opened. In either case the operation of colotomy is, as a rule, successful; the peritoneum need not be wounded, and the patient's life is often prolonged for months or even for two or three years. This operation should not to be delayed for many days after the time at which the administration of purgatives and enemata is discontinued. As a striking instance of the benefit that may result from colotomy, may be mentioned a case of Hilton's recorded in the 'Guy's Hospital Reports,' 1868, p. 219. There had been absolute constipation for twenty-eight days, but four days after the operation fæces began to pass through the rectum, and in a short time the wound in the loin closed. The symptoms, however, afterwards returned, and it was necessary to reopen the colon. After this a dilator was introduced twice a day with the object of keeping open the fistulous passage; but, in spite of this, it again became occluded. The patient, however, who was himself a medical man, began to regain his strength, and resumed his professional duties, being able to visit thirty families a day without too great fatigue. Ultimately he died of abscess in the left iliac fossa, which commuuicated with the interior of the hip-joint. The cause of the obstruction appeared, *post mortem*, to be a simple puckering of the coats of the sigmoid flexure at one spot.

Mr Bryant brought before the International Medical Congress of Copenhagen, in 1884, a remarkable series of 82 cases of colotomy performed by himself. Of these 60 were for cancerous and 19 for non-cancerous stricture, 2 for external obstruction, and 1 for volvulus of the sigmoid flexure. As the result, 26 of the patients operated on died within a month; but for the rest the operation gave marked relief, and prolonged life—in 16 patients for between one and six months, in 8 between six and twelve months, in

12 from one to five years, and in 5 for longer periods. The remaining 8 were Guy's Hospital patients, who left the wards convalescent some weeks after the operation, but were not traced subsequently. At the same meeting Mr Henry Morris contributed 23 cases, of whom 14 recovered from the operation, and Professor Studsgaard 20, of whom 17 survived for periods varying from a month to several years.

It is another matter when stricture or contraction affects the small intestine, or even the cæcum or ascending colon. In such cases the only thing that can be done is to open one of the distended coils in the groin, as advised by Nélaton. The peritoneum must then be wounded; and the chance of establishing adhesions and forming a fistulous opening must be small, unless the serous space should have been closed by previous inflammation. This procedure has occasionally succeeded, but is seldom justifiable.

The symptoms of acute obstruction of the bowels—of which strangulated hernia is the type—must now be considered. They differ to a considerable extent from those which belong to the chronic forms of the disease. The *constipation* is always absolute; any small fæcal masses that may be brought away come from below the occluded part of the intestine. The *vomiting* is early and severe. Fixed *pain* is seldom absent. The abdomen rapidly becomes distended, but its form presents comparatively little that is distinctive of one form of obstruction rather than another. Peristaltic movements are rarely, if ever, to be seen through the parietes, but the form of the intestinal coils may be plainly visible.

The most striking feature of cases of acute obstruction is, however, the early development of a symptom that has not yet been mentioned, namely, *collapse*. The face becomes sunken, with pinched cheeks, and dark circles round the eyes; the extremities are covered with a cold sweat; the pulse is very rapid and small, the voice is high pitched and feeble or whispering. The patient, however, often retains perfect consciousness, and is able to lift himself up in bed until just before his death. So close may be the resemblance between the general condition of a man suffering from this form of obstruction and that which occurs in Asiatic cholera, that during one epidemic a case at Guy's Hospital was actually supposed to be one of cholera with retention of the rice-water evacuations, until on *post-mortem* examination the disease proved to be strangulation of the intestine.

In acute obstruction of the bowels *the patient passes little urine*, and the secretion may be altogether suppressed. The late Dr G. H. Barlow, who first noticed this symptom, thought it was an indication that the seat of disease was high up in the jejunum, supposing that it depended upon diminution of the area for absorption of fluid. Subsequently Dr Habershon attributed it rather to the urgency of the vomiting which occurs when the upper part of the gut is strangulated; and Dr Brinton further pointed out that the mucous membrane above the seat of obstruction becomes a secretory rather than an absorbing surface. Both these writers admitted the fact, that when the urine is suppressed the disease is high up in the bowel. But, as was pointed out in the chapter on cholera (vol. i, pp. 283-4), there is reason to believe that this symptom is really one of the phenomena of collapse, and occurs in all forms of intestinal obstruction, whatever their seat, in which collapse is present.

Diagnosis of cause.—We have now to consider in what way, and to what extent, the different conditions that may cause acute obstruction of the bowels

can be distinguished from one another. In the first place the fact must be insisted upon that there is no one form of disease, capable of giving rise to obstruction, which may not present itself with acute symptoms. For instance, even in cases of stricture of the large intestine, constipation sometimes sets in suddenly, and quickly leads to vomiting and to collapse. The explanation appears to be that the bowel is then occluded, not directly by the disease of its walls, but indirectly by muscular spasm, or by the bending over of the portion of intestine above, which had gradually become overloaded with its contents. The proof of this is that, as in Hilton's case mentioned on the last page, when colotomy is performed, fæces soon begin again to pass through the natural passages. At the *post-mortem* examination, too, one can often pass the finger through a stricture which had caused obstinate constipation during life. As a rule, indeed, when acute symptoms are present in a case of stricture or contraction, these have been preceded by less grave symptoms for some days or even weeks. But in hospital practice it may be quite impossible to elicit this fact when the patient is admitted at an advanced stage of the disease. Thus there may be scarcely anything to distinguish the case from one of some essentially acute form of obstruction. The points of most importance, as indicating that the affection is of the latter kind, are its having commenced with absolute suddenness, the patient having been free from all intestinal symptoms until the moment when he was attacked.

The pathological conditions which may be regarded as the proper causes of acute obstruction of the bowels fall under four principal heads:

1. *Constriction*, or *internal strangulation*, both affecting chiefly the small intestine.
2. *Volvulus*, affecting most frequently the cæcum or sigmoid flexure.
3. *Impaction of a gall-stone* affecting the small intestine only.
4. That form of *intussusception* in which its own characteristic symptoms are absent; here the small intestine is almost always the part concerned.

Now, as regards clinical differences between these several affections unfortunately little can be said.

It is to be particularly noted that with *volvuli* there is little delay in the occurrence of sickness, as in the other forms of obstruction of the large intestine; on the contrary, all the symptoms develop themselves with peculiar rapidity; the abdomen becomes quickly distended in the greatest possible degree, and death may occur within three or four days. Previous constipation is the rule.

Impaction of a *gall-stone* in the small intestine is to be distinguished, if at all, by the fact that it occurs chiefly in elderly women.

Among the numerous causes of internal *strangulation* no diagnosis can as yet be attempted, the only fact worthy of mention being that obstruction by a band connected with a diverticulum scarcely ever occurs except in males, and chiefly in patients under twenty years of age.

Ætiology.—With regard to the causes of acute obstruction generally, there is very little to be said beyond what has been already mentioned incidentally. In the great majority of cases no directly exciting cause for the attack can be discovered, but it sometimes happens that the patient may shortly before have eaten something, as, for instance, high game or venison or raw fruit, which may be supposed to have set up excessive peristaltic action in the bowels. In other cases a fall or blow upon the abdomen seems to have been the starting-point of the disease; it was so, for instance, in a boy who

died in Guy's Hospital of strangulation of the ileum by a diverticulum, and who had a bruise in the right iliac fossa, the result of his having fallen upon some large stones.

Prognosis.—The *duration* of acute obstruction of the bowels is subject to considerable variations which (so far as their cause can be traced) appear to depend on the length of bowel which has its circulation arrested or is affected with paralytic distension. Mr Phillips has recorded one case in which death occurred within thirty-three hours. But such cases as this are very rare, life being almost always prolonged for three or four days, and generally beyond the first week. Indeed, one may sometimes avail oneself of this for the purposes of diagnosis; a case of obscure abdominal disease which terminates fatally within two days is much more likely to be perforation of the stomach or intestine than obstruction of the bowels.

Treatment.—In the treatment of acute obstruction of the bowels, the first point to be considered is whether one should recommend the performance of a surgical operation, that of opening the abdomen and searching for the band or other constricting agent, with the object of cutting it through and releasing the bowel. The analogy of herniotomy is all in favour of such a course if only one can be sure of the nature of the disease; and every pathologist has met with cases in which as soon as the intestines were exposed a band was seen, which could have been divided without the slightest difficulty. Moreover, there are cases in which this has actually been done during life with the result of saving the patient's life.

But, before we can decide as to the advisability of the operation of laparotomy, two or three questions must be answered. First, have we the means of distinguishing those forms of intestinal obstruction in which it would be of service from those in which it must necessarily fail? The answer to this cannot be regarded as satisfactory. By carefully selecting for abdominal section cases which presented in the most typical form the symptoms of acute obstruction, one could probably make it a matter almost of certainty that the operation should not be undertaken in a case of stricture or even of contraction. By excluding cases in elderly women, one would reduce to a minimum the chance of finding that the cause of obstruction was a gall-stone impacted in the intestine. Volvuli and the different varieties of internal strangulation would all be fair objects for such a procedure, although no doubt some of them would be far more favourable for it than others.

But another question has now to be answered, namely, whether the cases that would thus be selected for an exploratory operation are sure, if left to themselves, to terminate fatally; or rather whether the risk to life would be greater under such circumstances than if the operation were performed. Some years ago the author searched the records of *post-mortem* examinations at Guy's Hospital very carefully to find any case of internal strangulation of the intestine in which recovery had taken place and the patient had subsequently died of some other disease. The only instance discovered was that of a man who had been admitted with constipation and stercoraceous vomiting under the care of Dr Addison, and who got well and afterwards died of phthisis. But in that case, although there were two loose bridles, either of which might have strangulated the bowel, there was also adhesion of a coil of small intestine by another very short bridle, and the appendix cæci was firmly bound down. Pathologically, therefore, the case probably belonged to the class of "contractions" rather than to that of internal strangulation. Thus, so far as our experience goes, it lends no support to the

opinion that where there is actual mechanical constriction of a part of the bowel, this is ever released by natural processes. One can, indeed, well conceive that such a thing might occur. It is evidently possible for the affected portion of intestine to be disengaged by the peristaltic movements of the portion above, or for the constricting band, which is often very thin, to give way beneath the pressure to which it is subjected. When the cause of strangulation is a band attached to a diverticulum of the ileum, this is commonly in a sloughing state at the time of the patient's death; but unfortunately its tendency is to give way where it joins the bowel, so that the necessary result would be a fatal extravasation of fæces.

It is, however, undeniable that recovery sometimes takes place in cases which have presented all the symptoms of internal strangulation. As an example may be cited the case of a medical student who was under the author's care in 1874. He was first seized with abdominal pain one Sunday morning about 7 a.m., and soon afterwards had slight vomiting. There was absolute constipation, although he took several doses of purgatives. On the Tuesday his abdomen became distended, the coils of intestine being visible through the parietes. On the Wednesday, when seen for the first time, his face was shrunken and his extremities were cold. The sickness was severe; and on the Thursday afternoon he rejected a large quantity of brownish liquid which evidently came in part from the intestine; and although it did not actually possess a fæcal odour, it seemed to indicate the near approach of stercoraceous vomiting. That night, however, he passed an offensive stool containing numerous scybala; his urine at once became copious, and all his threatening symptoms quickly passed off. As he got better, one could feel an indurated mass in the region of the cæcum, and he had a relapse of short duration, in which there was an increase of tenderness in this part of the abdomen. There is therefore little doubt that the case was really one of typhilitis and not of mechanical obstruction.*

* For my own part, I am inclined to believe that in all probability whenever recovery takes place after symptoms of internal strangulation of the intestine, the disease has really been not mechanical obstruction at all, but inflammation of some part of the bowel, affecting mainly its serous surface; or, in other words, a local peritonitis. I have twice seen an exploratory operation performed when the cause of the symptoms has proved to be suppurative peritonitis starting from ulceration of the appendix vermiformis. A critical review of the history of each of these cases does indeed reveal some differences between their symptoms and those which belong to true internal strangulation, but it is easy to be wise after the event. (See on this point Mr Bryant's lecture 'Brit. Med. Journ.,' 1884, ii, p. 1182.)

I was myself at one time strongly disposed to advocate the performance of laparotomy, but I must confess that I am now very doubtful about it. I have seen several cases in which it has been done, and almost every one has terminated fatally. I believe, indeed, that in each of them death would have occurred within a few hours if abdominal section had not been attempted, but this only shows that if the operation is to have a chance of success it must be undertaken at an earlier period of the disease, before the patient becomes collapsed or is worn out by pain and suffering. This, in fact, is what Mr Howse recommends, and he has given several valuable directions in regard to the employment of the antiseptic method in such cases; but I fear that if such a course were adopted the disease would sometimes be found to be typhlitis. In that case one can hardly help thinking that the operation would remove the patient's last chance of life; yet it is true that nothing but acute peritonitis was found in a patient on whom Dr Buchanan, of Glasgow, operated, and the patient began to improve from the time when his abdomen was opened, and ultimately got well. On this point see the chapter on Peritonitis, *infra*, p. 473.

To sum up, then, I am inclined to think that but few successes are likely to be attained by the operation of abdominal section in cases of internal strangulation, and there would be at least an equal number of cases which would have done well without it, and in which, instead of increasing the chance of the patient's recovery, it would rather augment his risk. The question, however, can only be settled by a wider experience.—C. H. F.

If one should not intend to operate, there can be no doubt of the advantage derived from the free use of opium. A dose of one grain should be given every four, three, or two hours, according to circumstances; it often affords marvellous relief. All sickness and pain may entirely pass off for the time; at the worst the patient's death is freed from the suffering which would otherwise have attended it; and if spontaneous subsidence of the disease be possible, this chance is greatly increased.

Under no circumstances should a single dose of purgative medicine be prescribed, even at the very commencement of a case of acute intestinal obstruction.

A course which may be regarded as intermediate between opening the abdomen and trusting entirely to the efforts of nature is what Mr Hutchinson has termed abdominal taxis, *i. e.* inverting and shaking the patient, injecting large enemata from a height, and kneading the bowels, in the hope of mechanically reducing the displacement. This is undoubtedly sometimes followed by immediate recovery. Sir Thomas Watson relates the case of a lady who observed that the hands of two other medical men who were seeing her with him in consultation were heavy as they manipulated her abdomen; she fancied that their pressure had displaced something within, and almost directly afterwards she passed a liquid motion. Some years ago the procedure of kneading the abdominal parietes was adopted in a case which the author had seen in consultation a few days before, and within a very short time the bowels acted. But striking as such cases appear, it is quite possible recovery would have occurred if no manipulation had been attempted, and it is obvious that the treatment is fraught with danger, of which the extent cannot possibly be estimated. The same remark applies to the almost forgotten treatment by making the patient swallow metallic mercury, which has been recently revived.

Conclusion.—It is, no doubt, theoretically possible to treat every case of intestinal obstruction, which does not yield to medical treatment, by abdominal section, followed by operative release of the gut, or, when this is impossible, by resection of the strangulated or invaginated portion or of the volvulus, or by making a permanent fistula between the gut above and below the obstruction. But practically the difficulties would be enormous, and in most cases insuperable. We must remember that the actual mortality after laparotomy is far greater than is indicated by the published cases, and that the unavoidable delays in the course of opening the abdomen and searching for the seat of disease are far greater than might be supposed beforehand.

The appropriate treatment of typhlitis, of invagination, of impaction, and of stricture of the rectum or colon, is, we have seen, tolerably clear, and on the whole successful. In cases of internal obstruction, the seat and nature of which can be diagnosed with some approach to certainty, the results of an exploratory operation with a defined object are likely to justify the risk. But where, as unfortunately is most often the case, we have no such approach to certainty, it is very doubtful whether the expectant treatment—enemata followed by starvation and the full exhibition of opium—does not afford the patient a better chance of life than laparotomy. It is, however, fair to remember that the want of power on the physician's part to form a diagnosis frequently leads to so long delay that it is vain to expect a good result from the most skilful surgical treatment. Here, as elsewhere, improved practice must wait upon improved diagnosis.

The subjoined summaries of the result of treatment of these deeply interesting cases form a practical illustration of the preceding statements.

a. The following are the results of eighteen recent and consecutive cases of internal strangulation or other acute and severe obstruction (excluding herniæ, strictures, and impactions) collected by our medical registrar, Dr Shaw, from the clinical and *post-mortem* records of Guy's Hospital.

Eight patients out of the eighteen recovered, after periods of complete constipation varying from five to eight days in six cases, extending to twelve and fourteen days in the other two. In all these vomiting and other serious symptoms were present. All were treated by opium or by opium and belladonna with the frequent use of enemata, and without an operation.

Five patients died without operation, under the same general treatment as was used in the preceding cases. In all these thirteen cases the causes of obstruction were found to affect the small intestine. They were a kink of the gut, fatal on the thirteenth day; strangulation by a band in two cases, one fatal on the eighth day, the other protracted for four weeks: volvulus of the small intestine in two cases, fatal on the eighth and ninth days.

Five of the patients died after an operation. In all the bowel was found strangulated, in three by a fibrous band and in one by the appendix cæci. In three the constricting band was divided and the gut liberated; in two a fistulous opening was made in the bowel above the constriction. The operation took place on the seventh, eighth, ninth or eleventh days; later, no doubt, than the surgeon would have wished.

b. The following results refer to cases which have been under the editor's personal observation. Of the total number of patients (forty), thirty were male and ten female.

The youngest patients (18 months and 5 years) suffered from invagination; fifteen cases of strangulation (or contraction with adhesions) occurred at various ages, from $5\frac{1}{2}$ years to 46; two of volvulus at 23 and 31; two of impacted gallstone in women at 60 and 78; three of fæcal impaction in men above 50; and fourteen of cancerous stricture of the colon or rectum at 24, 38, 41, 44, and between 60 and 78.

Excluding the cases of stricture of the large intestine (in which the only treatment adopted was colotomy) the five cases of impaction recovered under belladonna and enemata; both cases of invagination died, one after successful laparotomy; and both cases of volvulus died, one after unsuccessful laparotomy.

Of the remaining fifteen cases of acute obstruction due to bands, internal hernia, adhesions and contractions, one recovered after abdominal taxis, and two under expectant treatment by starvation and opium; six died under the latter treatment, and four after laparotomy; while two recovered from contraction (the result of a precedent strangulated hernia) after an exploratory operation.

INTESTINAL WORMS

(CESTOID AND NEMATOID ENTOZOA)

TAPEWORMS.—*Anatomy and transformations—Tænia solium—its distinctive characters and distribution—its relation to Cysticercus cellulosæ of pork.—Tænia mediocanellata—its specific distinctions—its relation to the cysticercus of beef—its geographical distribution.—Bothriocephalus latus—its characters and distribution—its probable origin—Symptoms of the presence of tapeworms generally—Preventive and curative treatment.*

ROUND-WORMS.—*Ascaris lumbricoides—its anatomy—natural history—distribution—symptoms, diagnosis, and treatment.—Thread-worms—anatomy—symptoms and treatment—Trichocephalus dispar—Eustrongylus gigas.*

SCLEROSTOMUM DUODENALE.—*Its discovery — anatomy — habits — Anæmia caused by its presence—Treatment.*

TRICHINA SPIRALIS.—*Its discovery—anatomy, transmigrations, and encapsulation.—Trichiniasis—its symptoms, diagnosis, treatment, and prevention.*

FILARIA SANGUINIS HOMINIS.—*Its discovery in the blood—its transmigrations—Echinorhynchus—Dracunculus—Distomum—Bilharzia.*

THE human alimentary canal, as is well known, affords shelter and food to several species of animals, which are commonly known as intestinal worms. By zoologists these are placed in two separate groups, Cestoidea and Nematoidea. Their differences are very great.

There are also other vermiform parasites which inhabit the muscles and the blood, which may be more conveniently considered in this chapter than elsewhere, particularly as it is probable that they all inhabit the intestines at some period of their lives. On the other hand, the hydatid stage of Cestoidea will for clinical purposes be deferred to the chapter which deals with the diseases of the liver.

The Nematoidea, or "Round-worms," have long cylindrical bodies; they are provided with an alimentary canal, they are males or females, and they undergo but slight changes of form after leaving the egg.

The Cestoidea, or "Tapeworms," are flat, riband-like creatures, made up of a number of joints, which are arranged in a line from one end to the other. They have no alimentary canal. Each joint has a double sexual apparatus, both male and female. The joints are apt to become detached, and are then capable of maintaining for a time an independent existence. We will consider them first.

CESTOID WORMS.*—There was formerly much discussion as to the application of the term "individual" in the case of a tapeworm. On the one hand, a joint after its separation would seem to deserve this title; on the other

* *Synonyms.*— Tæniada — Tapeworms, including Flat-worms and Bladder-worms or Hydatids.—*Fr.* Vers cestoïdes, kystes hydatiques.— *Germ.* Bandwürmer and Blasenwürmer.

hand, the animal has at one end a so-called "head," provided with suckers, and often with a circle of hooks which fastens itself to the intestinal wall, and which at first sight suggests that the entire tapeworm should be regarded as the individual. As Leuckart admits, this view derives support from the fact that the movements of the creature take place by waves transmitted from one joint to another, large portions of it shortening or lengthening at the same time, as though the joints were all under the influence of a common directing impulse. But even if we deny to each segment the rank of an "individual" (as we deny it to the components of a compound salpa or of a polyp-colony) it is impossible to extend the term from such compound organisms as a sponge or a tree to the unconnected products of gemmation, some living and some dead. Yet this is logically required by adopting the definition of an individual as "the total product of a single fertilised ovum until fertilization is repeated." The fact is that the conception of an individual was originally derived from human consciousness, and is inapplicable to many of the lower classes of animals and to most plants.

By zoologists the tapeworm as a whole is called a *strobila*, the head (before budding) was a *scolex* or budding larva, and the separate joints are known as *proglottides*.

In the course of their development the Cestoidea pass through a most extraordinary series of changes, which bring them within the scope of pathology at various points, and it will be convenient to give a general account of these before describing individual species of tapeworms.

For a reason which will presently appear, the Cestoidea very rarely occur in the alimentary canal of any but carnivorous animals. Let us then suppose that a tapeworm is present in the intestine of a man, or of a dog or cat. Its joints or proglottides are by no means all alike. Those nearest the head are very small, and appear almost structureless; for the development of new proglottides is constantly going on at this part of the strobila, and these, as they are formed, separate the scolex further and further from those which preceded them. Thus, the greater the distance from the head the older are the joints, and towards the distal end of the tapeworm they are fully developed and possess elaborate hermaphrodite sexual apparatus. Here the ova are developed and impregnated. In each is presently formed an embryo, which is a globular body, provided at one part of its circumference with six curved hooks arranged in pairs; it is enclosed in a thick shell.

It does not appear that the ova are ever discharged from the proglottis through the genital canal or "vagina;" they are, indeed, too large to pass through it; they remain *in situ*, until the proglottis is ruptured and a way is made for their escape. In the meantime the proglottis enters upon a more or less prolonged series of adventures. We have seen that the tapeworm is constantly forming new joints near its head. At its other end the mature joints are as constantly being cast off. Thus every proglottis in turn is pushed on, until by the time that its ova with their embryos are fully developed it reaches the distal extremity of the tapeworm, and in its turn becomes detached. When this has happened it is either discharged with the fæces of its host, or wanders out of the rectum by its own movements; or perhaps it may be ruptured while within the intestine, in which case its ova are expelled with the fæces.

Having reached the external world, the proglottides creep about for a time. If warmth and moisture favour them, they remain alive and active

for some days. Leuckart supposes that they may crawl up the stalk of a plant or a blade of grass, and with this be swallowed by some herbivorous animal. Probably it more often happens that they die and become disintegrated, or that, as Cobbold suggests, the growth of the multitude of ova within them causes them to burst. In either case the ova escape and become scattered in all directions. Some perhaps are carried into streams and ponds, others on to the stems or leaves of plants, where they retain life for several days under favourable circumstances. The immense majority of them no doubt perish, but from time to time one of them meets with the fate which is necessary for its further development.

This fate consists in its being swallowed by some particular species of animal, which is generally herbivorous, but which may swallow the tapeworm ovum either in the water which it drinks, or in the food which it eats. As soon as the now ripe egg reaches the stomach of this animal, which is in future to be its host, its shell is dissolved by the action of the gastric juice, and the six-hooked embryo (larva or scolex or proscolex) is thus set free. It immediately starts upon an active migration. By means of its hooks it bores through the walls of the stomach or intestine of its host. In this way it is very likely to enter some radicle of the portal vein; and, being washed away by the current of the circulation, to be carried to the liver. In other instances it perhaps continues its active movements through the tissues, until it has reached some other organ far from its starting-point. However this may be, its migration ultimately ceases, and it takes up a position in some part of the body of its host, and there undergoes an entirely new phase in its development. In the first place it begins to grow, and loses its six hooks. It becomes surrounded by a layer of granular matter, which is an exudation from the tissues of its host. Within four or five days from the time when a rabbit was made to swallow the ova of a tapeworm, Leuckart found, on killing it, that its liver and lungs were studded with minute white grains, exactly like miliary tubercles, but each having in its centre a tapeworm embryo.

The embryo still goes on increasing in size, and when it has reached a diameter of 0·6 to 0·8 mm., it becomes hollow in the centre, the cavity being filled with a transparent watery fluid. From this time it presents the character of a more or less globular vesicle or bladder; and as it was recognised in this condition long before its relation to its tapeworm parent was understood, it was formerly known as a *bladder-worm*, or hydatid. The *echinococcus* of the human liver, the *cysticercus* of "measly" pork, and the *cœnurus* found in the brain of sheep, are all examples of the hydatid or cystic stage of a tapeworm. All of these are surrounded by capsules of fibrous tissue derived from the tissues of their host, in which they lie free and unattached, but which grow as they grow, and fit tightly to their exterior. It is from the blood-vessels of the capsule that they derive their nourishment.

After a time the growing bladder-worm begins to show a projection from one part of its inner surface, and this gradually increases in size and becomes pear shaped. Soon four suckers make their appearance in the interior of this body, and a circle of minute hooks; it thus acquires a striking resemblance to the head of a tapeworm, and after a time a kind of neck becomes developed, by which it is suspended in the interior of the hydatid. In strictness it should be added that the likeness is not exactly to a tapeworm head as one is accustomed to see it, but as it would appear if it were

withdrawn into its body, just as the finger of a glove may be turned inside out.

In this condition the bladder-worm may remain quiescent for a lengthened period, embedded in the tissues of its host. It may die there, and its remains shrivel up until only a small cheesy or calcareous relic is left. But if its host should die first it may be set free, and then its transformations may recommence. The condition required for its further development is that it should be swallowed by a carnivorous animal, with or without the tissues in which it is embedded. Thus the cysticercus or measle of pigs is swallowed by men or dogs; the echinococcus cyst of sheep or men by dogs or wolves.

Having thus reached the alimentary canal of a new host, the bladder-worm enters upon a series of changes, which end by its conversion into a tapeworm. In the first place, the parts which have been described as resembling a tapeworm's head and neck, inverted like the finger of a glove, now turn themselves inside out. Thus, instead of being suspended in the interior of the bladder-worm, the head and neck come to project from its exterior, so that some writers now call the original sac the "caudal vesicle." This, however, has but a brief existence. It is speedily dissolved by the gastric juice, except a small remnant, which for a time may be observed attached to the neck. The head and neck resist the solvent action, and pass on into the intestine. There they take on an active process of growth. Within a few days transverse lines show themselves on the neck, and these increase in size and multiply by gemmation, until in the course of some weeks a jointed tapeworm or strobila is developed. The circle of changes undergone by the parasite is thus completed. We have arrived at the point from which we started at p. 436.

It must be added that in individual species some of the steps in this marvellous series of transformations deviate slightly from the account just given. But all the Cestoidea, without exception, require two different hosts for the completion of their existence. The one host in which the entozoon assumes the form of a bladder-worm may be either herbivorous or carnivorous, but is most often the former. The other, in which the parasite becomes a tapeworm, must always be carnivorous, since it is only when swallowed with animal tissues that the bladder-worm is likely to enter the alimentary canal of its host.

As may well be supposed, the manner in which tapeworms are developed, and their relations to their respective bladder-worms, have only been discovered by patient investigations continued through many years. More than a century ago, in 1769, Pallas noted the close resemblance between common tapeworms and the *Cysticercus tenuicollis* from the abdomen of ruminants. But it was not until the year 1845 that the first definite suggestion as to the nature of bladder-worms was propounded. And even then the idea was not that they constituted a regular stage in the development of tapeworms, but rather that they were tapeworms which had "strayed" into a wrong animal, and had consequently become dropsical and degenerated. Very soon, however, this was shown to be a mistake, and in 1851 Küchenmeister administered the *Cysticercus pisiformis* of the rabbit to dogs and succeeded in rearing in their intestines the *Tænia serrata*; he also gave the *Cysticercus fasciolaris* of the rat or mouse to cats and found that it became developed into the *Tænia crassicollis*. In 1853 the first experiments of the converse kind were performed by the same observer proglottides of

the *Tænia cœnurus* of the dog were given to sheep and lambs with the result that bladder-worms (cœnuri) were found in their brains, the symptoms of "staggers" being also present, which are well known to be caused by this parasite. Since that time similar investigations have been prosecuted with many different species, and the result is that we have now complete experimental proof of the relations and mode of development of many of the Cestoidea.

The number of species of tapeworm which have been known to occur in the human alimentary canal amounts to seven or eight, but of these only three are common enough to require description in a work like the present.

1. *Tænia solium* was until recently believed to be the most common human tapeworm. When fully developed it measures from seven to ten feet in length, or possibly more. Its "head" is as big as that of a pin. This is provided with four suckers and with a proboscis on which is a circle of about twenty-six hooks, ranged with their points outwards. They are of two sizes, and are large and small alternately. The head is often black from the presence of pigment. The "neck" measures an inch in length. The joints are at first very small and broader than they are long. They gradually increase in breadth and still more in length, so that at about a yard from the head they are square, and towards the distal end of the strobila their length is considerably greater than their breadth. There the ripe segments measure about half an inch long by a quarter of an inch in breadth. They have often been compared to melon seeds and are in fact not unlike them.* The "genital pores" or orifices of the sexual apparatus are placed in a little papilla which is easily recognised on one of the free edges, more or less regularly on the alternate sides of each successive joint. The "uterus" consists of a central passage, running in the length of the proglottis and giving off at right angles from seven to ten branches on each side, which again have complex secondary branches. A good way of observing their characters is to compress a tapeworm-joint slightly between two plates of glass and hold it up to the light. The eggs are globular and measure 0·03 mm. in diameter. They have a shell which appears to be of considerable thickness, this being, however, the result of the presence of a number of rod-shaped projections which closely cover its surface and which under the microscope give it the aspect of being marked with a number of minute radiating lines.

The bladder-worm which forms a stage in the development of the *Tænia solium* is called the *Cysticercus cellulosæ* (*sc. telæ*). It is found chiefly in the pig, occasionally in the monkey, the dog, and some other animals, including man himself. In the pig it occurs principally in the connective tissue between the fasciculi of the voluntary muscles, where it is commonly called a *measle*, but also in the liver or the brain. Its relation to the tænia would be rendered probable by the identity of the scolex which it contains with the head of that creature. But this point has been conclusively demonstrated by experiments of both kinds. Van Beneden, Leuckart, and others have administered proglottides of the tapeworm to pigs; and the result has repeatedly been that the flesh of the animal has become full of cysticerci,

* Hence they were called *cucurbitæ* and the worm *Tænia cucurbitina*. According to Küchenmeister, the Arabs call the complaint "Chabb-al-kar," *i. e.* pumpkin-seed.

the size of which has corresponded with the length of time that may have been allowed to pass before it was killed. Two months and a half are required for the full development of the cysticercus. From observations made by Stich it is probable that its life within the tissues (at least in man) is limited to from three to five years; he found that at the end of such a period cysticerci in the subcutaneous tissues, which had been plainly felt through the integument, became flaccid and shrank away until their presence could no longer be discovered.

The converse experiment to that of feeding pigs with the proglottides of the tænia of course consists in the administration of cysticerci to human beings. This has been occasionally done, the victims being sometimes criminals condemned to death, sometimes persons who volunteered for the purpose. Perhaps the most striking instance is one of Küchenmeister's. He gave to a criminal twenty cysticerci on each of two occasions, one of which was four months, the other two months and a half before his execution; nineteen tapeworms were afterwards found in his intestines. A young man who of his own accord swallowed four cysticerci in Leuckart's presence began, for the first time in his life, to pass proglottides in his fæces three months and a half afterwards, and a month later took a dose of kousso, with the result that he passed two tapeworms each about two yards long.

The name *Tænia solium*, given to this parasite by Linnæus, was meant to imply that it occurred singly in the intestine;* and the same notion is expressed by the French title *ver solitaire*. But this is a mistake. Two or three are not uncommonly present in the same individual, and cases are recorded in which twenty-five have been passed by a single patient.

This parasite is more common in adults than in children, and it has been often found in butchers and in cooks; these facts are of course just what might be expected, since it is derived from measly pork. Out of Europe it is said to have been observed only in India, Algiers, and North America. The duration of life of this tapeworm is estimated by Leuckart at from ten to twelve years. Cobbold mentions the case of a patient who was infested with it for sixteen years. It is said to have been present for as long as thirty-five years; but Leuckart thinks it probable that in such instances the worm was not the *T. solium* but the *T. mediocanellata*, which will be presently described.

The cystic form.—Cysticercus cellulosæ is also sometimes found in the human subject, and this is the only known instance in which man is liable to both the larval and mature forms of a cestode entozoon. As a bladder-worm, the parasite is observed most commonly in the eye and in the brain, but it is very likely that it is really most frequently present in the muscles and subcutaneous tissue, where, however, it is very apt to escape notice. It appears to be found from time to time in the German dissecting-rooms. It is often solitary or present only in small numbers, but in Stich's case at Berlin more than three hundred could be felt through the skin. A person who has a tapeworm in the intestine cannot derive cysticerci directly from its ova; they must first pass through the stomach, where their shells are removed by the action of the gastric juice. Still it is remarkable that such patients do not more commonly become affected with bladder-worms.

* It should then have been written *T. sola*. The same notion was expressed by the specific name *T. solitaria* (Bradley). *T. cucurbitina* (Pallas) referred to the proglottides, *T. dentata* (Gmelin), *T. armata* (Brera) to the hooklets.

The ova are very apt to hang about the anus and must frequently be carried thence, particularly at night-time, and finally might reach the alimentary canal. Moreover, long-continued retching may bring the worm itself into the stomach. As a matter of fact, very few of those who have a tapeworm become affected with cysticerci; but conversely von Graefe found that among thirteen patients with cysticercus in the eye five had tapeworms.

2. *Tænia mediocanellata* has only recently been recognised as a distinct kind of tapeworm, having before been confounded with *T. solium*. It is, however, of very frequent occurrence. The most striking distinction between the two species is in the head. This, in the *T. mediocanellata*, is flat at the summit, and has neither proboscis nor circle of hooks. It commonly, but not always, contains much pigment deposited round the four suckers. It is much broader than that of *solium*, so that it has a square form. According to Küchenmeister its water-vascular system is more simple in its arrangement, and he gave to this species the name of "mediocanellata," believing that it had a median water-vessel in addition to the two lateral ones which other tæniæ possess; but this was apparently a peculiarity of a malformed specimen which he had examined. It is often called the "unarmed" tapeworm, to distinguish it from *solium*, which is armed with its circle of hooks.

The strobila of this parasite also presents peculiarities which a medical man must be acquainted with, as it is often desirable that the species should be determined before the head can be obtained. It is considerably longer than *solium*. Leuckart says that it may reach four yards in length. It is also firmer in texture and flatter, and of a darker colour towards its distal extremity. Its joints are more numerous. The sexual organs attain their full development, as in *solium*, about the 450th joint from the head, but whereas in that species the uterus is full of ova at about the 200th join further on, in *mediocanellata* this is not the case before the 360th or 400th joint.

The ripe proglottides are larger, measuring three quarters of an inch length and a quarter to one third of an inch in breadth. They are more apt than those of *solium* to creep out of the patient's anus independently of defæcation. They also more generally rupture and discharge their ova while in the intestine, so that those which are passed per anum are shrivelled and empty.

But the most important peculiarity of the proglottides of *mediocanellata* is in the form of the uterus. This has from twenty-five to thirty branches on each side of its longitudinal channel (*solium* having only from seven to ten); they are necessarily packed much more closely, and they are simply forked over and over again, and terminate in round, club-shaped ends, not in the notched or leaf-like broad pouches which are seen in *solium*. The eggs are not globular, but slightly oval in form.

Another peculiarity of *T. mediocanellata* is its liability to malformations. Sometimes there are two or three genital pores in a single proglottis, each corresponding with a separate double sexual apparatus; sometimes the segmentation is incomplete; sometimes a supernumerary proglottis projects by the side of the continuous line of joints. But the most remarkable malformation of all is one in which there are two distinct chains, united in their whole length by one edge at an acute angle, so as to constitute a "double monster."

This tapeworm has only been recognised as a distinct species since Küchenmeister's original account of it was published in 1852. Bremser, indeed, had previously noticed that the tæniæ which he obtained from human beings in Vienna had no hooks, but he thought that they had dropped off in consequence of the age of the worms. Other observers adopted this view, although it obviously could not account for the fact that all the tapeworms in a particular district were unarmed. When Küchenmeister took up the subject, he found that not only were the hooks absent but also the proboscis which is possessed by the *T. solium*. He also drew attention to the other peculiarities that have already been mentioned.

For some time longer the source of *Tænia mediocanellata* remained undetermined. It had, however, been observed that the tapeworm which was known to be common in Abyssinia belonged to this species, and that the people there ate, not raw pork, but raw beef and mutton. It was also noticed that infants to whom raw beef grated fine was given under medical advice got a tapeworm, which at any rate in one instance was unarmed. Küchenmeister related the case of a patient who had harboured this parasite ever since a particular period when he had fed several times on raw beefsteaks. Putting these facts together, Leuckart came to the conclusion that the bladder-worm corresponding with this tænia probably occurred in horned cattle. He therefore in 1861 gave part of a stobila on two occasions to a young calf. Twenty-five days after taking the first portion of tapeworm the calf unexpectedly died. All the muscles (including the heart), the lymphatic glands, and other parts, were all full of minute round or oval vesicles, embedded in an opaque, whitish substance, which made them much more conspicuous objects than they would otherwise have been. They looked very like tubercles, and indeed the affection has sometimes since been spoken of as "acute cestode tuberculosis." The experiment has since been repeated by Leuckart and others, with the same result. It has also been shown that this form of bladder-worm has but a brief existence; if its host is allowed to remain alive, it perishes and calcifies in about eight months.

The frequency of *mediocanellata* as compared with *solium* varies, as might be expected, in different countries, according as the people live more largely on beef or on pork. It is stated that in Bavaria and the south of Würtemberg the armed tapeworm is never met with, whereas in North Germany that species is said to occur almost to the exclusion of the unarmed species. In England, Cobbold found that *Tænia solium* is more common among people of the lower class who eat much pork, whereas *T. mediocanellata* occurs in those who are better off, and can procure veal or beef. But he was gradually more and more decidedly led to the conclusion that the latter, on the whole, is the tapeworm which is most prevalent in this country.*

3. The only other species of tapeworm which is found in man often enough to require description in this work is now known by the name of *Bothriocephalus latus*, or the "broad tapeworm." It is said to have been originally distinguished by Felix Plator in the seventeenth century. It is

* A third species of the same genus, *T. nana*, was once discovered by Bilharz in large numbers in the intestine of a boy at Cairo. Others have been described in isolated cases, from Iceland, North America, and the West Indies. *T. echinococcus* has never been found as a strobila in man.

larger in every dimension than any other parasite which occurs in the human alimentary canal. It measures seventeen to twenty-six feet in length, and has from three to four thousand joints. These present the characteristic feature that their breadth exceeds their length. In the middle of the strobila they are nearly half an inch broad by one seventh of an inch in length. Towards the distal end they increase in length and diminish in breadth until at last their form is almost square. This tapeworm is peculiarly transparent looking. It has a longitudinal projecting ridge traversing its whole length. Its head is unarmed; it is club shaped, and has two deeply-grooved longitudinal suckers, one on each side, whence it takes its generic name of "pit-headed." The reproductive organs differ altogether in appearance from those of the tæniæ. The genital pore lies in the middle of each segment, opening upon its ventral surface. The uterus is an unbranched tube, which is bent on itself four or five times each way. When distended with ova its loops are flattened against one another, so that it resembles a rosette. The eggs are larger than those of the tæniæ; they measure 0·07 mm. in length, and are oval in form, with an operculum or lid at one end which rises to allow the escape of the embryo.

A peculiarity of this tapeworm is that its joints do not come away singly, but that portions of the strobila from two to four feet in length are expelled with the fæces.

The *Bothriocephalus latus* is almost, if not quite, limited to the inhabitants of certain countries of Europe. The locality for it which is best known is the western part of Switzerland; in Geneva one person in every four is said to harbour it. It also occurs in the north-west of Russia, in East Prussia, in Sweden (in one province of which the whole population is said to be infested with it without exception), in Poland, Holland, and Belgium. Leuckart speaks of its having been observed in persons living in London, and implies that this has sometimes been the case in those who could not have obtained the parasite from abroad; but this is doubtful. It is not impossible that *Tænia mediocanellata* may have been sometimes mistaken for it; for Leuckart remarks that muscular contraction may shorten the joints of that species, and anyone accustomed to the comparatively small *T. solium* might assume that a worm so much larger was the so-called *T. lata.*

The source from which this parasite enters the human body has not yet been certainly determined. The observation has long been made that the districts in which it is met with are low-lying regions, situated either near the sea, or at least near some large lake or river, and it has been suspected that the corresponding bladder-worm inhabits some kind of fish, or possibly a fresh-water mollusc. Salmon, trout, and bleak have especially been mentioned as likely to prove to be the resting-place of the immature form of the bothriocephalus. Such a view derives some support from the fact, first discovered by Schubart, but more fully made known by Knoch in 1862, that by keeping the ova several months in water, each of them gives out an embryo possessing the usual six hooks, but enclosed in a membrane which is completely covered with beautiful long delicate cilia. These enable it to keep up a constant rotatory movement, like that of a volvox. After four to six days, it escapes from the ciliated membrane and becomes free. Its further fate has as yet eluded observation. Knoch, indeed, thought that he had proved that the administration of proglottides of bothriocephalus to

puppies led to the direct development of the tapeworm in their intestine, but the validity of his experiments is disputed by Leuckart.*

Symptoms of tapeworms generally.—The effects of the presence of a cestoid worm in the human intestine are of a somewhat vague kind. Adults in robust health, and even healthy children, experience as a rule no discomfort whatever; it is only when proglottides or portions of their strobila are evacuated that a suspicion arises that they are otherwise than perfectly well. And, as Leuckart remarks, when this has once happened, the patient often begins for the first time to complain of pains and other symptoms, of which nothing had before been heard, although the parasite must have been present for several months. The sensations which are said to have been directly caused by it are described as an "uncomfortable feeling in the abdomen," "a colicky pain," a "gnawing pain at the epigastrium, especially when the stomach is empty or after certain kinds of food." Sometimes the patient is convinced that he can feel the movements of the worm; and, in reference to this, it is to be said that the sluggish contractions of the strobila outside the body give no idea of its activity while under the influence of the warmth of the intestine. Leuckart particularly speaks of the vigorous motions of its segmented body, of the continually varying play of its suckers, and of the bendings of its neck. It always hangs towards the lower part of the intestine, but sometimes it is bent on itself, or rolled up.

Leuckart mentions that in the dog he has sometimes observed injection of the mucous membrane, separation of the epithelium and even ulceration, produced by tapeworms; but it is doubtful whether such changes in the human intestine, even if they occur, would go further than the mere presence of the parasite towards explaining any symptoms that might be observed. Foulness of the breath, and irregular and craving appetite, constipation, or, very rarely, diarrhœa, are said to be caused by the existence of a tapeworm in the human subject.

Morbid sensations are also sometimes produced at distant parts,—itching of the anus, itching of the nose, so that the patient is always picking it, headache, giddiness, lassitude, a tendency to faintness. As might be expected, such symptoms are observed chiefly in persons of nervous, irritable temperament. Grinding of the teeth at night is another symptom, and patients have been known to have hysterical fits, epileptic fits, and even maniacal attacks, which have been cured by the expulsion of the worm. Dr Graves relates the case of a young lady, who was attacked with what were regarded as alarming symptoms of bronchitis. She had a dry, hollow cough, which was repeated every five or six seconds, night and day, whether she was asleep or awake. Bleeding, tartar emetic, blisters, antispasmodics, were tried in turn, but without result. Dr Graves and her medical attendant gave up the case in despair. At last she had a sudden attack of colic, for which an old servant of the family gave her a full dose of oil of turpentine with castor-oil. She passed a large piece of tapeworm, and from that moment every symptom of pulmonary irritation disappeared. One is not justified in prescribing anthelmintics indiscriminately for all cases of spasmodic nervous affections of which one happens to be unable to find the cause, but one should at least not forget to inquire as to the presence of worms in such cases. The bothriocephalus is said to give rise to more marked symptoms

* Another species, *B. cordatus*, has been more than once observed in human beings in Greenland, and a third, *B. cristatus*, twice in France.

than the tæniæ, but even it may be altogether latent. Bremser mentions the case of a Swiss, who had been eleven years away from his native country before he discovered that he was the bearer of this parasite.

Prophylaxis.—To prevent the development of tapeworms in the human intestine two distinct measures may be taken which, however, do not apply to the bothriocephalus, since the seat of the corresponding bladder-worm has as yet only been guessed at. In the first place, meat which is observed to contain cysticerci should not be eaten at all; and in the second place, all meat, should be subjected to such processes before being eaten, as will destroy any cysticerci that may chance to be present. Measly pork may often be easily recognised; the bladders are of considerable size and may be present in very large numbers. But it is remarkable that in the flesh of horned cattle cysticerci have never yet been seen, except after the experimental administration of proglottides of *T. mediocanellata* to the animals. The reason evidently is that an ox or heifer is a more cleanly feeder than a pig, and so its only chance of being infected with the cysticercus is by swallowing stray ova on the leaves of the plants which it eats or in the water which it drinks.

The second precaution against tapeworms consists in eating only meat which is thoroughly well cooked. The cysticercus cannot survive the temperature of boiling water. For more reasons than one, people should take especial care not to eat sausages which are underdone in the middle. Pork or ham which has been thoroughly smoked or salted may, it is said, be safely eaten, even though it may not have been cooked.

Several physicians in this country have recorded instances in which persons have been infected with a tapeworm who have been addicted to eating meat raw. But the most striking instance is that given by Kaschin, of the Bürater of the Baikal. These people live almost exclusively upon flesh, which they neither properly clean nor thoroughly cook, and which they eat from tables that are never washed, and that are used also for cutting up the meat. Even when stationed as Cossacks at Irkutsk, so that many of them had been away from their native country for years, they were infested with tapeworms to such an extent that in 130 autopsies only two bodies were found to be free from the presence of the parasite; often there were several, and once as many as fifteen, in the intestines of the same individual.

The curative treatment.—This consists in the administration of some substance which has the power of killing the creature without injuring its host. At the present time, no substance is used so largely for this purpose as the liquid extract (or "oil") of male fern. Its dose is generally said to be from fifteen to thirty minims, but at Guy's Hospital we have been in the habit of giving a drachm or a drachm and a half. Sir William Gull, many years ago, published in the 'Guy's Hospital Reports' (3rd series, vol. i, 1855) a series of 200 cases thus treated with much success. It never does any serious harm, but Cobbold speaks of it as causing irregular effects on the nervous system if its dose is too large. Another useful drug, derived from Abyssinia, is kousso, which consists of the dried flowers of the *Brayera anthelmintica;* from a quarter to half an ounce of this is infused in boiling water, and it is swallowed, powder and all. Oil of turpentine, again, is often effectual, of which from half an ounce to two ounces may be taken for this purpose; a single large dose is less apt than repeated small doses to cause the strangury which

sometimes results from its administration. A decoction of the bark of the pomegranate root is another valuable anthelmintic; the direction is that three or four doses of from one to two ounces each should be given at intervals of about half an hour; it often causes faintness and giddiness.

Whatever medicine may be chosen, it is advisable for the patient to fast for several hours before taking it, although Cobbold objected to this. The intention is that, the alimentary canal being empty, the drug may with more certainty come into contact with the tapeworm. And for the same reason a dose of castor-oil is sometimes given three or four hours before the anthelmintic.

The administration of one of the remedies above mentioned almost always brings away a large portion of the tapeworm, if there be a fully developed tapeworm in the patient's intestine. But, unfortunately, the strobila very commonly breaks at the neck. The head then remains behind; and as it still retains its vitality, it at once begins to form fresh segments. Now, if the parasite belong to either species of tænia, it is remarkable that after such an accident, an interval of three months (Cobbold says thirteen weeks) is almost invariably found to elapse before proglottides again begin to be passed. The writer has repeatedly known this to occur on almost the very day which had been predicted. This period of three months corresponds exactly with the length of time which is required for the full development of the tapeworm from a cysticercus; it therefore follows not only that under the influence of anthelmintics the line of fracture is constantly at one part of the worm, but that it is quite close to the head. It no doubt sometimes happens that the creature breaks in the middle, particularly if the dose of the anthelmintic is inadequate. But this, at any rate, may be said,—that if a portion of tapeworm be brought away, in which part of the narrow neck is recognised, and if the patient should in much less than three months begin again to pass proglottides per anum, it is certain that more than one tænia is present.

The patient must always be told to look very carefully in his evacuations for the head, the appearance of which should be described to him. An enthusiastic practitioner may himself search for it. Cobbold recommends that the whole mass of fæces should be passed through a sieve. If the head be not discovered, the patient may either wait for three months to learn whether the treatment has been effectual, or he may take a second dose. One would have thought it doubtful whether drugs would act satisfactorily upon a tapeworm of which nothing but the head is left; and it would be obviously very difficult to obtain evidence as to the frequency with which a cure is effected under such circumstances. But Cobbold relates one instance in which, having brought away almost the whole of a tapeworm with one dose of extract of male fern, he gave another dose the next day and actually succeeded in finding the head with its four suckers in the patient's fæces.

The remaining species of intestinal worms belong to the Nematoidea.*

1. The round-worm (*Ascaris lumbricoides*), as its specific name implies, is somewhat like the common earth-worm (*lumbricus*). When alive it is of a

* Sometimes spelt "Nematoda." The word is derived regularly from νῆμα, a thread, and εἶδος, appearance. The group nearly corresponds with the Cœlelminthia, *vers entozoaires cavitaires* of Cuvier.

reddish-brown colour with a tinge of yellow, but after its death this colour slightly fades, and it becomes greyish. It has a disagreeable smell, which cannot be removed by washing, and which, according to Leuckart, is due to an odorous principle having its seat in the deeper muscular layers of the body.

Dr Bastian and some others have suffered from irritation of the eyes, sneezing, and other symptoms like those of hay-catarrh from dissecting this worm.

The female is fifteen inches long; the male, which is comparatively seldom met with, measures only ten inches; its circumference is also much less than that of the female. They are both cylindrical in form, tapering at each end, but rather more gradually towards the head than the tail.

The life-history of this parasite has not yet been completely ascertained. The female discharges ova which certainly do not undergo any development while they are in the human body. After their escape with the fæces, however, an embryo slowly appears in each egg if it be kept in water or in moist earth. Davaine and others formerly supposed that the ova were swallowed in this condition either in drinking-water or upon uncooked vegetables or fruit, and that their shells having been removed in the stomach, the embryos gradually became developed into full-grown worms. But experiments made for the purpose of confirming this hypothesis have uniformly failed; several German investigators have deliberataly swallowed large numbers of ova and have given them to children, but no specimen of the ascaris has hitherto been obtained in this way. Another possibility is that the embryos escape from the ova and enjoy an independent existence for a time before they enter the human body. Thus Dr Paterson, of Leith, is quoted by Aitken as having observed that certain families who drank the water of a particular well were very subject to the parasite, whereas towards the other end of the same street families who drank the pure water supplied to the town of Edinburgh were free from it. The well-water came from a dirty pond in the vicinity, and in it numerous minute vermiform animalculæ existed, which perhaps were larval ascarides. But Leuckart lays stress on the fact that the embryos of *Ascaris lumbricoides* show little or no tendency to escape from the ova, and that their organisation is not like that of embryos which are destined to maintain an independent existence. He therefore thinks it most probable that the ova are swallowed by some intermediate host—perhaps a worm or the larva of some insect—and that within the body of this animal the embryos pass through such further changes as may prepare them to undergo their complete development on being afterwards transferred to the human digestive canal.

The *Ascaris lumbricoides* appears to infest the human intestine in all parts of the world, but certain classes of people are much more liable to it than others. It is rare in infants under a year old, although Leuckart refers to one instance in which it occurred in a child of eleven weeks. Children between three and ten years of age afford the most numerous specimens of the round-worm. It is more common in rural districts than in towns, and particularly in low and damp localities. It is met with more often in the autumn than at any other season; this Leuckart connects with the hypothesis that eating summer fruits has in some way a share in introducing it into the human body. Persons who are poor and dirty are more subject to it than those in better circumstances. In the insane it is very

often found. Vix found that among thirty lunatics of dirty habits in the Hofheim Asylum, there was not one who was free from this parasite. In the Southern States of America, the West Indian islands, Cayenne, and Brazil, the negroes at all ages are with scarcely an exception infested with *Ascaris lumbricoides*. It is also much more common in some parts of Europe than in others, particularly so in Finland and Holland.

The *Ascaris lumbricoides* inhabits the small intestine. It may be solitary, or there may be two, three, or any number of them. When numerous they often cohere together in knots, and they have sometimes been found filling almost the whole of the intestines. Children have been known to pass some hundreds of them in the course of a few weeks. Cruveilhier found more than a thousand in the intestine of an idiot.

It is considered probable that each individual worm remains only a few months within the body of its host. If they pass down into the large intestine they are voided from the anus, either alone or with the fæces. If they make their way upwards into the stomach, they are generally vomited. Sometimes, however, one is discharged through the nose; and it has even been known to enter the larynx, and cause death by suffocation. A curious point, to which Dr Cobbold has especially drawn attention, is that the *Ascaris lumbricoides* is very apt to insinuate itself into any kind of small ring that may be swallowed by its host, such as the eye of a lady's dress or the shank of a button. A single worm has been found with two buttons attached to it. This peculiarity goes far towards explaining the fact that the parasite sometimes makes its way into the bile-duct or gall-bladder, setting up jaundice, or even suppuration in the liver. In other cases it has escaped into the peritoneal cavity through the floor of an intestinal ulcer, supposed to have arisen independently. And in yet others it has been found within the cavity of an abscess generally situated either at the umbilicus or in the groin. There has been much discussion whether or not the inflammation has in such instances been originally set up by the presence of the worm; a strong point in favour of this view is that after its escape the abscess has generally been found to heal, and thus to end much more favourably than an ordinary fistulous opening from the bowel.

The *symptoms* produced by these worms vary according to the number of them which are present and the irritability of their host. It is only in a very delicate subject that a single *Ascaris lumbricoides*, or even two or three, would cause any appreciable discomfort. When symptoms do arise, they are generally such as indicate irritation of the intestinal mucous membrane, pain in the abdomen (especially in the umbilical region), nausea, foulness of breath, irregularity of appetite, tumidity of the abdomen, and the presence of mucus in the stools. It is true that in the bodies of those who have died from other causes, and in whom worms are found, the intestine does not generally present any morbid appearances that can be attributed to their presence; but Barthez and Rilliet say that they have seen the mucous membrane reddened by vascular injection at points occupied by several round-worms, and not anywhere else.

More serious cases have been recorded of fatal ileus caused by masses of the parasites rolled up together; but it is more than doubtful whether this ever happens. It is perhaps more probable that the presence of worms may lead to invagination.

In certain patients the *Ascaris lumbricoides* has given rise to reflex symptoms similar to those which have been described as effects of the presence

of tapeworms : dilatation of the pupils, swelling of the eyelids, squinting, irritation of the nostrils, grinding of the teeth during sleep.

Diagnosis.—There are two conditions under which one has to deal with this parasite :—1, where a patient, generally a child, presents some of the symptoms just enumerated, which, in the absence of any more obvious cause, are supposed to be possibly due to its presence ; and, 2, where one ascaris is said to have been vomited or passed per anum, and there is a question whether there are still others in the intestine. In the second class of cases one has sometimes to bear in mind that impostors have been known to bring earthworms with them, which they pretend to have passed from the bowels. The true lumbricus, however, is readily distinguished from an ascaris ; it is much redder, it tapers less at its extremities, and it has rows of small bristles, which aid it in locomotion ; its mouth is a short fissure on the under surface of its rounded head, whereas the mouth of the *Ascaris lumbricoides* is a triangular aperture at the more pointed end of the animal, surrounded by three tubercles or lips.

When the presence of this parasite in the intestines is suspected, from whatever cause, a common practice is to give a dose of medicine, on the chance that it may bring away an ascaris. But Leuckart and others have pointed out that the question may very readily be answered by a microscopical examination of the patient's fæces, which, if the worm is there, are sure to be full of its ova. It has been calculated by Eschricht that in the female ascaris there are at one time about sixty millions of eggs, and if these were a year in being discharged from its genital passages, the patient's evacuations would contain 160,000 of them every day. No wonder, therefore, that a single microscopical specimen often places the presence of the parasite beyond dispute. The ova are elliptical in form, measuring $\frac{1}{340}$th of an inch by $\frac{1}{440}$th of an inch ; they are of a dirty brownish colour and nodulated on the surface, from the presence of a thick layer of an albuminous substance deposited outside their proper shell. In illustration of the value of this method of diagnosis Dr Ransom gives (in his article on the entozoa in 'Reynolds' System of Medicine,' vol. iii, p. 197) the case of a child who was admitted into hospital for abdominal pains and disordered digestion, and because she had passed two round-worms previously. The evacuations contained the eggs of the parasite. Medicines on several occasions brought away one or more specimens of the ascaris, and her symptoms entirely disappeared. But ova were still detected in the stools, and therefore she was kept under treatment three months and a half longer, until seventeen worms in all had been passed. No more of the ova could then be discovered, and she was accordingly sent out of the hospital.

Treatment.—The drug which appears to possess more power than any other in effecting the expulsiou of the *Ascaris lumbricoides* is santonine. Its dose, for an adult, is from three to six grains twice daily, and for a child one to three grains. While it is being taken, an occasional purgative should also be prescribed. An inconvenience sometimes produced by santonine is a curious temporary disturbance of vision, objects appearing of a yellow, green or blue colour. The urine may also be reddened, but that is of no consequence. Cobbold says that it sometimes produces tenesmus, spasms, and even hæmorrhage from the bowels, so that it should not be too long continued. Dr Ransom speaks of *Dolichos pruriens* and oil of turpentine as being also worthy of trial.

As to preventing the ascaris from entering the body, all that can be said is that one should be careful to drink only pure water and to have all solid food thoroughly cooked.

The allied species *Ascaris mystax* has occasionally been observed in the human intestine both by German and British helminthologists.

The *Oxyuris vermicularis*, or "thread-worm," *Germ.* Madenwurm, is very much smaller than the round-worm just described. It was formerly called *Ascaris vermicularis* and in England this name is not yet quite obsolete, for thread-worms are still commonly called "ascarides." There is not much risk of confusion in this use of the term, for medical men never have occasion to speak of the *A. lumbricoides* as present in large numbers in a living patient, but it is incorrect according to modern zoological classification, and is liable to mislead the student. Etymologically, however, the word ascaris (from ἀσκαρίζειν, to leap) is applicable rather to the thread worm, which performs brisk movements, than to the comparatively sluggish round-worm.

The oxyuris may best be compared to a small piece of white thread. The female measures four tenths of an inch in length, the male one sixth of an inch. The latter is much less often seen and was formerly supposed to be very rare, but now it is supposed that there is about one male to every nine females. They taper towards the tail, as their generic name denotes.

Thread-worms occur only in the large intestine. They derive their nourishment from the fæcal matter of their host; and its yellow colour can plainly be recognised in the interior of their bodies. They are often present in vast numbers, and are found either singly in the mucus lining the interior of the bowel, or matted together with this mucus into little balls.

The eggs of oxyuris are oval in form, and flattened on one side, with a smooth surface. They measure $\frac{1}{1100}$th of an inch by $\frac{1}{490}$th of an inch. Unlike those of the *Ascaris lumbricoides*, they contain embryos at the time of their liberation from the parent worm. It might therefore be supposed that no impediment existed to the multiplication of the oxyuris within the human intestine for an indefinite period. But Leuckart and other modern observers believe that the ova are incapable of undergoing development until they have passed into the external world and been swallowed by the same or by another individual. One is at first startled when one is told that every single thread-worm in a child's intestine represents an ovum which the child must have taken into its mouth. But, as Leuckart points out, this theory is supported by the analogy of all other parasitic worms, none of which are capable of reproducing themselves indefinitely *in situ*. Indeed, these creatures produce such immense quantities of ova that there would be no limit to their numbers, were not their development subject to some such conditions as are suggested by the theory in question. Leuckart further observes that one never finds *young* oxyurides in numbers bearing any proportion to those of the ova, whereas, if they arose directly out of them, they ought to be far more abundant. He tested the point, as far as he could, by swallowing a few ova and giving some to those of his pupils who volunteered to share in the experiment. At the end of the second week, three out of the four individuals experimented on began to pass thread-worms.

This question is one of considerable importance in reference to the measures which should be adopted for preventing the occurrence of oxyuris. This parasite creeps out of the rectum of its host, especially at night; it wanders among the folds of the anus, and in women often passes into the

vagina. It is therefore quite conceivable that it might pass or be carried to the anus of another individual sleeping with the one from whence it came. Küchenmeister supposed that this was the way in which the worm obtained access to the human body; he thought that a single female oxyuris passing from one bedfellow to another might make the latter the victim of this parasite for the rest of his days.

Modern helminthologists rather explain the emigration of thread-worms from the rectum as follows. The worms and their ova often become adherent to the skin and hair in the neighbourhood of the anus; they dry up, and ultimately break down into a fine dust, containing enormous numbers of ova still capable of springing into life if brought under suitable conditions. Thus every opportunity is afforded for what may be termed "self-reinfection."

Again, every fæcal evacuation of a person infested with thread-worms probably contains hundreds of thousands of their ova. They must be carried into drinking-water, taken up by flies, deposited upon vegetables and fruit, and so in a thousand ways gain access to the human alimentary canal.

The *symptoms* produced by thread-worms are not like those caused by other entozoa, being chiefly the effects of the irritation which they produce by creeping about the anus and genital organs of their host. Chief among them is a sense of heat and tingling or itching at the fundament. This comes on at a particular time, generally about 9 or 10 p.m., when the patient is in bed, but sometimes before he has retired to rest. Marchand quotes the account which a man gave of his own sufferings, as follows:—"Every evening about 5 or 6 o'clock, when I first feel the worms, I become pale and troubled, and sometimes I have even shivered; my companions often notice it; I am restless and obliged to walk about; even if I am at a place of entertainment, I leave instantly and hasten to employ a cold enema; this does not always give me relief and I am then in torture; I tear my perinæum and scrotum. I am obliged to micturate every instant." Irritability of bladder is well known to be sometimes caused by the presence of thread-worms, and there is reason to believe that they sometimes excite priapism or nymphomania, and thus indirectly lead to the practice of masturbation. Another symptom may be tenesmus; and the fæces may contain a large excess of mucus. Cruveilhier recorded the case of a child who was awakened every night at the same hour by an agonising pain in the anal region, so that he screamed and writhed about in bed. The periodicity of the attacks led to the administration of quinine, but with no success, until the part was looked at, when the cause was at once discovered.

The *treatment* is a less easy matter than might be expected; and there is still some uncertainty as to the best way of ridding a patient of thread-worms. Until lately it has been taught that the rectum and sigmoid flexure were the parts of the intestine chiefly infested by this parasite; it was known that it might be found as high as the cæcum, but this was regarded as exceptional. The older writers, therefore, recommended enemata for their removal. Sir Thomas Watson says that he has often relieved patients of thread-worms by prescribing infusion of quassia as an injection. Lime-water, solutions of chloride of sodium, of perchloride of iron, and many other substances have been recommended for the same purpose. The rule has generally been that an enema should be given every third or fourth day for two or three weeks.

Of late, however, the opinion has been gaining ground that the oxyuris occurs in the cæcum and in the upper part of the colon more generally than

had been supposed. Cobbold believes that the cæcum is its head-quarters; he therefore recommends active saline cathartics, repeated for several days in succession, and large draughts of infusion of gentian; also an Indian remedy, *Aristolochia bracteata*. He mentions that the introduction of a little mercurial ointment within the verge of the anus, as the patient retires to rest, will effectually prevent thread-worms from creeping out of the rectum, but this plan must require some caution, lest salivation should follow. Relief is sometimes afforded to the itching by the application of oil or of dilute red oxide ointment.

Children, particularly about five or six years old, are much more commonly infested with thread-worms than adults, but the former are often very easily freed from their presence, whereas in the latter this parasite is apt to resist remedial measures with great obstinacy.

If the modern view of the life-history of the oxyuris be correct, it is most important that scrupulous cleanliness should be maintained in all the surroundings of a person infested with it. But we cannot at present hope for such success in this direction as would justify Dr Ransom's remark, that a person who adopted the requisite precautions against reinfection from himself or others would probably get well in a few weeks without treatment by drugs.

The *Trichocephalus dispar* (*Germ.* Peitschenwurm), another nematode worm, has its seat in the cæcum. It is remarkable for its very long and thread-like neck, which forms about two thirds of its whole length of one and a half to two inches. This parasite appears to give rise to no symptoms; and it has scarcely ever been discovered in the evacuations. It therefore possesses no clinical interest. But, as its ova may be found in the fæces, it is well to mention that they are bluntly spindle-shaped, with transparent ends, and that they measure 0·023 mm. in breadth by 0·051 mm. in length.

The *Sclerostomum duodenale** is of far greater importance; except, indeed, for the circumstance that it is not found in this country. It is occasionally met with in Italy, where it was discovered in 1838 at Milan by Dubini; and occurs very commonly in Egypt and in Brazil. At a meeting of the Pathological Society in 1867 some specimens of it from the latter country were exhibited by Dr Hermann Weber. (See his paper, with figures, 'Path. Trans.,' xviii, p. 274.)

In Cairo, Bilharz found it in almost every dead body which he examined. It inhabits chiefly the jejunum, generally lying between the valvulæ conniventes, with its mouth firmly fixed in the mucous membrane by means of its four conical chitinous teeth. The female occurs in larger numbers than the male.

The sclerostomum is a small round worm with its head bent nearly at right angles. The male measures nearly half an inch in length; the female seven tenths of an inch. It may be present in enormous numbers, as many as 1250 having been counted in a single patient. It feeds, not upon the intestinal juices, like other worms, but upon blood, which fills its digestive canal, and gives its body a red colour. It fixes itself firmly by means of two pairs of teeth (of which the ventral pair is the larger) into the mucous membrane of the duodenum or jejunum; and within its mouth there are two moveable blades, which doubtless serve to incise the tissues.

* Also known as *Ancylostomum duodenale* (Dubini), as *Strongylus* or *Dochmius duodenalis* (Leuckart, Diesing), and as *Str. quadridentatus* (von Siebold).

The spot to which it is attached is indicated by an ecchymosis; and Leuckart thinks that it shifts its position from time to time, and that the punctures which it leaves may then go on bleeding. The cavity of the bowel is sometimes found full of blood after the patient's death, although hæmorrhage per anum seldom, if ever, occurs during life. The body of the worm commonly hangs free within the gut, protected more or less by the ridges of the mucous membrane; but sometimes it is rolled up in a hollow space in immediate contact with the muscular coat.

The female sclerostomum throws off numerous eggs, which are oval bodies, with a thin, transparent shell, of nearly the same size as those of the oxyuris, but less elongated, measuring only $\frac{1}{20}$ mm. in length. They also differ in having no operculum, and in their yolk being undivided or only just segmented at the time of their expulsion in the fæces, whereas the eggs of the oxyuris already contain embryos. The life-history of the sclerostomum has not been directly traced, but it is believed to be the same as that of the allied *Dochmius trigonocephalus* of the dog. If this be correct, the ova become hatched when they pass into mud or water, and produce slender worms which exhibit active movements. These require no intermediate host, but develop into sexually mature animals when they are swallowed and reach the human alimentary canal. It is not surprising that the parasite should be met with chiefly in hot climates, where men are often compelled to drink water from dirty pools exposed to contamination in every way.

As hundreds and even thousands of these parasites are sometimes present in the same individual, it is not surprising that they should give rise to profound anæmia.

It was Griesinger who showed, in 1854, that the form of anæmia known as Egyptian chlorosis was due to the presence of this parasite ('Arch. f. phys. Heilkunde,' xiii, 557). Wucherer, of Bahia, found in 1866 that the parasite gives rise to a similar complaint in Brazil. It occurs in the Comoro Islands, and Dr Strachan has reported cases from Jamaica ('Brit. Med. Journ.,' June 27th, 1885). One would have expected that the disease would sometimes have been seen in England in persons recently arrived from Egypt, just as a somewhat analogous affection, due to the Bilharzia, is often brought to this country from South Africa.

The sclerostomum was the cause of severe epidemic anæmia among the workmen in the St Gotthard Tunnel in 1880 (see a paper by Dr Bugnion 'Brit. Med. Journ.,' March, 1881, p. 882). One of these cases came under the care of Prof. Bäumler, of Freiburg.

The resulting anæmia may prove fatal in a few weeks, or it may run on for years, unless death occurs by dysentery. At first the patient appears well nourished, and even fat, but at length he becomes wasted and dropsical. The only special symptoms mentioned are disorders of digestion, and a cutting pain in the abdomen.

The other symptoms of this "tropical anæmia" appear to be identical with those of other forms of anæmia. They may last for years, until the patient is at length carried off by diarrhœa or pneumonia, or some other accidental malady. Much more rarely the chlorosis proves directly fatal after causing dropsy.

Microscopically, the eggs may be recognised by diluting the fæces with water, stirring, and allowing them to settle.

The medicines which have been suggested for the *treatment* of this disease are chiefly oil of male fern, santonine, oil of turpentine, assafœtida, aloes, and iron, but Dr Weber says that none of them have proved to be perma-

nently successful. If the patient can be removed to another climate, and placed under favourable conditions, recovery may take place.

Strongylus (*Eustrongylus*) *gigas* is a very large nematoid worm inhabiting the pelvis of the kidney in certain carnivora and other mammals. It is not very uncommon in dogs and wolves, in the coatimundi (*Nasua*), racoon, otter, and seal, and has also, it is said, been met with in horses and oxen. It is excessively rare in man : most recorded cases are spurious, and refer to ascaris or to fibrinous clots passed per urethram. Küchenmeister quotes fourteen cases, but only those of Grotius (1595), Ruysch, and Blasius, and a more recent one of Moublet, seem to be authentic. Diesing can only adduce three clear cases. A specimen in the Hunterian Museum is said to be taken from the human kidney. This species is the largest nematode entozoon known : the male grows to a length of ten inches, and the female to thrice that length. One huge strongylus of this species was found free in the abdominal cavity of one of the Esquimaux dogs which McClintock took on his Arctic expedition in search of Sir John Franklin. It was sent from Greenland to Steenstrup at Copenhagen, and by him given to Leuckart. The account of the strongylus given by this anatomist is based on that and only two other specimens, one from a coati, and the other from an American mink (*Mustela vison*).

An allied species (*Strongylus armatus*) produces aneurysms in blood-vessels after it has wandered from the cæcum or colon, in horses, asses, &c.

*Trichina spiralis.**—This nematode is truly an intestinal worm, although until recently it was only known as being now and then found unexpectedly in the muscles of the human body. How it found its way into the muscles was for a long time a puzzle which exercised the minds of the ablest zoologists. But, as we shall presently see, its life-history is not altogether unlike that of the other nematodes which infest the human intestine. The principal difference is that, instead of its ova being discharged through the rectum of the host, they develop within the parent worm into young trichinæ, which, as soon as they are born, begin an active migration among the tissues of their host.

Tiedemann, in the year 1822, was the earliest observer who took note of the presence of a number of white stony concretions in the human muscles, but he did not describe them further, and Leuckart doubts whether these were trichina capsules. In this country Hilton was the first, in 1832, to record the fact that he had met with them.† He could not determine their precise nature, but thought that they were probably very small cysticerci. Next Mr James Paget, then a student of St Bartholomew's, independently described them. Two years later Owen showed that they were hollow capsules, and that each of them had coiled up within it a minute nematoid worm, to which he gave the name of *Trichina spiralis*, and fully described its anatomy.

The capsules themselves are just visible to the naked eye ; they measure $\frac{1}{78}$th inch in length, and $\frac{1}{130}$th inch in breadth. They are perhaps best described as lemon-shaped. Their long diameter is always in a line with the muscular fibres among which they lie. They occur in all

* Hair-worm—*θρίξ, τρίχος*, a hair.

† 'London Medical Gazette,' 1833, p. 605. Owen's paper is in the 'Zool. Trans.,' vol. i, p. 315. The oldest preparation of trichinæ is one of the sterno-hyoid muscle in the Guy's museum No. 1361[30]. It was put up by Mr H. Peacock in 1828.

striped muscles, even in the tensor tympani, but are most numerous in the diaphragm and the muscles of the trunk. The heart is scarcely ever affected, and in non-striated muscles they are never met with, so that the œsophagus contains them only in its upper part. They feel gritty when touched with a knife; this is due to the deposition of calcareous matter, which sometimes renders them altogether opaque, but is usually present only in their extremities, leaving their centres transparent, so that under the microscope the little worm in the interior of each of them is at once visible. There is an excellent description of the trichina with figures by Dr Bristowe and the late Mr Rainey, in the 'Pathological Transactions' for 1854; and good plates are given in Cobbold's 'Entozoa.'

Not only rats and swine (the usual bearers of the disease to man), but cats and dogs, hedgehogs and moles, and herbivorous animals as horses, calves, rabbits, guinea-pigs, may be infected. The muscles of birds remain free from invasion though the worms multiply in their intestines, and trichinæ do not breed in cold-blooded animals.

The earliest experiments which throw any light upon the way in which this remarkable entozoon gains access to the human body were those of Herbst, who in 1851 showed that when the flesh of animals containing trichinæ was given to other animals their flesh in turn became infested with the parasite. He did not trace the intermediate steps by which this result is brought about, and afterwards observers were for a time led astray by Küchenmeister's hypothesis that the trichina was a stage in the development of *Trichocephalus dispar*. In the spring of 1860, Virchow and Leuckart showed that this was not the case.* They fed animals with trichinous meat, and found that the worms at once escaped from their capsules and developed into sexually mature entozoa of a kind that had never before been recognised.

The experiment has since been repeated by many observers, and with uniform results.

For example, an animal to which trichinous flesh has been given is killed at the end of forty-eight hours; the mucus lining its alimentary canal is found to contain numbers of minute living worms already sexually mature. They are not visible to the naked eye, but they are easily recognised under the microscope. The majority of them are females; these measure one twelfth to one ninth of an inch in length (2—3 mm.). The males are smaller, being only one eighteenth to one fourteenth of an inch long (1·2—1·6 mm.); they are further distinguished from the females by having two conical projections from the caudal extremity. If the animal be left until the sixth day before it is killed the female worms contain free embryos, which are bent and closely packed side by side in the uterus. These may even be watched under the microscope as they become extruded from the vagina and afterwards move about the field. It is estimated that at least 150 young worms are produced by each female trichina.

It is therefore clear that from the sixth or seventh day after the ingestion of trichinous flesh by man or any other animal, living trichina-embryos are poured in enormous numbers into the alimentary canal. These at once begin to bore their way through the coats of the intestine. They enter the peritoneal and other serous cavities, the lymph-glands, the viscera, and

* Leuckart, in his excellent account of the subject ('Die Menschlichen Parasiten,' vol. ii p. 525), recalls the early observation of trichina in a case of "pneumonia and pericarditis with muscular rheumatism," *i. e.* of trichiniasis, at Bristol, by Dr Wood, in 1835 ('Lond. Med. Gaz,' p. 190).

above all the muscles. Observers are not yet altogether agreed as to the way in which the trichina-embryos reach distant parts of the body. Leuckart and Virchow suppose that they make their way along the connective-tissue spaces. But the rapid migration of the parasite to the most distant parts of the body is strong evidence that the embryos enter the blood-stream and ultimately reach the tissues through the walls of the systemic capillaries. However, this may be, it is certain that the voluntary muscles are the only parts in which they find the conditions necessary for their further development. If they enter other tissues they either perish or migrate again until they reach their proper seat. Even when they have reached the muscles they seem at first to move on in the course of the fibres, for they are found in larger numbers towards their tendinous insertions than elsewhere, as though these formed obstacles arresting their further progress. They have sometimes been distinctly seen within the sarcolemma of the primitive fibres, and Leuckart asserts that this is always their position, but others maintain that they more commonly lie between the fibres. However this may be, it is certain that they rapidly increase in size, and from being structureless and undistinguishable from minute filariæ of different species, they gradually acquire a distinct alimentary canal, and even rudimentary sexual organs. At this time they roll themselves up, and round each of them a capsule is developed. This is first to be recognised about the fourth week after their immigration. It consists originally of a nucleated transparent material, the product of the irritation of the tissues which their presence causes. After a time calcification begins in it. Dr Thudichum says that in rabbits he has seen the capsules perfectly opaque within ten weeks. But in the human subject a high degree of calcification does not occur in less than a year. Rupprecht found capsules still transparent in the muscles of a man who had had an attack of trichiniasis two years before.

Calcification of the capsule does not necessarily interfere with the life of the trichina within. The parasite remains quiescent, waiting for the death of its host, which would call forth at any moment its latent forces. But in course of time it may itself perish; it then becomes converted into an almost structureless mass, which under slight pressure breaks up into fragments.

The number of trichinæ which may be contained in the muscles of the human body is enormous. From data obtained in experiments on animals it has been estimated at from twenty to thirty millions. This, however, applies to cases which would perhaps always terminate fatally before the worms would have time to become encapsuled.

The entrance of trichina-embryos into a muscle produces certain changes in its fibres which have yet to be mentioned. They lose their striation, become brittle and homogeneous, and show numerous minute fissures. To the naked eye the muscular substance appears of a pale reddish-grey colour.

Trichiniasis.—At the very time when Leuckart and Virchow were working out experimentally the life-history of the trichina, Zenker had just observed a case in which this parasite caused a fatal illness in the human subject. On January 12th, 1860, a girl was admitted into the Dresden Hospital suffering from what at first appeared to be fever. She died, and on *post-mortem* examination the characteristic lesions of fever were all absent, whereas the muscles were full of living trichinæ as yet unencapsuled. The girl had been in the service of a butcher, who had killed a pig about a week before her illness commenced. She had been concerned in making sausages of the pork, and had very likely eaten some of it in an uncooked state. The sausages and a

ham from the pig were examined and found to contain trichinæ. It was also ascertained that the butcher and two other persons had been taken ill about the same time, but had recovered.

Symptoms.—Since the publication of Zenker's case several others have been recorded, so that the clinical aspect of trichiniasis is now well known. At first it is undistinguishable from those of other febrile diseases. The patient complains of loss of appetite, sleeplessness, and a sense of extreme lassitude and depression, and it may be of nausea and vomiting, but at the end of a week, or a little later, the arms and legs begin to be stiff and painful. The elbows and knees become flexed, and great pain is produced by any attempt to straighten them. After a time the limbs are sometimes rigidly extended, and the body is as though affected with opisthotonos. The muscles are tender to the touch; when grasped they feel hard and swollen, and as if they were distending the fasciæ in which they are enclosed. The jaws are often closed for several weeks, after which the muscles may become suddenly relaxed again, with an audible crack. Movements of the eyes are painful, no doubt from the presence of trichinæ in the recti and obliqui, but the power of accommodation is lost at the same time, and this is less easy of explanation. The breathing becomes shallow and hurried from implication of the thoracic muscles; and coughing, sneezing, and yawning may be almost impossible.

About the end of the second week the eyelids are observed to be œdematous, and sometimes the rest of the face and even the neck. Afterwards the legs and the parts round the joints become swollen.

The fever is not generally high in trichiniasis; the temperature seldom rises above 102° Fahr. There is often profuse sweating, and a miliary eruption may develop itself. The pulse is sometimes very rapid. The tongue is red, slightly furred, and rather dry. The bowels are sometimes constipated; but diarrhœa may occur, especially in severe cases.

Fatal cases usually terminate in the fourth or fifth week, but sometimes much earlier. The immediate cause of death may be exhaustion, or pneumonia, or ulceration of the colon. If the patient should recover, convalescence is tardy, lasting three or four months.

Diagnosis.—In well-marked cases this does not appear to be difficult the symptoms, taken together, are unlike those of any other disease. It has several times been established by the microscopical examination of a minute portion of muscle removed during the patient's life. Another way of verifying it is to search the fæces for adult worms. Rupprecht found numbers of them in the stools of patients to whom large doses of calomel had been given.

Very often, however, the discovery of the disease is facilitated by the fact that it occurs epidemically. Thus, at Plauen, in 1862, thirty persons were attacked about the same time. At Hettstädt, near the Hartz Mountains, four separate outbreaks of trichiniasis occurred between September, 1861, and March, 1864; in the most important of these 158 persons were attacked, of whom twenty-eight died. Other epidemics have been observed at Stassfurt, Dessau, Leipzig, and elsewhere. Of all of these full details may be found in a paper by Dr Thudichum which appeared in 1864 in the seventh Report of the Medical Officer to the Privy Council.

In England the only cases of trichiniasis which have been detected during life, or attended with symptoms (since Wood's in 1835) are those recorded in 1871 by Mr Dickinson, of Workington, in Cumberland. The

patients were a farmer's wife, his daughter, and a serving man. They had all been eating sausages and pork from one of the farmer's home-fed pigs, the flesh of which was subsequently found by Dr Cobbold to be full of trichinæ. It has been supposed that cases in which symptoms are produced by the migration of trichina-embryos into the muscles do really occur from time to time in this country, but are overlooked.* Indeed the fact that encapsuled trichinæ are now and then found in the bodies of those who have died of other complaints, shows that the conditions which are necessary for the entrance of this parasite into the human body are not entirely absent from English life. But there is reason to believe that even in this form the parasite is rarely met with here as compared with its frequency in Germany. In Dresden, Zenker detected it in four out of 136 *post-mortem* examinations; and in Berlin Virchow found it six times in the course of a single year. These observers mention that often a very small number of trichinæ only were present, so that they might easily have been overlooked. Probably, in such cases the symptoms, if any, would be very slight, merely such as might be attributed to a "rheumatic affection." It is tolerably certain that, as a severe or fatal disease, trichiniasis has not yet occurred in any London hospital—at least since the publication of Zenker's case. Even on the Continent the disease is far more common in Northern Germany than anywhere else, and most common of all in Saxony. In districts where pork is not eaten raw, it is rare.

The severity of a case of trichiniasis appears to depend upon the number of embryos which penetrate into the muscles. Thus the most important guide to *prognosis* is believed to be the state of the patient's limbs, as regards mobility and pain on movement.

Treatment.—The first indication is to expel as many of the trichinæ as possible from the alimentary canal. For this purpose castor-oil is recommended, or calomel in twenty-grain doses, repeated at intervals. Experience is said to have shown that the latter medicine gives relief to the symptoms. Friedreich at one time proposed the picrate of potash; he gave it in one case which terminated in recovery; but live trichinæ were afterwards found in this patient's muscles. Glycerine is said to kill the trichinæ when directly applied (probably by abstracting water), and it has therefore been administered in cases of trichiniasis in the United States, and, it is reported, with success.

The *prevention* of trichiniasis resolves itself into two distinct questions. First, how can animals intended for human food be kept free from the parasite? The answer to this is that they must be kept out of the way of eating trichinous flesh. It is indeed conceivable that adult living trichinæ (a few of which are known to be sometimes passed in the fæces of animals in whose intestines the worm is undergoing development) might afterwards enter the alimentary canal of another animal, and that their young might migrate into its muscles. But such an explanation goes but a very little way towards accounting for the great numbers of encapsuled trichinæ which are often found in pigs; and they doubtless derive them from the filth which they devour, particularly perhaps when rats infest the sty, for rats are the most frequent trichinæ-bearers of all animals. The muscles of hedgehogs, moles, and pigeons are known to be often infested with the parasite; and it has been shown to retain its vitality even after the flesh containing it has become putrid.

* The outbreak of a febrile disease among the boys on the "Cornwall," related by Mr Power ('Lancet,' vol. i, 1880), was at first ascribed to trichiniasis.

Secondly, if flesh containing trichinæ should by accident be used for human food, what precautions would prevent those who might eat it from having the parasite develop within their bodies? The answer is that all danger is obviated if the meat is thoroughly cooked.

FILARIA SANGUINIS.*—In 1866 Wucherer detected, in cases of chylous urine occurring in Brazil, certain minute living organisms, evidently the embryos of a nematode worm. Six years later, in 1872, the late Dr T. R. Lewis, in India, discovered similar embryos in the blood. It was very soon found that this was no isolated occurrence, and that the hæmatozoon (as it was called) was by no means limited to persons affected with chyluria. In South China, for instance, among 1000 natives taken at random, about 100 are said to be infested with this parasite. In that country similar larval entozoa (as of *Filaria immitis*) are very commonly seen in the blood of dogs and in many species of birds, so that their presence in man excited the less surprise in the minds of experienced helminthologists. Among those persons who harbour the *Filaria sanguinis hominis* (as Lewis termed it) some appear to be in perfect health, but others are affected with one or more of a limited number of diseases, of which the chief are lymphangeitis with varicosity of the lymph-channels in the inguinal glands, lymph-scrotum, elephantiasis of the scrotum or of the leg, and chyluria.

The next step was the discovery of the parent worm from which the embryos found in the blood are derived. This was effected in 1876 by Dr Bancroft, of Brisbane, in Australia. He first obtained a dead specimen from a lymphatic abscess in the arm, and afterwards four living ones from a hydrocele of the spermatic cord. These he sent to England to Dr Cobbold, who gave to the entozoon the name of *Filaria Bancrofti*. They were all females, and as yet no perfect specimen of a male seems to have been found. The length of the female is from three to three and a half inches; its breadth from $\frac{1}{100}''$ to $\frac{1}{90}''$. It has a circular mouth, destitute of papillæ, a narrow neck, and a bluntly-pointed tail. Its body is smooth, and of an opaline appearance, and it has been described by Dr Manson, of Amoy, in China, as looking "like a delicate thread of catgut, animated and wriggling." This observer, in 1880, while operating on a case of lymph-scrotum, removed at the same time a portion of a living worm, and showed that it lay in the interior of a dilated lymphatic. He also proved that the parasite is naturally viviparous, for he saw fully-formed embryos, exactly like those which are found in the blood, escaping from the animal's vagina. Strictly speaking, indeed, the accuracy of this last statement is open to question. For it has been known from the first that the embryo in the blood is always enclosed in a delicate sac or sheath, which fits it accurately, except that a collapsed or unoccupied part is seen projecting beyond either the head or the tail, according to the direction in which the worm happens to be moving; and it seems now to be certain that this sheath is nothing else than the envelope or shell of the ovum, which, as the embryo develops, yields before it, and so continues to be stretched out over the skin. Still, the fact remains—and it is one of which we shall presently see the importance—that the parent filaria, instead of throwing off ordinary oval and motionless eggs, gives birth to active organisms, capable from the first of vigorous spontaneous movements. The size of these embryos is such as not at all to interfere with their traversing the lymph-paths through any glands that may come in their

* The thread-worm (*filum*, a thread).

way, and so passing on from the lymphatic vessel in which the parent worm lies into the thoracic duct, and beyond this into the blood-vessels. Their diameter, indeed, is only about $\frac{1}{3200}''$, not more than that of the leucocytes which circulate through the lymph-glands; their length is about $\frac{1}{90}''$.

So long as the embryos of the filaria remain in the blood they continue to be of the same size, and show no indication of undergoing further structural development. This fact is of itself sufficient to suggest to anyone acquainted with helminthology the idea that they are waiting to be transferred to some other host; and it seems to have occurred both to Dr Bancroft, at Brisbane, and to Dr Manson, at Amoy, independently of one another, that this host might probably be some species of mosquito which feeds on human blood. Dr Manson remarks that the limitation of the parasite to certain parts of the earth's surface was almost sufficient to exclude from his consideration many blood-sucking animals, such as fleas, lice, bugs, and leeches, which are found pretty well everywhere. He therefore came to the conclusion, in 1877, that is was likely to be either a mosquito or the sandfly that took the embryo filaria from its human host and supplied to it the conditions requisite for its development into a more mature form. Had he been at that time aware of another extraordinary fact that he afterwards discovered, he might safely have set aside the sandfly. This fact is that instead of the young filariæ being found in the blood throughout the whole twenty-four hours, none of them can generally be detected in it during the day, even when they are abundantly present in it during the night. At about 6 or 8 p.m. they begin to make their appearance; by midnight their numbers reach the maximum; as morning approaches they become fewer and fewer; by 8 or 9 a.m. they cease to be discoverable. What becomes of them in the interval is not at present known, but it must be borne in mind that all that has as yet been proved is that the capillary blood-vessels of the integument contain them at night, but not in the daytime. There seems to be no necessity for supposing that they circulate with the blood like its normal constituents, the red discs and leucocytes. Possibly during one part of the twenty-four hours they may be all collected in the pulmonary capillaries or in those of the deeper structures generally, but when the patient retires to rest they may betake themselves to the vessels of the skin. In a case which Dr Stephen Mackenzie has recorded in the 'Pathological Transactions' for 1882, observations at intervals of three hours were made for weeks together, and the periodicity was found to be as complete as it possibly could be. Dr Mackenzie also submitted his patient to the experiment of having his habits of life reversed, so that for nearly three weeks he remained out of bed all night, and rested in the daytime, the hours of his meals being arranged accordingly; the result was that during this time the filariæ were found in the blood during the day, but not at all, or only in much smaller numbers, during the night. Obviously, therefore, that which determines their migrations is the resting or moving condition of their human host. Moreover they were not tempted to come out during the day by even the thickest London fog. But it is nevertheless impossible not to recognise the fact that their usual habit of entering the capillaries of the integument at night-time is precisely adapted to bring them within reach of the proboscis of a nocturnal blood-sucker, like the mosquito. In Dr Mackenzie's case the blood seems always to have been taken from the patient's finger; as much of it was examined as would lie beneath a five-eighths inch cover-glass. In this quantity of blood, at midnight, there were often

fifty or sixty, and sometimes eighty or ninety filariæ. Dr Mackenzie therefore calculated that from thirty-six to forty millions of them were probably present in the whole mass of circulating fluid; but it is obvious that this estimate is enormously too high, if it is only the capillary vessels of the skin that contain them in abundance.

Dr Manson lost no time in verifying his hypothesis with regard to the mosquito. He persuaded a Chinaman, known to be infested with the filaria, to sleep in a "mosquito house." Next morning the gorged insects were caught and examined. The blood in their stomachs was found to contain filariæ in even larger numbers than that of the man from whom it had been derived. It is supposed that they become entangled by their lashes in the proboscis of the mosquito and are so removed from the blood-vessels. Having reached their new host, some of them proceed to enter upon a process of development. They lose their sheaths, grow to the length of one thirtieth of an inch, and acquire a distinct alimentary canal, a mouth crowned with three or four nipple-like papillæ, and rudiments of generative organs. Their movements also become extremely active. These changes are completed in from four to six days. During this time the mosquito, having retired to a shady place, has been digesting her solitary meal, and maturing her own ova; she is now prepared to deposit them upon the surface of water; after which, her career being finished, she dies, and probably falls into the water on which her eggs are laid. The progress of the filaria has not been directly traced further. But there can be little doubt that it makes its way out of the body of the dead insect into the water. And in all probability the next step is that it is swallowed by a human being, from whose stomach it bores a passage into the thoracic duct or into some lymphatic vessel; and along this it then works up stream, in obedience to some strange instinct, until it reaches a spot which it takes for its permanent abode. Here we must suppose that it is joined by another parasite of the opposite sex, after which it proceeds to furnish in the lymph-channels and to the blood-current of its host those swarms of larvæ which formed the starting-point of our inquiry as to the life-history of the entozoon. How long the parent worm lives we do not as yet know; but a case observed by Dr Manson shows that it may be at least as long as thirty-two years; he found living filariæ in the blood of a man aged fifty, who had had lymph-scrotum from the age of eighteen. There is some reason for supposing that it may be killed by the occurrence of severe acute disease in the host. At any rate Dr Stephen Mackenzie's patient was attacked with rigors as the result of going out of the hospital on a cold and windy day in October, and after the following day no embryos were ever discoverable in his blood. Pleurisy set in, and an abscess formed near the left collar-bone; and when he died two and a half months later no trace of the worm could be found. Dr Mackenzie supposes that it became dislodged during the rigor, and reaching the termination of the thoracic duct on the left side of the neck, excited both the pectoral abscess and also the pleurisy. However this may be, it seems clearly to have perished from an early period of the man's fatal illness, and its body must be supposed to have undergone disintegration.

It now becomes an important question to determine how it is that the filaria produces injury of the lymphatic vessels. The only hypothesis that can be said to account for this result is one that has been formulated by Dr Manson, in a paper in the 'Pathological Transactions' for 1882. His idea is that so long as the discharge of embryos goes on after the manner above

described, the parasite is perfectly innocuous to its host. But from some cause or other it happens in certain cases that instead of the larval filariæ enclosed in their sheaths, ova in a much earlier stage of development, with unstretched shells, are extruded from the maternal vagina. Dr Manson has twice obtained such ova from the lymphatics; and probably they have been found in the urine also. Now, according to Dr Manson, they measure $\frac{1}{750}''$ in breadth by $\frac{1}{500}''$ in length; according to Dr Cobbold, $\frac{1}{1650}''$ by $\frac{1}{1000}''$. In either case their transverse diameter is far greater than that of the embryos; and nothing is more likely than that they should fail to pass along channels which the embryos would find no difficulty in traversing. Dr Manson supposes, for instance, that when they are carried by the lymph-stream to a gland they become impacted in the small channels formed by the afferent vessel. The necessary result must be, as anastomosing paths become one after another obstructed, a more or less complete stasis of lymph, not only in the neighbourhood of the spot where the parent worm is situated, but also in the whole of one or both of the lower limbs, and in the scrotum.

The pathological effects of the filaria will be described hereafter in the chapter on affections of the urine and in that on elephantiasis.

The guinea-worm (*Dracunculus medinensis*) is an enormously long nematode worm inhabiting the subcutaneous tissue, usually of the legs, to which it gains access from the water tanks, in India and other hot countries. Its effects are purely those of local irritation.

Two trematode worms have been found in the human body. Of these the liver-fluke (*Fasciola hepatica* or *Distomum hepaticum*) infests the gall-passages, but it is very rarely found in man, at least in civilised Europe (it is said to be more common in Bosnia), although in sheep it causes a common disease, the sheep-rot.

The other (*Bilharzia hæmatobia*) inhabits the pelvic veins of persons living in Egypt and Natal, and produces serious hæmaturia. It will be again referred to in the chapter on that malady.

An acanthocephalous or thornheaded worm, *Echinorhynchus*, sp., has only once been certainly discovered in the human intestine by Lambl ('Prager Vierteljahrschrift,' Feb., 1849). A second case, reported from Netley in 1872, is doubtful. It is common among pigs in England.

DISEASES OF THE PERITONEUM

ACUTE PERITONITIS.—*Importance—Clinical symptoms—Origin: secondary to visceral disease—Puerperal—Urœmic—Extreme rarity of idiopathic acute peritonitis—Morbid anatomy—Local peritonitis and circumscribed abscess—Diagnosis—Treatment: medical and operative.*

CHRONIC PERITONITIS.—*General effusion—Thickening—Adhesions—Locular effusion—Frequency—Causes.*

TUBERCLE OF THE PERITONEUM.—*Anatomy—Symptoms—Relation to phthisis—Diagnosis—Treatment.*

CANCER OF THE PERITONEUM.—*Commonly secondary—Anatomy—Symptoms.*

ASCITES.—*Its physical signs—its diagnosis—its prognosis—Treatment by drugs and by paracentesis abdominis.*

THE peritoneum is part of the great body cavity (*cœlom*) formed by the mesoblast splitting into somatopleure and splanchnopleure. It is a huge areolar space or lymph-sac, and its most intimate pathological relations are not with skin or mucous membranes—not even with the joints or the so-called arachnoid space—but with the pleura, pericardium, and tunica vaginalis.

The diseases of these three divisions of the same original cavity are the same: acute inflammation, serous or purulent, traumatic or septic; chronic adhesive inflammation with hypertrophy, chronic irritative effusion, and passive dropsical effusion, hydrothorax, hydropericardium, and ascites. All three are liable to be invaded by tubercle, and also by cancer. All three are prone to follow the pathological fate of the viscera which they cover; they are all apt to suffer in the course of Bright's disease; and, lastly, they are often all affected together by inflammation, by tubercle, or more rarely by cancer.

On the other hand, rheumatism seldom or never affects the peritoneum as it does the pericardium, and peritonitis is seldom or never the result of exposure to cold, as pleurisy undoubtedly is. Although not so constant a companion of inflammation in any abdominal viscus as is pleurisy of pneumonia, the most frequent cause of peritonitis is undoubtedly the presence of irritating products of inflammation from one or other of the organs it covers, the effect ranging from the adhesions which slowly form about a source of irritation to the rapid and violent inflammation which blazes up when pus or fæcal material finds its way into the cavity.

The most important viscera from this point of view are, for men-patients, the intestines and the stomach, next the liver and gall-bladder, and then the bladder. In the case of women the ovaries, Fallopian tubes, and uterus are a more frequent source of peritonitis than any other viscera.

ACUTE PERITONITIS.—This disease is exceedingly fatal; cases in which it was the immediate cause of death make up a large proportion of the total mortality from disease and injury, at least in hospital practice. Thus, in 1873, taking a year at hazard, of 434 inspections made at Guy's Hospital,

in at least 52 death was directly attributable to acute peritonitis, or nearly one in eight.

The symptoms of this disease vary greatly in different cases, and they are often combined with and masked by those of other affections, so as to make its diagnosis difficult. Let us take the case of a person previously supposed to be healthy, who is from some cause suddenly seized with peritonitis.

We shall find him lying in bed on his back with anxious looks, features pinched and drawn, eyes sunken and often dark. He complains of a sharp cutting or burning pain in the abdomen. This is constant, but it is liable to aggravation if he changes his posture in any way; if he coughs, or sneezes, or strains, and also when there is movement of gas in his intestines. It becomes worse when he stretches out his legs in bed, so that he keeps them drawn up.

Pressure on the abdomen—sometimes the lightest possible application of the hand—causes much pain; the tenderness may either be diffused equally over the whole surface, or it may be most intense at some particular spot, probably the starting-point of the inflammation. The movements of the diaphragm and of the contents of the abdomen during the act of breathing cause so much pain that the patient instinctively keeps these parts at rest, and uses the upper ribs only; the inspirations are therefore shallow, and are repeated forty or even sixty times a minute.

At the commencement of an attack of peritonitis, there are often sharp rigors. These are followed by more or less fever. The *temperature* may rise to 104°, or to 105°. On the other hand, it is important to remember that a normal temperature is no guarantee that peritonitis and even purulent peritonitis is not present. We found the same exception in cases of pleurisy, and even of empyema. When death is approaching, the temperature falls to normal, or even below it. The hands and feet are then icy cold. The *pulse* is frequent, ranging from 100 to 150. At first it is often small, hard, and strong: the wiry pulse. In the later stages it becomes still smaller—feeble, irregular, or imperceptible—the thready pulse.

In fatal cases death usually occurs by collapse, the mind often remaining clear to the last moment. But there may be great restlessness towards the end, the patient tossing about in delirium for a few hours before death, which is sometimes preceded by convulsions.

The fact that the inflammation penetrates to the subserous and muscular coats is perhaps the reason why there is almost always *constipation* in peritonitis, even when the inflammation did not start from the intestine. The bowels can, however, be moved either by purgatives or enemata, if the dangerous mistake is committed of interfering with them, and Dr Fordyce Barker states that in puerperal peritonitis diarrhœa is more frequent than constipation.

Vomiting is among the earliest symptoms, even when the disease has begun in the pelvis. The *tongue* is small, red, slightly furred, and dry. When the case is approaching a fatal termination the patient is sometimes tormented by obstinate hiccough; micturition is often painful and difficult, particularly when the serous coat of the bladder is involved in the inflammation.

The surface of the abdomen in peritonitis is not only tender to the touch, but also much harder than natural. Its walls are distended, sometimes enormously, with gas which accumulates in the paralysed bowels—a condition aptly called *tympanites* and *meteorismus* by the Greek writers. The recti and

other muscles are rigid, and the semilunar and transverse markings may be plainly discerned through the integuments. At first the percussion-note is everywhere tympanitic as in health, but after a time it may become dull in places, and the presence of fluid may be indicated by fluctuation. Effusions of lymph may sometimes be detected by the stethoscope, a friction-sound being heard like that of pleurisy. But this is seldom the case in acute general peritonitis. Fluctuation points to the case becoming chronic, and friction to its being local.

Ætiology.—Acute peritonitis, as above stated, is most commonly caused by extension from one of the *viscera* of the abdomen. It would be impossible to enumerate all the affections that may act in this way, and even a complete list would not be of much practical value. As a rule peritonitis is much less apt to follow lesions of the solid than those of the hollow viscera. The reason for this is that the latter, besides being capable when inflamed of undergoing perforation, and of discharging their contents into the serous cavity, are also much more subject than the former to those septic forms of inflammation which, when they have reached the peritoneal surface, excite it to the same unhealthy action. Thus cirrhosis of the liver does not set up acute general peritonitis, nor does Bright's disease of the kidneys, nor chronic inflammation of the ovaries, nor the swollen spleen of ague or of enteric fever; but a ruptured hepatic abscess, a perforated gall-bladder, a sloughing enteric ulcer, a ruptured bladder or ovarian cyst, or the putrid lining of the uterus and Fallopian tubes after delivery or abortion—these are almost certain to produce acute and fatal peritonitis. A sloughing block in the spleen (the result of ulcerative endocarditis) may occasionally set it up. In three instances its starting-point was suppurative inflammation of the kidney. Other exceptional causes are malignant tumours, or, still more rarely, tubercular lymph-glands.

Among diseases of the stomach the perforating ulcer is by far the most common cause of general peritonitis. Any perforating intestinal ulcer will equally set it up. Even tuberculous ulcers, which are rightly said to have comparatively little tendency to perforate, have done so in several cases at Guy's Hospital within the last twenty years. Hernia and intestinal strangulation frequently give rise to peritonitis by extension of inflammation; but chronic obstruction, even fæcal impaction of the bowels, may also produce fatal peritonitis by ulceration and perforation of the dilated part of the gut above the seat of obstruction. Lastly must be mentioned surgical operations on the abdominal organs as occasions of peritonitis, although this result is rendered far less likely by strict antiseptic precautions. Typhlitis, with the various forms of intestinal obstruction, and pelvic suppuration in women, are the most frequent causes of all.

In some of these varieties of peritonitis the cause is obvious; in others it may be utterly obscure, the patient being apparently in perfect health until he is attacked by acute inflammation of the whole abdomen. This is frequently the case when the starting-point of the disease is a perforating ulcer of the stomach or duodenum; and even when it is a typhoid ulcer of the ileum, the occurrence of peritonitis may be the first indication of illness, for, as we have seen, enteric fever may be entirely latent. But the most important of all these obscure causes of peritonitis is typhlitis, and next to this, affections of the pelvic organs of the female, which may be altogether latent.

In most, if not in all cases, *puerperal peritonitis* starts from an un-

healthy inflammation of the lining membrane of the uterus, which reaches the serous membrane either along the Fallopian tubes or through the tissue of the organ, the venous channels in which are often filled with pus. Miscarriages, again, are not rarely followed by peritonitis; and it may also be set up by extra-uterine fœtation, a pelvic hæmatocele, suppuration of a Fallopian tube, sloughing of an ovary, and various other affections of the organs in question. The most careful vaginal examination should therefore be made in every case of peritonitis the cause of which is not evident.

Acute peritonitis is not in all cases traceable to an antecedent local disease. Sir Thomas Watson, in common with many of the older writers, gave exposure to cold as one of its causes; but the pathology of the dead-house lends no support to such an opinion. Dr Fordyce Barker, indeed, speaks very confidently of having seen puerperal peritonitis caused by a chill; and at least shows clearly that in some cases no inflammation can be detected in the uterus or the neighbouring organs. But it is surely more probable that there is really an undiscovered local cause than that these cases of peritonitis differ from all others in being set up by exposure to cold.

Smallpox, typhoid fever, gout, rheumatic fever, glanders, pyæmia, erysipelas, have all been at one time or another regarded as causes of acute peritonitis. But the evidence for these statements would be difficult to produce.*

Of general predisposing causes, Bright's disease frequently causes general acute peritonitis. Sixteen cases of this kind were observed in Guy's Hospital between the years 1854 and 1872. The inflammation was generally suppurative; and there was often a marked absence of vascular injection of the serous membrane. The kidneys were, as a rule, enlarged, and in a more or less advanced stage of epithelial nephritis; but in three cases they were contracted and granular.

At Guy's Hospital during twenty years only two cases of acute peritonitis are recorded which could not be attributed either to renal disease or to extension from a viscus. In 1874, however, several children at a school at Wandsworth were attacked at the same time with acute peritonitis. The late Dr Anstie investigated this epidemic; and the conclusion at which he arrived was that the disease was caused by exposure to the influence of sewer-gas. It was in making a *post-mortem* examination in one of the fatal cases that he received the wound in his finger which cost his valuable life. Dr Shirley Murphy has since met with the following case, which appeared to be attributable to a similar cause. A woman, aged thirty-six, died on her way to the Homerton Fever Hospital. A *post-mortem* examination showed that

* Dr Wilks has recorded a case in which this disease occurred as part of an erysipelas that had started from an ulcer in the groin; but probably the inflammation then extended through the parietes, just as it sometimes does when the surface of the body is severely burnt. As regards pyæmia, the only case that I know of in which it seemed to have given rise directly to peritonitis was that of a man who died in less than an hour with cerebral symptoms, and in whom the only lesions found after death were, softness of the spleen, and the presence of about six ounces of pus in the lower part of the peritoneal cavity, with a smaller quantity of pus in the left knee-joint. As no history of the case could be obtained, I had no means of knowing whether he had suffered from any symptoms of illness previously; but it was certain that he had been giving evidence in a court of law just before he was attacked with a fit, which rapidly passed into fatal coma. This case, however, was in the highest degree exceptional. Instances of ordinary pyæmia are constantly presenting themselves in the *post-mortem* room; and I do not know of one in which acute peritonitis was present, and in which this did not start from some local mischief. I believe, therefore, that at the bedside it is needless to think of pyæmia (apart from abdominal abscess) as one of the possible causes of peritonitis.—C. H. F.

acute peritonitis was the cause of death, the coils of intestine being matted together by lymph. The intestines, uterus, and other viscera were healthy, and no local starting-point for the inflammation could be discovered. It was afterwards ascertained that the patient had been living in a house the drain-pipe of which was obstructed, so that for two or three weeks the sewage had been spread over the yard. When first taken ill she shivered, fainted, and vomited; next day she compained of pain in the left iliac fossa, with purging; and two days later she died.

Anatomy.—The morbid changes which occur in the peritoneum under inflammation present considerable variations, but rather of degree than kind. They are essentially the same as in other serous membranes. The surface first becomes reddened, from injection of the minute vessels. This injection is often not uniform, but is especially marked along two longitudinal lines, which run over the bowel, at a little distance from one another, parallel with the attachment of the mesentery. The explanation of this appears to be as follows:—In health atmospheric pressure keeps every part of the serous surface in contact with some other part of it; the intestines are not (as one is apt to suppose) regularly rounded tubes; they are flattened against one another and the abdominal wall. But the distension caused by peritonitis leads them, on simple physical principles, to assume a cylindrical form; and the result is that blood is forcibly drawn into the angular spaces between them. The red lines so produced may therefore be termed "suction lines," as was proposed by Dr Moxon. They are not always present, being of course wanting when the intestines fail to become distended, when air has access to the peritoneal cavity, and perhaps also when inflammatory effusion is poured out very early and in large quantity.

The further morbid appearances vary according as the inflammation tends rather to the effusion of lymph or to suppuration. In the former case the membrane becomes dull and lustreless, and very soon it presents shreds and small patches of fibrin; in the latter case it is even more lustrous than in health, and feels greasy to the touch.

The inflammation in some cases does not go beyond the formation of lymph. This forms a layer of greater or less thickness, which may either be limited to certain parts or cover the whole surface of the serous membrane. Microscopically it consists of fibrinous threads which cross one another in all directions, leaving interspaces in which are masses of cells.

But fluid effusion does not always precede the deposition of lymph. When inflammation of moderate intensity extends to a serous membrane from limited areas of disease in a subjacent organ, the corresponding parts of its surface become covered with local patches of fibrin. And this is the case even where the affected parts are on the sloping sides of the lungs or liver, from which any fluid must at once gravitate away. The minute observations of Rindfleisch, Ranvier, and Klein prove that some of the cells are derived by proliferation from the endothelium; others are probably exuded leucocytes. Klein, in his 'Anatomy of the Lymphatic System,' describes the lesser omentum and mesentery being œdematous and swollen to five times their normal thickness in animals in which he had set up artificial peritonitis. The occurrence of effusion into the subserous tissues accounts for the well-known fact that in peritonitis the membrane can be stripped off much more readily than in health.

Whenever the inflammation goes beyond a certain degree of intensity, fluid is also effused into the serous cavity. This is always somewhat turbid,

and under the microscope exhibits more or less numerous leucocytes. When they are sufficiently abundant to give to the liquid a milky colour, this is said to be purulent. Thus every gradation may occur between the purely serous form of inflammatory exudation and that which is pure pus. All depends upon the proportion of leucocytes to serum.

If, on the other hand, the peritonitis remains at a lower degree of intensity it leads to adhesion of the opposed surfaces of the serous membrane. The active agents by which this is brought about appear to be the cells embedded in the fibrin. Some pass into spindle-cells, and ultimately form perfect connective tissue, while others develop into blood-vessels, the walls of which are at first exceedingly soft, consisting entirely of opposed cells. These readily give way if the exudation-fibrin is subjected to pressure or traction by the movements of the organ beneath. Spots of hæmorrhage are consequently often seen, and sometimes the amount of blood effused is very great. Writers have described a hæmorrhagic form of peritonitis which appears to arise in this way.

The adhesions resulting from peritonitis may be universal, the cavity being obliterated and the abdominal organs united together by connective tissue, from which they have to be dissected out when a *post-mortem* examination is made. More frequently, perhaps, the opposed surfaces adhere in certain places only. They still move on one another as in health; and thus the tissue which connects them becomes stretched into bands or cords which may acquire a considerable length. These we have already found to be causes of intestinal obstruction (p. 419).

Even when the inflammation has gone on to the effusion of a large quantity of fluid, the possibility of its terminating in adhesion is by no means excluded. The fluid may be absorbed, and the two surfaces may then come together and unite. The connecting fibres seem then to be formed from the cells of a layer of granulation tissue, which covers each surface, and is derived from the endothelium of the serous membrane. Even pus may dry up and become converted into a caseous mass embedded in the fibrous tissue of the adhesions.

Circumscribed peritonitis.—In most cases of peritonitis the inflammation starts from some one spot and diffuses itself over the whole abdomen. This process is doubtless much accelerated by the movements of the intestines, so that parts already inflamed are brought into contact with others which had not hitherto been reached by the disease. When the stomach or intestine has been perforated, the extravasated matters may be carried to the most distant parts of the cavity. But it by no means necessarily follows that the inflammation should thus spread over the whole surface of the peritoneum. The omentum often seems to retard for a time its progress towards the organs situated above it. And even when pus is poured out, it may be limited by the agglutination of the two surfaces of the serous membrane round the space which it occupies. Thus peritonitis starting from the uterus may lead to a circumscribed abscess occupying the pelvis and more or less of the lower part of the abdomen. In one case of this kind, the pus had been discharged through the bladder during life, and in another through the umbilicus. So also peritonitis arising from ulceration of the intestine often gives rise to localised collections of pus. This is especially apt to occur when the cæcum is the starting-point of the disease. The abscess then forms a swelling in the right iliac fossa. It sometimes points near the crest of the ilium, but not infrequently it passes down below

Poupart's ligament and discharges in the groin. In other cases, again, it makes its way backwards towards the loin. Such abscesses are sometimes difficult to distinguish from those caused by diseased bone, and the difficulty is increased by the fact that when the crest of the ileum lies in the way of the pus, part of it sometimes becomes denuded of its periosteum so as to be within easy reach of a probe (cf. p. 386). The bowels sometimes communicate freely with an abscess of this kind, and much fæcal matter may be discharged with the pus. In one case for a considerable time before the patient's death almost all the fæces passed through an opening in the groin; in that instance the abscess was secondary to cancerous disease of the cæcum. The fact, however, that the pus discharged from a circumscribed abscess in the abdomen has a fæcal odour does not prove that there is a communication with the intestine. Several writers have pointed out that matter collected in the neighbourhood of the bowels may acquire such an odour as a result of the diffusion of the intestinal gases. And, on the other hand, when there is an opening, this does not invariably prove that the abscess started from the ulcer. Dr Habershon has insisted on the fact that the intestine may be perforated secondarily by a suppurating gland.

Another form of circumscribed abscess in the peritoneal cavity is limited to the sac of the lesser omentum. In one such case this cavity contained two or three pints of pus, the mischief having started from disease of the pancreas. Such cases are exceedingly rare. On the other hand, abscesses limited to one or other hypochondrium are by no means uncommon. In the 'Guy's Hospital Reports' for 1873–74, several cases of this kind were recorded by the author. In some of them the abscess started from an ulcer of the stomach, or other disease in the neighbourhood, but in most it resulted from some direct injury, particularly when seated in the right hypochondrium. In cases of the latter kind the suppuration is often preceded by a circumscribed effusion of blood, which may itself form a distinct swelling, and which is more or less closely analogous to the pelvic affection known as peri-uterine hæmatocele. In one of the most interesting of these cases the patient had been kicked in the left side, and came in with a large rounded tumour in the hypochondrium. After a time we found that air had entered it, for curious musical sounds, synchronous with the heart's beats, were heard over it, and the percussion-note became tympanitic. Yet there were no symptoms indicative of constitutional disturbance, and the man left the hospital, refusing to believe that anything serious was the matter with him. Some time afterwards he returned, saying that he had voided a quantity of matter, and that the tumour had disappeared; on examination, no trace of it could be discovered. Cases of abscess in the hypochondrium, however, do not often terminate so favourably.

Diagnosis.—This may be considered under three heads.

(1) When a person, previously supposed to be well, is suddenly seized with pain in the abdomen, it may be far from easy to determine whether the attack is peritonitis, or whether it is colic or hysteria. The chief distinction is found in manipulation of the abdomen. In *colic*, pressure and friction give relief; the belly is usually hard and contracted; the pain intermits from time to time, so that the patient has intervals of complete ease; and when his sufferings are at their worst he is restless, and tosses about in search of relief. In *hysteria*, on the other hand, there often appears to be the most extreme tenderness of the surface; but if the patient's attention be diverted, no further complaints

are made, and after a time considerable pressure is perfectly well borne, while the abdominal walls are quite soft and supple. The exaggerated susceptibility and sensitiveness to the lightest touch are in such cases the very symptoms that show the absence of serious disease. One must inquire whether the patient has ever had hysterical attacks, or amenorrhœa; but we must not forget that acute peritonitis from perforating ulcer of the stomach occurs in anæmic young women who are very likely to have had hysterical symptoms. It is possible that the thermometer may sometimes show, in cases of mere hysteria, a rise of temperature; and, on the other hand, pyrexia may be absent in peritonitis. The probability that the pain is due to colic is of course greater if the patient has eaten any indigestible food, or if the gums present the dotted lead-line. In all doubtful cases, however, one must remember that to attribute to peritonitis a pain really due to colic is an error from which no evil consequences can follow, whereas the converse mistake may be fatal to the patient; and a few hours' delay will always solve the question.

The rupture of a concealed aneurysm into the abdominal cavity by a small aperture, or into the subperitoneal tissues, is another possible cause of sudden severe pain that must not be overlooked, particularly if the patient fainted when the attack began, or was pulseless from the first. It is sometimes impossible to distinguish this from perforation of the stomach or intestine, and the same applies to hæmorrhage from the Fallopian tubes.

(2) But when one has come to the conclusion that peritonitis is present or impending, one has not completed one's diagnosis. Its *cause* remains to be discovered. As already pointed out, among the very numerous affections that may give rise to inflammation of the peritoneum, comparatively few are likely to be absolutely latent until the time the peritonitis is set up. Hence, when a person supposed to be healthy is attacked, the range of possible, or at least of probable, causes is after all not very extensive. Perforating ulcer of the stomach or duodenum, perforating typhoid ulcer of the ileum, disease of the cæcal appendix, and pelvic affections in the case of a woman, are the chief. Now, a perforating ulcer of any part of the direct channel of the alimentary canal is commonly fatal in a few hours, or in a day or two at the latest; it may even be still more rapid. The 'Pathological Transactions' contain reports of one case in which the patient died within an hour, and of another in which the subject of the disease, an Oxford professor, fell down dead while walking in the streets of London. Hence when peritonitis runs a more protracted course than this in a male patient (or even in a woman, if the ovaries, Fallopian tubes, and uterus can be proved to be healthy), there is a very strong presumption that it started from the cæcal appendix; and, if recovery should take place, a hard mass can often be discovered in the position of the cæcum.

(3) There is also a *negative* side of the difficulty in the diagnosis of peritonitis; *e.g.* acute suppurative peritonitis, starting from disease of the vermiform appendix, may run its course without characteristic symptoms. Such instances are doubtless very rare, but under other circumstances it not uncommonly happens that peritonitis remains altogether latent. In enteric fever, for instance, all physicians know that perforation of the intestine is often found in the *post-mortem* room to have taken place some hours or days before death, although there had been neither increased pain nor tenderness in the abdomen, nor, indeed, any marked aggravation of the symptoms, to suggest that the fatal issue was being

thus brought about. The cause of this is generally supposed to be that patients suffering from fever have their senses and intelligence stupefied. But the truth is that peritonitis remains latent no less frequently in persons whose minds are clear to the last. Thus in making a *post-mortem* examination after an operation for hernia, ovariotomy, or the like, we have repeatedly found universal peritonitis when those who had watched the patient most closely had detected no evidence of it during life. In any case in which symptoms of intestinal obstruction have been present for a few days, one can never assert positively that inflammation of the peritoneum has not already set in. But then it is to be observed that the symptoms of ileus really scarcely differ in kind from those of peritonitis; the principal distinction is the fact that the constipation is insuperable in the one case but not in the other.

Again, in the majority of cases in which acute peritonitis is set up by Bright's disease, its presence is first discovered on the *post-mortem* table, the patient having at most complained of pain in the abdomen such as might have arisen from some trifling cause.

In latent forms of peritonitis the effused fluid is generally pure pus. In connection with this, it may be noted that some of the older writers describe the pain in peritonitis as subsiding when free suppuration has taken place; and they add that it is important for the practitioner to be aware of this, lest he should commit the error of supposing that the patient is about to recover when in reality his death is surely approaching. The most trustworthy guides to a prognosis in peritonitis are, they say, the aspect of the patient and the state of the pulse. The more frequent its beats, the greater the danger.

Treatment in acute peritonitis must to a great extent be varied according to the conditions under which the disease arises. When it is the last in a series of morbid changes that tend irresistibly to destroy the patient, there is nothing to be gained by active interference. Very often little can be done beyond the application of hot poultices or turpentine stupes to the abdomen, and the administration of morphia by subcutaneous injections for the relief of pain.

The case is very different when the disease is set up by a perforating ulcer of the vermiform appendix or by any similar local affection. In the whole range of therapeutics there is nothing more important than the treatment of peritonitis of this kind. It is not saying too much to assert that a single error in conducting such a case may at almost any period be the immediate cause of death; and, on the other hand, that skilful and judicious management is often the direct means of saving life. *Physiological rest* to the inflamed parts is the one thing essential. The patient must be kept in bed from the moment that the existence of peritonitis is suspected, and must maintain the recumbent position most scrupulously, being forbidden to sit up for any purpose whatever. A pillow may be placed beneath the knees to support the thighs in a flexed position. No purgative of any kind should be administered or allowed to be given, even though the bowels should remain closed for many days; in most cases not even an enema. The importance of this rule may be made apparent by quoting a remark of Dr Habershon's that in cases of this kind he has, at the *post-mortem* examination, seen castor-oil floating on the contents of the abdominal cavity.

Whether nourishment and medicines should be given by the mouth depends upon the cause to which the peritonitis seems to be attributable.

When there is reason to suspect that it is due to perforating ulcer of the stomach this organ should be kept perfectly empty. A most striking instance of success from what may be termed the "starvation" plan of treatment was many years ago recorded by Dr Hughes in the 'Guy's Hospital Reports' (2nd series, vol. iv, p. 332). A young woman became collapsed, and was seized with severe pain in the stomach. The last food which she had taken was a little gruel, four hours before; for some days previously she had eaten almost nothing. She sent for the late Mr Ray, of Dulwich, who (instead of giving her brandy and castor-oil, as so many medical men would have done) administered twenty minims of tincture of opium in a little water. She rallied somewhat, and was carefully removed to the hospital. She was there ordered half a grain of opium in a pill every three hours and to have nothing whatever to drink except two measured teaspoonfuls of toast and water. After two days she complained much of thirst. An enema of five ounces of strong, tepid beef-tea was therefore administered, with five minims of the tincture of opium. This was afterwards repeated three times a day. She was also allowed to suck one teaspoonful of beef-tea jelly, instead of the toast and water. It was not until the ninth day that she was permitted to have two tablespoonfuls of strong mutton broth. She completely recovered, and was discharged from the hospital. Nearly four months afterwards, having been so foolish as to indulge largely in cherries and gooseberries, she was attacked with the same symptoms as before. She had brandy and water given to her, and died in nineteen hours. An ulcer in the stomach was found, which had become torn away from a thick layer of old lymph by which it had before been closed. In its neighbourhood there were old vascular adhesions. It seems almost certain that in this case perforation of the stomach occurred during the first attack, as well as the second, and that she would have died on the former occasion had a less skilful plan of treatment been adopted.

In a case of the same kind it would perhaps be well to allow the patient to have nutrient enemata from the first, so as to assuage the thirst; and to administer even the opium by the rectum or subcutaneously as morphia. Perhaps it is never wrong to let the patient suck ice broken up into small pieces (ice *pills* as the Germans call them), but he must take them very slowly, so as hardly to make more frequent efforts to swallow the water derived from them than he ordinarily makes to get rid of the saliva which is constantly being poured into his mouth.

Unless these precautions be adopted, perforation of a gastric ulcer, even if it should happily have taken place at a time when the stomach was empty, terminates fatally in a few hours, or at latest within a day or two. If, therefore, one is called to a case of peritonitis which has already run on for some days, one may, in the absence of direct evidence, commonly conclude that it is due to disease of some other viscus; and very small quantities of milk, beef-tea, and the like, may be allowed at intervals, as the stomach may be able to bear them.

Opium should be administered freely in all cases of peritonitis. This practice was first introduced by Dr Graves, of Dublin, who, in 1822, ordered it in very large doses, to relieve the agony experienced by a woman in whom inflammation had set in after the operation for tapping. Her case seemed hopeless; but to his great astonishment she recovered. Dr Graves, however, used also to give calomel, which at the present day is believed to be unadvisable. Two grains of opium should be given at first, and afterwards

one grain every two or three hours, the action of the drug being of course carefully watched. There is great tolerance of this remedy in cases of peritonitis. A lad, who had probably never swallowed a dose of opium before, once took as much as twelve grains daily, without being made sleepy or having a furred tongue, and without his pupils being in any way affected by the drug. When the disease subsides, the greatest caution should be exercised in discontinuing the remedy. In one case the bowels began to act regularly every day, while the patient was still taking a grain of opium every two hours throughout the day and night.

In many cases a few leeches may be applied to the most painful part of the abdomen with advantage; the pain is often much relieved by them. Warm fomentations or large poultices should be used constantly, and changed as often as they cool. When there is much meteorismus, great relief is often afforded by a flannel, wrung out of boiling water, and sprinkled with turpentine. A long tube, introduced into the rectum, and cautiously pushed upwards, has sometimes been known to afford escape to a large quantity of gas from the colon; but it much more often fails.

If death should seem to be impending from tympanitic distension, it may be necessary to puncture the intestine, with a very fine trocar, through the parietes, but this procedure is attended with more risk than in cases of mere mechanical obstruction, because the coats of the bowel when inflamed lose their elasticity, so that the hole made by the trocar is apt to gape after its removal, and may allow fæcal matter to escape into the peritoneal cavity.

When the distended and immoveable abdomen, the small and rapid pulse and the other symptoms above enumerated show that general and acute peritonitis is already present, the ill-success of all treatment, even that by full doses of opium, to do more than procure an easy death, has led to attempts in other directions; and the experience of surgeons in performing ovariotomy and other operations involving the peritoneum has encouraged the bold procedure of opening the abdomen, washing out the products of inflammation, and putting in a drainage-tube. This plan of treatment has been carried out in several cases, sometimes with temporary relief, sometimes hastening death, but sometimes undoubtedly saving an otherwise forfeited life.*

Prognosis.—It still remains that something should be said with regard to the prognosis of acute peritonitis, which, however, could not precede the consideration of its treatment. The intensely dangerous character of this disease is manifest. Sometimes death is inevitable, particularly when the inflammation is set up by perforation of an ulcer in a stomach containing a considerable quantity of food; and when perforation occurs in enteric fever, recovery is doubtless exceedingly rare, although instances of it have been recorded. But that form of peritonitis which is set up by ulceration of the cæcal appendix is, when properly treated, far less dangerous than is supposed, if only its nature is correctly diagnosed, and if it is treated according to the rules laid down above, with no purgatives or enemata. Nor of late years have any fatal cases occurred in Guy's Hospital, except such as died very shortly after admission. Hence one may give a favourable though a guarded

* See Dr Buchanan's case referred to in the note on p. 432, also an important paper by Mr Thos. Smith, in the 'St Barth. Hosp. Reports' for 1873 (vol. ix), one by Sir Jos. Lister, in the 'Lancet' for 1881 (vol. ii, p. 863), and cases brought before the Royal Medical and Chirurgical Society in 1885, by Mr Howard Marsh and Mr Treves, and before the Clinical Society in the present year (1887), by Dr Knaggs and Dr K. Clarke, of Huddersfield.

prognosis in cases of typhlitis, even when symptoms of diffused peritonitis are present.

Simple Chronic Peritonitis.—In this affection the whole surface of the peritoneum is thickened and opaque. Adhesions often exist between different parts. Thus the liver, spleen, and stomach may be united into a single mass by firm connective tissue, and may be closely adherent to the diaphragm and abdominal parietes. Very frequently the omentum is drawn up, and its folds are inextricably blended together; so that, with the fat which it contains, it forms a solid mass, binding the colon to the stomach. The intestines may be fixed to the front wall of the abdomen; indeed, the entire peritoneal cavity may be closed by adhesions in cases of this kind. Far more commonly, however, the small intestines, if adherent at all, are so only among themselves, and they are then collected in a more or less rounded mass in front of the spine. Sometimes the membrane which unites the several coils can be stripped off, leaving the intestines still covered with a serous coat. This tendency to the formation of adventitious membranes, looking like thickenings of the peritoneal coverings of the viscera, but in reality superimposed upon them, may be seen in all parts of the abdominal cavity. Such a "reduplication" of the capsule of the liver is one of the most remarkable features of the affection known as perihepatitis; and perihepatitis, beside occurring as an independent disease, forms part of very many cases of chronic peritonitis.

The newly-formed membranes may further form adhesions among themselves, dividing the general cavity into a number of *separate chambers* each containing fluid. A very remarkable instance of this occurred in Guy's Hospital in 1860. A woman, aged forty-four, was sent to the hospital, supposed to be suffering from cystic disease of the ovaries. The physician under whose care she came doubted this, and thought that there was fluid in the peritoneal cavity. After some weeks she died. At the autopsy it appeared at first as though the original diagnosis had been correct. Nothing could be seen but a mass of cysts covering the intestines, the stomach and the liver. Presently, however, it was seen that these cysts had been formed, not in the ovary, but in the peritoneal cavity. Several of them lay between coils of intestine, and some contained a fluid of milky appearance, from the admixture of chyle. Another very similar case came not long ago under the author's care. The abdomen contained a considerable quantity of fluid; and this would have been regarded as passive ascites (caused by the heart disease for which the patient was admitted) had it not been that the physical signs were in some respects anomalous. After death the peritoneal sac was found to be divided into a number of distinct chambers by adhesions; one of them was above the transverse colon, another occupied the middle of the abdomen, a third filled the right loin.

In the great majority of cases of chronic peritonitis, however, the small intestines are not compressed by an adventitious membrane, nor even adherent among themselves. Their coils are still capable of moving on one another, and their mesentery is fan shaped. But the mesentery is remarkably shortened. It may measure not more than about two inches from the spine to the attached edge of the bowel, which is thus tethered very closely to the back of the abdomen, instead of floating freely. Moreover, the length of the bowel itself is greatly diminished. It may not be more than a few feet long from the duodenum to the cæcum; so that the mucous membrane

of the ileum is thrown into folds, resembling the valvulæ conniventes of the jejunum. Its diameter is no less contracted, so that it may hardly admit the little finger. The muscular coat of the bowel is generally thin, but that of the stomach is sometimes much thickened, so that it resembles an india-rubber bottle more than anything else (cf. p. 366).

In the great majority of cases of chronic peritonitis, a more or less transparent straw-coloured fluid is effused into the abdominal cavity. Even when no liquid is found after death, it had probably been present at a former stage of the disease. Sometimes, instead of being pale, the liquid is darkened by the presence of blood; sometimes it contains flakes of lymph or even pus; but the latter, when present, is generally the product of an acute inflammation, supervening upon the chronic disease, as a result of paracentesis. The surface of the peritoneum, besides being thickened, is opaque. But opacity is not a proof that chronic peritonitis has existed; in cases of dropsy the peritoneum generally, perhaps always, looks white and opaque. This has been regarded as a cadaveric change, due to imbibition by the dead tissues, but it may also be due to the action of the fluid upon the serous membrane during life. In a large proportion of cases of chronic peritonitis, however, the peritoneum is not white, but blackened, or of a slaty colour, from effused blood. This appearance is often particularly marked over the intestines.

Age.—Simple chronic peritonitis is very far from being a rare disease. In Guy's Hospital there is on an average one case of this kind to two of ascites from cirrhosis of the liver, the most common of the local causes of ascites. Of thirty-four cases, eighteen occurred in males, sixteen in females. There was in these cases a very wide range in the age of the patients. Between twenty and thirty, there were almost as many cases as between thirty and forty, or between forty and fifty; several patients were more than sixty years old, and one had passed the age of seventy. It appears to be about equally frequent in both sexes.

Causes.—Very little can as yet be said of the causes of chronic peritonitis. Sometimes we can trace its origin in what may be called a subacute attack. The peritoneum fills with serum, with little or no pain or febrile symptoms. In these cases it may be removed by diuretics or by tapping and not return again, although more often it goes on to the ordinary chronic ascites with thickened peritoneum. In other cases the process is insidious from the first.

In a remarkable case we had several years ago in Guy's Hospital, the writer saw the origin of the disease in a healthy country lad of fifteen, and its slow increase, until at last it proved fatal, after nearly two years. Here there was a similar chronic effusion into both pleuræ and into the pericardium, with enormous thickening of all the serous membrane, including the tunica vaginalis, with which an open inguinal canal communicated. There was no trace of tubercle found *post mortem*, and the viscera, including the kidneys, were perfectly healthy. It was an example of a concomitant affection of the peritoneum, pleura, and pericardium, comparable to those of tuberculosis of the serous membranes, which will presently be described.

The disease seems very seldom to be distinctly traceable to any one of the subjacent viscera; at least only three such cases are recorded in our *post-mortem* books: in one it was believed to have started from the cæcum, in another from old pelvic cellulitis, and in the third from the same disease, which itself arose from morbus coxæ. Sometimes, perhaps, perihepatitis is

its origin. Like that affection, it commonly occurs in patients who have Bright's disease, which therefore may perhaps be regarded as its principal cause. Many patients affected with chronic peritonitis have been intemperate, some have suffered from lead, some have been gouty, and others have had disease of the heart.

The principal *symptom* of chronic peritonitis is the presence of fluid in the abdominal cavity. This can most conveniently be discussed further on, together with its diagnosis, prognosis, and treatment (*infra*, p. 480).

Tubercular Peritonitis.—In this disease the peritoneum is covered with minute grains, which, however, are seldom uniformly distributed over its surface, but are much more numerous in some parts than others, especially on the under surface of the diaphragm and in the flanks. The serous surface of the intestines is sometimes comparatively free. The omentum often contains a large quantity of yellow cheesy material, or of recent tubercle, and it is drawn up into a flattened mass, which may be as much as two or three inches thick, lying below the stomach and over the colon. The abdominal cavity is often found to contain a considerable quantity of turbid serum or pus; but more frequently it is closed by adhesions, or there are merely a few scattered collections of liquid here and there between the viscera. Tuberculous affections of other parts are commonly associated with tuberculous peritonitis. Thus, in women, the Fallopian tubes are almost always affected; they are much enlarged, lined with a thick caseous layer, and very often contain pus. Sometimes the same condition is present also in the cavity of the uterus. Dr Moxon thinks that the disease spreads into the open mouths of the tubes from the serous surface; he has observed that the tuberculous change is often limited to the ends furthest from the uterus. In men, the epididymis or testis (on one side or both) is sometimes the seat of tuberculous mischief. When this can be made out during life, it may afford great help in the diagnosis.

Other serous membranes often become affected in the same way as the peritoneum. Thus one or both of the pleural cavities may contain a considerable quantity of fluid, or they may be covered with tubercles and closed by adhesions; and in at least two cases tuberculous pericarditis existed, attended with the effusion of a large quantity of pus (cf. vol. i, p. 948). The intestines often show tuberculous ulcers. In seven cases out of nine the lungs contained tubercles, but it very seldom happens that pulmonary disease is present in such a form as to be capable of recognition during life.

The symptoms of tubercular peritonitis are often very vague and obscure. The patient becomes out of health and loses flesh. He complains of pains in different parts of the abdomen. He may have diarrhœa, particularly if the intestines are ulcerated. The abdomen may be tender and harder than natural, and it may feel hot. Very often it is rather retracted than enlarged, but sometimes it is tumid, and there may even be marked fluctuation, and other indications of the presence of fluid in considerable quantity. Clinically, ascites is more often detected than might be supposed from mere *post-mortem* observations; for at an early stage of the disease the peritoneum frequently contains fluid, which is absorbed in its further progress.

Age and sex.—Tubercle of the peritoneum is one of the common diseases of childhood, and when associated as it often is with tubercular ulceration of the bowels and secondary tubercle of the mesenteric lymph-glands, it

assumes the familiar clinical aspect of tabes mesenterica. But it would be a great mistake to suppose that tuberculous peritonitis is only a disease of early life. In twenty-eight successive fatal cases at Guy's Hospital, two patients were under ten years of age, six between ten and twenty, eight between twenty and thirty, five between thirty and forty, three between forty and fifty, and four over fifty. The disease is more than twice as common in men as in women. Of the twenty-eight cases only eight were in females; and in all of those above the age of puberty, with one exception, there was coexistent disease of the Fallopian tubes.

Diagnosis.—In the account just given of the symptoms of tubercular peritonitis there is little to distinguish it from other forms of chronic and subacute abdominal disease. Great assistance, therefore, is often afforded by the induration of the omentum, which may be felt as a rounded tumour running more or less obliquely across the abdomen above the umbilicus. It has been mistaken for the edge of the liver, depressed and rounded by thickening of its capsule; but a resonant percussion-note can be elicited *above* the mass, where, if it were a hepatic tumour, there must have been absolute dulness. Another sign of tubercular peritonitis is the existence of inflammation and thickening, and even of erysipelatous redness, round the umbilicus. This may sometimes result from adhesion of the small intestine to the abdominal wall at this spot; for, in one case, a fæcal fistula resulted. More commonly, perhaps, it is caused by an extension of the inflammation of the parietal peritoneum to the surface along the track of the obliterated umbilical vessels, just as we shall see that cancerous disease is often propagated. In some of those rare cases of strumous peritonitis in which the abdomen becomes distended with pus, the umbilicus gives way and allows the fluid to escape.

The diagnosis of tubercular peritonitis may be confirmed by the discovery of coexistent effusion into one of the pleural cavities, or into the pericardium.

Lastly, whenever we suspect tubercular peritonitis in a female patient, we must not forget how constantly this disease is associated with tubercular disease of the female generative organs. One patient has had amenorrhœa for eighteen months, another has had menorrhagia, a third had a miscarriage a month before her abdomen began to enlarge, a fourth had one period which lasted a fortnight, and in which the flow was excessive; she then missed her next period, and from that date her abdomen began to swell and her fatal illness commenced.

Prognosis.—The clinical recognition of tuberculous peritonitis is the more important because the disease has by no means so decided a tendency to terminate fatally as might be supposed. We have had several instances in which there was reason to believe that recovery from it took place, and in one case the diagnosis was afterwards proved to be correct by a *post-mortem* examination. The patient (who had left the hospital apparently well) came in again some months afterwards, and died with tubercles in almost all parts of his body; and it was clear that the peritoneum had been the seat of former mischief of the same character. But the most remarkable instance of recovery from tuberculous peritonitis is one recorded by Sir Spencer Wells. The patient, a female aged twenty-two, was believed to have an ovarian tumour, and had twice been tapped. It was decided that ovariotomy should be performed. But, on the abdomen being opened, the peritoneum was found studded with myriads of tubercles. Some coils of small intestine

were floating, but the great mass was bound down with the colon and omentum, all nodulated with tubercles, towards the back and upper part of the abdomen. The fluid was pumped out and the wound closed. The patient went through a sharp attack of peritonitis, but recovered, and she afterwards married; six years later she was stout, hearty, and well.

Treatment.—It appears probable that in children tubercular peritonitis is capable, in the majority of cases, of being cured by the local application of linimentum hydrargyri. This practice has long been carried out in Guy's Hospital, the liniment being spread freely over the surface of a flannel belt, which is stitched tightly round the abdomen. We have more than once seen the greater part of the fluid removed within a few days under such treatment, and the patient has also improved in health and gained strength. It is true that there has been no direct proof of the tuberculous nature of the affection, but the cases in question were such as are commonly regarded as instances of "strumous peritonitis," and many of them were running a chronic course. There is evidence to show that in children all tuberculous affections tend towards a fatal termination less uniformly than in adults. It is no doubt advisable to give cod-liver oil, syrupus ferri iodidi, and the like, but in several cases these have failed, and the mercurial application has proved successful.

Malignant Disease of the Peritoneum.—This—the third chronic disease to which the serous membrane of the abdomen is liable—is of considerable importance in several respects.

Anatomy.—It consists in the presence of an immense number of roundish or flattened nodules or small tumours, with which the peritoneal surface is studded over, and which are sometimes isolated, sometimes aggregated together. Often, each little tumour is distinctly umbilicated, and it may send out processes which show a strong tendency to pucker and drag the neighbouring parts of the serous membrane towards it as a centre. In this way, as we shall presently see, the calibre of one or more of the hollow viscera may be very considerably diminished. It is probably by means of a somewhat similar process of contraction that the omentum becomes drawn up and converted into a solid mass, which lies transversely across the abdomen, below the stomach. This induration of the omentum is present in most cases of malignant disease of the peritoneum. In other respects the distribution of the nodules may vary widely. Sometimes the mesentery is covered with them; in other cases it is comparatively free. Often, as Dr Moxon pointed out, the growth is far more abundant on the peritoneum lining the flanks and the diaphragm than elsewhere.

Origin and course.—Malignant disease of the peritoneum is generally spoken of as though it were a primary affection of the serous membrane. But it seldom happens that some one or other of the subjacent viscera is not the seat of a similar growth; and to this in all probability the peritoneal affection is secondary.

As Virchow pointed out, the organs most frequently concerned are the *stomach* and *ovaries*. Out of forty-five consecutive cases of extensive malignant disease of the peritoneum that occurred at Guy's Hospital, in only six were all the viscera free from the same disease. In nineteen the ovaries were affected, and were often converted into large tumours by the growth. In seventeen the stomach was diseased in the same way; seven times without, and ten times with, malignant disease of the ovaries. In three cases the

peritoneal affection appeared to have started from the *uterus*, in two from the *rectum*, in three from the neighbourhood of the *pancreas*. Even these facts fail to indicate the full frequency with which the pelvic organs are diseased in cases of this kind. In two there was a hard mass in front of the rectum; and in several cases (including some of those in which the disease seemed to have begun in the ovaries) the uterus and its appendages were matted together and fixed to the adjacent parts by a large diffused growth in the subperitoneal tissue.

In one instance, which occurred at Guy's Hospital in 1861, the affection of the peritoneum seemed to have started from a cancerous growth in the ascending colon. The omentum formed a solid mass an inch thick, which was spread over the intestines, and reached down to the pubes.

The way in which malignant disease spreads from the stomach or ovaries over the whole peritoneal surface is a matter of much interest. When such disease reaches the serous surface of an organ, it is well known to be capable of infecting the surface opposed to it, without the formation of adhesions between them. Lately we had an excellent instance of this. The uterus had its body affected with cancer, which reached its outer surface. The omentum was long and hung down into the pelvis, so as to touch the uterus, and in its extreme lower end there was a hard mass, resembling the uterine cancer exactly in its character. There was no malignant growth in any other part of the peritoneum. It is probable that such local infection of the omentum is really not uncommon, and forms the starting-point of the remarkable change in this structure already mentioned. The infection of the general surface of the serous membrane probably arises in the course of the movements of the contained organs. It is even possible, to use the words of Rindfleisch, that "the mutual friction of the viscera may detach fragments of the nodules, and carry them hither and thither over the smooth surface of the membrane, until they find their way into some fold or recess, when they give rise to the development of fresh nodules."

Histology. — The microscopical characters and real structure of the malignant nodules doubtless vary in different cases. Virchow is disposed to include a considerable number among the sarcomata, but most other writers speak of them as "cancerous" in the strictest sense of the word. However, they generally yield but little juice from their cut surfaces; and their structure is to a large extent fibrous.

Sex and age.—Unlike tubercular peritonitis, cancer of the peritoneum appears to occur much more frequently in women than in men. Out of the forty-five fatal cases referred to above, only eleven occurred in males. Under the age of thirty this disease is exceedingly rare. Between thirty and forty it is not very uncommon in women, but is very seldom seen in men. In each sex the most numerous cases occur between the ages of fifty and sixty; it is also common between sixty and seventy, and in one instance it was found in a man who died at the age of eighty-two.

Diagnosis.—Clinically, malignant disease of the peritoneum presents itself in different cases with very different symptoms. The growth may, by the contraction and puckering which it causes, so narrow the intestine as to interfere with the passage of its contents, and even to give rise to well-marked ileus (cf. p. 417). More frequently, however, the most marked effect of the disease is ascites. Another character, which was first pointed out by Sir William Jenner, is the occasional presence of a hard mass in the skin

and other tissues round the umbilicus. Probably the growth travels along the connective tissue in the path of the obliterated umbilical arteries or vein.

In other instances, again, the principal symptom of malignant disease of the peritoneum is an increase in size of the abdomen, without any fluid being present. In these cases the growth is a true carcinoma, which has undergone *colloid* degeneration.* All the organs may be enveloped in thick layers of this substance, in the form of round, gelatinous masses, many of which are attached only by the most delicate threads, or seem to be free. At the present time (December, 1887) we have a patient in Guy's Hospital, a woman, the subject of this disease, who has been tapped forty-five times for recurrent ascites, due to enormous masses of colloid cancer in the peritoneum. Characteristic elements have been detected in the fluid, and one minute fragment which left no doubt of the diagnosis. Yet she suffers no pain, except from distension; the other organs are unaffected, and she has continued in apparently good health for more than two years.

ASCITES.†—Apart from chronic inflammatory effusion (whether with or without tubercle or cancer) the peritoneum is also liable to passive dropsical effusion which is known as ascites. This is sometimes first discovered by the physician, when there had before been no suspicion of it; sometimes the patient finds it out for himself, by the fulness and sense of weight in the belly to which it gives rise. An examination of the abdomen, however, is always required to determine the presence of fluid with certainty. For the patient may experience exactly the same sensations from the accumulation of flatus in the bowels, and of fat in the subserous tissue, so that those who seek advice for abdominal dropsy are really free from this complaint.

Physical signs.—Palpation and percussion are both useful in revealing the presence of fluid in the peritoneal cavity.

1. *Palpation* may be employed in two distinct ways. If any solid organ or tumour lies at a little distance from the anterior wall of the abdomen, separated from it by fluid, one can often, by a sudden movement of the fingers, depress the abdominal wall, and push aside the fluid, so as to feel the solid mass beneath in a way that would be impossible if no fluid were present. Thus one may not only detect an enlarged liver, but also at the same time determine the presence of ascites. This procedure is sometimes spoken of as "dipping for the liver;" it requires a little dexterity, and should be carefully practised by the student.

The other method of discovering by palpation whether there is fluid in the peritoneal cavity, is by observing whether *fluctuation* can be felt.

This term, as every student knows, is commonly employed by the surgeon to designate the peculiar elastic sensation which results from manipulation of an abscess or other cavity containing fluid. But the way in which

* Many years ago, when I was a senior student at the hospital, a medical man in the country asked me, during the vacation, to look at a case in which he was about to tap the peritoneal cavity for ascites. I found that although there was very great enlargement and dulness on percussion over the whole abdomen, yet no fluctuation could anywhere be discovered. I happened to remember hearing Dr Wilks describe colloid cancer of the peritoneum, and ventured to suggest that the case was one of this kind, and that paracentesis would lead to no result. During a subsequent vacation I made the autopsy, and found that I had been right.—C. H. F.

† Ascites (*ἀσκίτης* sc. *νόσος*, from *ἀσκός*, a winesack) was distinguished by the Greek physicians, as was also *tympanites* (from *τύμπανον*, a drum), sometimes called the false or windy dropsy.

"surgical fluctuation" is detected is not that which proves the presence of ascites. To understand the latter, we must bear in mind the fact that the walls of the cavity in which the fluid lies are everywhere more or less yielding. Hence, when an impulse is given to the wall of the abdomen at one spot, the fluid can transmit it freely in the form of a wave. If, for example, the left hand be placed on one side of the patient's abdomen, and a tap be then given to the other side with the right hand, the left hand receives a distinct shock. When the parietes are thin, and other conditions favourable, the slightest touch may cause a thrill that can be felt all over the belly. There is perhaps no other physical sign which the tyro recognises so easily as this. If, however, the parietes are massive, and very hard, or loaded with fat, the detection of fluctuation may be difficult. The two hands must then be placed near one another; and a smart blow must be given with one hand, while attention is closely directed to the reception of the impulse with the other. Sometimes the fat in the abdominal walls gives a sensation that might be supposed to be due to fluctuation. To avoid the possibility of error from this source, one may get an assistant to hold a thick piece of cardboard between one's two hands, with its edge pressed upon the surface of the abdomen. In some cases we may fail to obtain fluctuation, although a large quantity of fluid is present; probably the walls of the space containing it are too unyielding on every side for a wave to be transmitted.

It is remarkable what small quantities of fluid can often be detected in the way just described. One might have expected that unless it were present in large amount it would all have gravitated into the loins or into the pelvis (according to the position of the patient), where it would have been out of reach. On the contrary, distinct fluctuation can frequently be felt over parts of the abdomen when the intestines can be proved to lie immediately in contact with the parietes. This depends upon the same principle which has been used to explain the red lines that are observed in the bowels in acute peritonitis (p. 467). When fluid is poured out into the abdominal cavity, it enables the intestines to assume their natural cylindrical form, filling the angles and corners between their convolutions and the anterior wall of the abdomen.

2. *Percussion* is also of service in detecting the presence of ascites, and still more in distinguishing this from some other conditions which resemble it in causing abdominal enlargement. Whenever the amount of fluid is at all considerable, that part of the abdomen which contains it gives a dull note on percussion. But a small quantity, lying among the intestines in the way just described, may fail to affect the natural tympanitic note, and this although it gives rise to distinct fluctuation.

Diagnosis.—For the determination of ascites, however, something more is required than the mere discovery of dulness on percussion, or even of fluctuation. The former might be caused by a solid tumour, and the latter might depend upon a collection of fluid within one of the hollow viscera, or in an adventitious cyst. Cystic disease of the ovary is by far the most important of all the affections that may be confounded with ascites. But there are several other conditions that, as a matter of fact, have been mistaken for it. It is recorded that John Hunter once tapped the bladder in the belief that the patient had abdominal dropsy; and Murchison relates a case in which 480 ounces of urine were drawn off by a trocar introduced midway between the umbilicus and sternum, it having been thought that there was a hydatid

tumour. A arge and elastic tumour may yield physical signs more or less like those of ascites; so may a renal cyst, and, still more commonly, a pregnant uterus.

In the great majority of cases one can readily distinguish an accumulation of fluid in the peritoneal cavity from all these conditions, by noticing which parts of the abdomen are dull, and which are resonant on percussion, particularly if one makes the patient assume different positions in turn. In ascites the fluid, being specifically heavier than the intestines, tends, in the main, to sink towards the more dependent part of the peritoneal cavity; while they may be said to float in it. Hence, when the patient lies upon the back, the bowels fill the umbilical region, and the percussion-note there is tympanitic, whereas in the flanks it is dull. But if the patient is made to turn upon one side, the position of the intestines at once becomes altered; whichever side is uppermost is now resonant, while the dulness on the other side undergoes a corresponding increase. And, when the patient stands upright, the fluid gravitates towards the lower part of the abdomen, which, up to a certain level, becomes uniformly dull. Again, when in ascites the border of the dull region is percussed firmly, the left-hand finger being pressed backwards as much as possible, one can often detect a resonant note from the presence of intestine beneath.

All these characters are wanting when enlargement of the abdomen is due to *cystic disease of the ovary*, or to *pregnancy*, or to *distension of the bladder*. Moreover, all of these rise from the pelvis into the front of the abdomen, pushing the intestines backwards. Consequently, when the patient lies upon the back, the front of the abdomen yields a dull note on percussion.

Thus, then, most cases of ascites present *positive* characters, the recognition of which renders it impossible for a mistake to be made.* Another sign of some value is prominence of the umbilicus, or occasionally the presence of a protrusion there containing fluid. This is very different from the deep depression of the navel in cases of mere *obesity*. Moreover, in the latter case two transverse lines can generally be traced above the pubes and above the umbilicus.

But it sometimes happens that enlargement of the abdomen is really due to the presence of fluid in the peritoneal cavity, and yet that the signs which are distinctive of ascites are wanting, the whole of the front of the abdomen being dull, in whatever position the patient may lie. This may arise in two ways. When the quantity of fluid is very large, the intestines may stretch the mesentery to its full extent, and yet be unable to reach the anterior abdominal wall. But it is probable that this very rarely occurs if the parts concerned are in a normal condition. In the immense majority of cases, when the anterior part of the abdomen is dull in ascites, the reason is that the mesentery has been shortened by chronic inflammation so that it tethers the bowels closely, and prevents their floating.

Under these circumstances the results of percussion may be said to be *negative* so far as concerns the diagnosis between ascites on the one hand, and ovarian disease, pregnancy, &c., on the other hand. We have then to consider what are the *positive* signs of these several conditions.

* I do not make an exception for the case of an ovarian cyst containing air as well as fluid because I can hardly believe that the physical signs would then be really like those of ascites. Alteration in the position of the patient might be attended with changes in the percussion-note; but these would generally be limited to a part of the abdomen. In the instances of this kind that I have seen, a very marked splashing sound has been caused by manipulation of the abdomen: and the outline of the cyst has also been very evident.—C.H.F.

Now, *pregnancy* is distinguished by many indications: the shape of the growth, the state of the breasts, the absence of menstruation, the condition of the cervix, independent of our being able to feel the movements of a living fœtus, or to hear the beatings of its heart or the rush of blood in the placenta.

The positive signs of an *ovarian cyst* are likewise in many cases conclusive. The patient may be able to say that the swelling distinctly began on one side of the abdomen. Again, a careful examination of the swelling will often lead to the detection of a solid substance in some part of it, if it be due to an ovarian tumour, or the outline of the cyst may be felt at some part of its circumference, or at least when the patient draws a deep breath a transverse line (corresponding to the upper border of the tumour) may be seen to descend. On this last sign Sir Spencer Wells lays especial stress, and the same writer also points out that whereas in ascites the greatest circumference of the abdomen is at the level of the umbilicus, in ovarian disease it is often some inches below this; and again, that in ascites the umbilicus usually retains its natural position, being about one inch nearer to the pubes than to the ensiform cartilage, but that in ovarian disease its distance from the pubes is increased.

Sometimes, however, all the positive signs of ovarian disease are wanting, on the one hand, just as are those of ascites on the other hand. It may then be impossible to make a diagnosis. In such cases, when paracentesis has to be performed, the character of the fluid which is drawn off often clears up the doubt as to the nature of the disease. That which comes from the ovarian cyst is frequently viscid and of a dark, greenish-brown colour, quite unlike the secretion of a serous membrane. Its viscidity is said to depend upon its containing a modification of albumen (paralbumin of Scherer) which does not coagulate when boiled with a small quantity of acetic acid. Paralbumin is said never to be present in ascitic fluid, and, on the other hand, the latter often deposits fibrin, which is absent from the contents of an ovarian tumour. Thus it is said that a liquid containing both paralbumin and fibrin must necessarily have been originally secreted by an ovarian cyst, which afterwards burst into the peritoneal cavity. Ovarian fluid, however, is not always viscid, nor of a dark colour; it may be pale yellow, and in fact undistinguishable in appearance from the fluid of ascites.

The fluid drawn from the single cysts which form in the *parovarium** from the remains of the Wolffian body is characteristic,—clear, transparent, and consisting of nothing but water and salts, unaltered by heat. The only liquid like it is the contents of a hydatid cyst, but that contains hooklets.

In cases of ascites, we have next to determine whether it is *passive*, and due to portal obstruction or to general dropsy, or whether it is *active*, the result of inflammation of the peritoneum. It is often supposed that the various forms of chronic peritonitis with effusion are comparatively rare; but at Guy's Hospital they are very frequent, and include at least one third of all the cases of ascites which occur independently of heart-disease or Bright's disease and unattended with jaundice.

Now, so long as the quantity of fluid in the abdomen is not very large, one can generally without much difficulty distinguish ascites caused by obstruction of the portal veins from effusion due to chronic disease of the peritoneum. In cases which come under the first head, the area of dulness

* Known as the organ of Rosenmüller, and corresponding to the epididymis in the male.

in the right hypochondrium is diminished, the intestines float freely towards the anterior wall of the abdomen, there is often a history of intemperance, with the chronic disorders of the digestive organs that result from it, the face is often blotchy, and the urine is high coloured, depositing lithates stained with purpurine. In cases belonging to the second head, the front of the abdomen is very generally dull from retraction of the bowels, there may be no history of intemperance, the patient may have a clear complexion, and (in the case of malignant or strumous disease of the peritoneum) the omentum may be felt hardened and nodulated; or, again, there may be a cancerous tumour of the ovary, or evidence of cancer of the stomach, or a cancerous nodule at the umbilicus.

But whenever the abdomen is greatly distended with fluid,—so that it is universally dull in front, and yet one cannot tell whether the mesentery tethering the intestines backwards is contracted or not—the cause of the effusion is beyond recognition by means of physical examination; and it is in these very cases, when they occur in females, that it is impossible to determine with certainty the absence of cystic disease of the ovary. We have then to judge from the general appearance of the patient, and a knowledge of her habits; but these afford very uncertain grounds for diagnosis.

When paracentesis has been performed, the nature of the fluid may throw some further light upon the question. *a.* The characters of the fluid contained in cystic tumours of the ovary have already been described. *b.* In cases of cancerous disease of the peritoneum, the fluid, although free from viscidity, is often of a brownish colour, or reddened, from the presence of blood. *c.* In cases of simple chronic peritonitis, and also in cases of ascites from disease of the liver, the fluid is generally straw-coloured. *d.* In very rare cases an opaque white fluid has been removed by tapping; its milky appearance is the result of admixture with chyle, some lacteal vessel having opened into the peritoneal cavity by ulceration.

A thorough physical examination of the abdomen should always be made after paracentesis; this often clears up a doubtful case, by leading to the discovery of a solid tumour, or of some disease of the liver or intestines, that could not previously be detected.

Prognosis.—In almost all cases of ascites this is unfavourable. Some of the diseases that give rise to it run, from the first, a course that tends inevitably to a fatal termination; and others, of which the progress is less rapid, do not cause the effusion of fluid into the peritoneal cavity until they have reached a very advanced stage. In cirrhosis of the liver, especially, death occurs in the majority of cases in from six weeks to six months after the detection of ascites. There are, indeed, exceptions to this rule. One patient who recovered from ascites and jaundice under medicinal treatment, remained well for several months, after which the fluid reaccumulated and he returned to the hospital to die. He had been supposed to have some syphilitic affection of the liver, but it turned out that the disease was cirrhosis. A patient lately in Philip Ward (1886), had been four years before in the hospital with hematemesis, coma, ascites, and other signs of cirrhosis which had all disappeared. When, however, recovery takes place from ascites (whether after paracentesis or otherwise) the great probability is that the effusion was the result of either chronic peritonitis or perihepatitis.

In children and young persons a form of ascites is not infrequent which is curable, even when it is the result of tuberculous peritonitis.

It may here be remarked that ascites does not always go on increasing

until the pressure caused by the fluid destroys life. Sometimes the umbilicus, having been first forced outwards, gives way, and allows the contents of the abdomen to escape. Sir William Jenner has recorded a case in which (no doubt from the presence of air as well as fluid) the rupture was attended with a report loud enough to be heard at a distance from the patient's bed. The fluid may continue draining away for a time; but it rarely happens that this postpones for any long period the fatal issue.

Treatment.—Sometimes diuretics may be prescribed with advantage. Of these none appears to be more efficacious than copaiba; but it very often disturbs the stomach, so that the patient cannot continue to take it. The resin is far less likely to disagree, and is equally or more efficient, than the oleo-resin. The acetate or the bitartrate of potass, the spirit of nitrous ether, the compound spirit of juniper, the decoction of broom-tops, the infusion of digitalis, are other remedies of approved value; and a favourite prescription at Guy's Hospital has always been a diuretic pill containing the grey oxide of mercury, powdered digitalis leaves, and powdered squill (of each a grain), which is given night and morning. A useful and pleasant remedy is "Imperial drink," an infusion of lemons with cream of tartar and sugar, of which the patient should drink as freely as possible.

Purgatives, also, are useful, especially those which cause watery discharges from the bowels, such as the compound jalap powder. Murchison recommends an electuary composed of this powder, mixed with confection of senna; and in giving the usual advice that aperients should be taken in the morning, he lays stress upon the reason for this, namely, that otherwise the food which had been newly taken may be swept away by them, and so the nutrition of the patient be interfered with. He also insists on the caution required in the administration of drastic purgatives, lest they should set up fatal enteritis. Our experience at Guy's Hospital fully confirms the importance of this suggestion.

Paracentesis.—In most cases, however, tapping for the withdrawal of the fluid is sooner or later necessary. It should be performed only when the distress caused by the distension of the abdomen becomes insupportable.

The best indication that it is really necessary is perhaps afforded by the state of the breathing, which becomes greatly hurried and very shallow, from the diaphragm being pressed upwards and the lower ribs stretched. The heart also is felt beating above the nipple, and not in the usual place; but it is to be noted that this is often observed when the ascites has by no means reached a very advanced stage.

In performing paracentesis abdominis, the surgeon should use a trocar of moderate size. This is to be introduced in the median line below the umbilicus, it having first been ascertained that the spot selected yields a dull note on percussion, and consequently that the intestines are not in the way. The trocar should be fitted with a long piece of elastic tubing by which the fluid can be carried into a pail placed below the patient's bed, and the entrance of air prevented.

The operation is by no means unattended with risk, immediate and prospective. The patient has sometimes fainted, and even died, while the fluid was escaping through the trocar. It has, however, long been recognised that the cause of such an accident is the sudden removal of pressure from the viscera, and the danger is obviated by having a jack-towel folded round the abdomen before the operation is commenced, which is held by assistants and tightened as the fluid escapes. When the fluid ceases to flow,

the operator removes the cannula with one hand, while with the other he grasps the surrounding integument, so as to prevent the entrance of air into the abdominal cavity. A pad of lint is then placed over the wound, and upon this a few broad strips of plaster.

It occasionally happens that this fails to close the opening into the peritoneal cavity. The fluid then keeps oozing out, and saturates the patient's clothing or the bed on which he lies. Such cases generally terminate fatally within no very long period.

In other instances, again, tapping is quickly followed by peritonitis, which proves fatal in the course of two or three days. Cases of Bright's disease are particularly liable to such a result.

If this be escaped, the fluid almost always begins at once to reaccumulate, being, indeed, poured out much more quickly than before, in consequence of the absence of pressure upon the serous surface. The operation soon has to be repeated, and the patient is again exposed to the same risks as before, with an ever-increasing certainty that the relief will be but temporary and of short duration. Sooner or later he dies, exhausted by the drain of fluid, or by diarrhœa, or by hæmorrhage from the stomach or bowels.

But even though paracentesis abdominis may thus fail to prolong life, it is not therefore useless. It almost always affords great relief to the patient's sufferings, and it should never be delayed when the urgency of the symptoms demands its performance.

Moreover, in some rare cases this operation is as successful as one could possibly wish. There may be no return of the ascites at all; or, as is more often the case, the fluid may be very slow in reaccumulating. Moreover, paracentesis, by relieving the kidneys and veins of pressure, may do much to assist diuretics and increase the flow of urine.

DISEASES OF THE LIVER

BILIOUS DISORDERS AND JAUNDICE

BILIOUS OR HEPATIC DYSPEPSIA—*Disturbance of secretion or elimination of bile—Disturbance in nitrogenous metabolism—Lithæmia—Bilious symptoms—Treatment by diet, drugs, and exercise.*

ICTERUS.—*Symptoms—Tests for bile-pigment and for bile-acids in the urine.*

Simple or idiopathic jaundice: its course, pathology, and diagnosis.

Symptomatic jaundice—Febrile jaundice—from general pyæmia—from portal pyæmia, suppurating hydatid and acute tubercle—Jaundice from cirrhosis—from simple and malignant obstruction of the ducts—from tumours, &c.

Jaundice from gall-stones—Their structure and varieties—Symptoms and events of biliary colic.

Effects of permanent jaundice on the liver—Effects on the bowels and the skin—Pathological theories of jaundice.

Treatment of jaundice—primary, symptomatic, and calculous—of permanent jaundice—Treatment of other effects of gall-stones.

BILIOUS DYSPEPSIA.—Beside distension and tightness in the epigastrium after meals, dyspeptic persons occasionally experience a dull aching in the right hypochondrium, with a sense of weight and fulness there, which is often greatly increased by lying on the left side, and is also worse after meals. Another pain, of which such patients sometimes complain, is situated in the right shoulder. The conjunctivæ have a slightly yellow tint. The bowels are confined, while the urine is generally scanty and high coloured, and as it cools deposits lithates of a bright pink colour.

Such complaints are apt to occur in persons of the middle period of life. Those persons are particularly liable to them who take but little exercise, or who eat or drink to excess. On the other hand, they are often at once removed by a few days' shooting or hunting, or by any other kind of bodily exercise which is sufficiently attractive to induce men to give up sedentary habits.

Probably most people belonging to the richer classes, who live in cities, take more food than they require, and many of those who live in the country do the same. Men get into certain ways as regards diet when they are young, or when they are leading active lives; but later on, when their circumstances are altered, they are apt to forget that their habits ought to be altered likewise. Some kinds of food are much more likely than others to produce the symptoms above described. The most injurious are generally fatty and saccharine matters, and certain alcoholic drinks, especially those which contain much sugar. All malt liquors are apt to disagree with people who suffer in this way, but particularly porter and the stronger kinds of ale. Among wines, port, Madeira, champagne, and brown sherry are especially

harmful; and brandy and sweet liqueurs among spirits. The evil effects are most marked in hot climates, and in the warmer seasons of the year.

Theory of this condition.—Patients are wont to speak of the symptoms under consideration as indicative of a "torpid state" of the liver, and to think that they are caused by a deficiency in the amount of bile secreted by that organ. The correctness of this opinion was formerly supposed to be established by the fact that the complaint is often easily removed, at least for a time, by certain medicines (particularly mercurials) which at the same time bring away from the bowels a considerable quantity of semifluid or fluid fæces, apparently loaded with bile. This explanation was once taught by medical authors. But for many years past physicians have been aware that there are great difficulties in the way of its acceptance. In the first place, a large number of experiments have been made to determine whether mercury, and the other drugs above alluded to, possess the power of increasing the amount of bile secreted; and no such power has been discovered.

Another way of explaining the complaint is to attribute it to congestion of the portal system, and the remedies for it are those supposed to act by emptying the overloaded blood-vessels. It is well known that digestion is always accompanied by an augmented flow of blood through the liver. Hence it is not a far-fetched hypothesis to suppose that the liver becomes permanently congested in people who eat and drink too often, too much, or too richly.

There are, however, certain facts of the case which this theory leaves altogether unexplained, particularly the presence of excessive quantities of lithates (urates) in the urine of these patients, and the superiority of mercurials over other purgative drugs which ought to be no less efficacious, if the indication for treatment were to relieve congestion.

The late Dr Murchison, in his Croonian Lectures (1874), supplemented the ancient theories upon this subject by referring to certain facts which appear to show that the healthy liver plays a very important and perhaps the principal part in carrying on those chemical changes by which albuminous substances are disintegrated in the body, and which normally result in the production of urea. Some of these facts have been acquired by physiological experiments upon animals, but others are pathological, particularly the fact that urea is absent from the urine in yellow atrophy of the liver, in which disease the hepatic cells are destroyed.

He suggested, therefore, that in the cases now under consideration there is not only a defective secretion of bile, but also an interference with the normal processes by which albumen is disintegrated in the liver with the result that instead of urea (CH_4N_2O), lithic or uric acid ($C_5H_4N_4O_3$), a less oxidised body, is formed. He further supposes that mercury has a double action. On the one hand, whether or not it increases the amount of bile secreted by the human liver, he points out that it certainly increases the quantity which is eliminated from the bowels. Less of the biliary ingredients is reabsorbed, and the result is that the blood is freed from their presence in excess. On the other hand, he imagines that the drugs in question may have a special power of promoting or in some way influencing the disintegration of albumen.

The significance of these views is by no means limited to the class of cases which we are now considering; for the substitution of uric acid for urea as the final product of disintegration of albuminous substances within the body is otherwise a most important matter. The comparative insolubility of the lithates of soda and potash prevents their being readily excreted by the kidneys like urea. Hence, they accumulate in the blood, and a con-

dition arises which Murchison proposed to term *lithæmia*. The urate of soda is very apt to crystallize out into the cartilages of the joints and elsewhere; and this is *gout*. Again, even when the lithates have been separated from the blood by the kidney, the acid is often deposited from the urine either in the renal pelvis or in the bladder. We have then the common form of *gravel*, and those important varieties of *calculus* of which lithic acid and lithates are the main ingredients.

Symptoms. — A bitter or "coppery" taste is sometimes experienced in the mouth, especially in the morning; this, Murchison suggests, may possibly be due to the presence in the blood of tauro-cholic acid. Intestinal hæmorrhage is not unfrequent, especially in patients beyond middle age. Each attack is usually preceded by a feeling of oppression and heaviness, or by creeping sensations, and more rarely by severe neuralgic pains, suggesting the presence of gall-stones. Aching in the limbs, again, and lassitude, coming on about an hour after a full meal, are complained of by persons affected with lithæmia, and sometimes an irresistible drowsiness. Severe cramps in the legs and in different parts of the body may be another indication of the same condition. Murchison says that they often come on during the night, particularly in cold and damp weather, and quotes from Bence Jones these two remarkable instances.

The first occurred in a gentleman aged forty, who for years had constantly had deposits of lithic acid and lithates in the urine. He then became subject to attacks of violent pain in the stomach, coming on an hour or more after a late dinner. The pain was intermittently spasmodic; its greatest intensity was reached in half a minute, it then relaxed, to return as badly as before in two minutes. When about an hour had passed, the suffering gradually subsided, leaving tenderness on pressure and irritability after food for two or three days. After the attack the urine always deposited crystals of uric acid. The complaint had lasted for several months, but under care and diet and the use of alkalies it entirely ceased.

The second case is that of a patient, also the subject of lithæmia, who was seized with violent cramps in the rectum, coming on six or eight hours after food, and lasting from half an hour to an hour. The same treatment was completely successful in this case.

Another frequent symptom of hepatic disorder is *headache*. This is commonly a dull heavy pain, seated in the forehead, or more rarely in the occiput. It generally comes on when the patient first wakes in the morning, and it may either quickly pass off or last the greater part of the day. This kind of headache usually follows some indiscretion in diet or is preceded by constipation of the bowels. It must be noted that it is quite distinct from what are commonly called *bilious* or "sick headaches." They have been described among the diseases of the nervous system, under the name of migraine (vol. i, p. 779). Still one cannot deny that typical migraine is also sometimes more or less due to lithæmia. Dr Liveing mentions a family in which the father had gout, his son migraine, and his son again gout; and Trousseau speaks of having seen migraine and gout alternate in the same patient, an attack of gouty inflammation in certain joints being followed by the cessation of sick headaches which had previously been of frequent occurrence. Indeed, the symptoms now described are those which were formerly ascribed to "suppressed" or "latent" gout.

Another effect of lithæmia is *giddiness*. Dr Wilks says that, if due to digestive disorder, swimming in the head is especially apt to come on when

the patient stoops or lays his head upon the pillow, and that it often passes off when he assumes the erect posture.

Another symptom, first described by Graves, which appears to be caused by lithæmia, is *grinding the teeth.* He has related some remarkable cases of this kind. The affection is described as depending upon a disagreeable uneasy sensation referred to the teeth, and relieved for the moment by grinding them together. In one case, that of the Countess of E—, this habit was so confirmed that she practised it all day, and consequently was obliged to give up society for several years before her death. The teeth became worn down to their sockets. During sleep the grinding entirely ceased, so that the affection was altogether different from that which is so common a symptom of irritation of the brain, and especially of tubercular meningitis. Graves states that all the cases he had seen have been in patients of confirmed gouty habit.* He was never able to discover any means of alleviating this troublesome complaint.

Possibly *convulsive attacks* may sometimes be due to the same cause. Murchison relates the case of a gentleman who had long suffered from hepatic derangement, and who became subject to severe spasmodic twitchings in his legs. These were followed on three occasions by epileptiform seizures. A little later he had a first attack of gout, and afterwards he suffered frequently from that disease, but he had no return of the convulsions or muscular twitchings.

Noises in the ears are a more frequent effect of lithæmia. One patient, says Murchison, has the feeling of a strong wind blowing into his ear; another compares the noise to that of flowing water, or describes it as a singing or buzzing; while in yet another the sound pulsates with the beats of the heart.

Then, again, there may be *sleeplessness.* As has already been stated, lithæmic patients are often heavy and drowsy after a full meal and fall asleep at once when they go to bed, but after three or four hours they often awake, and they may then lie awake for hours or keep dozing off and waking again after unquiet dreams. Such patients never sleep so well as after a dose of calomel or blue pill. Depression of spirits and irritability of temper are well known to be frequent effects of the same cause.

Hepatic and particularly flatulent dyspepsia causes *palpitation* and fluttering of the heart, with intermission or, more rarely, irregularity of the pulse.

Lastly, Murchison enumerates chronic catarrh of the fauces, gouty bronchitis, and spasmodic asthma, as results of lithæmia.

Treatment.—Formerly many cases of headache, giddiness, and dimness of sight, which are now easily cured by quinine and similar remedies, were submitted to severe mercurial treatment with injurious results. But of late years that kind of practice has fallen into disuse. The younger school of physicians, unable to prove that mercury is capable of increasing the amount of bile secreted by the liver, have forgotten that this after all is not the whole question. Hence they have given up the use of remedies

* Some observers have supposed that the mere fact of the teeth being ground down, so as to show the dentine in section, is to be regarded as evidence of a "gouty diathesis" or of lithæmia. According to Mr Moon, however, there are several causes which may lead to this condition. One of these is that formation of the jaws which gives what is termed an edge-bite; another is the absence of enamel at the summits of the teeth. Early wearing down of the teeth often depends, not on their being too soft or on a gouty diathesis, but on the food being too hard. In races who live on coarsely prepared flour and hard vegetable food, the teeth are ground down to the gums, as in old horses.

by which the class of cases just described are for the most part readily and safely relieved. For it is unquestionable that three or four grains of blue pill, with as much of the compound colocynth pill, followed (if necessary) by the traditional haustus sennæ or some less nauseous aperient, do great good in cases of "torpid liver." Podophyllin has been very much recommended for cases of this kind, but the slowness and uncertainty of its action are great objections to its use. A much better substitute for blue pill is euonymin, in doses of two or three grains.

The popular "cure" for a torpid liver, suppressed gout, hepatic dyspepsia, or lithæmia, is a visit to certain German watering-places, particularly Püllna, Friedrichshall, and Carlsbad, all of which contain sulphate of soda, and the former two sulphate of magnesia also. The proper dose is about five ounces of Püllna water, seven ounces of Friedrichshall water, eight or ten ounces of Carlsbad water. The necessary quantity should be mixed with a little hot water and taken the first thing in the morning, or about an hour before breakfast. It secures a free action of the bowels, and with this advantage over vegetable purgatives, that there is less constipation afterwards, nor does the dose always require to be increased. On the contrary, the quantity taken may sometimes be gradually reduced without any diminution of its efficacy. After a course of about six or eight weeks the remedy should, however, be omitted, at least for a time; but patients sometimes continue to take it regularly for four or five years.

A most instructive lecture on this subject by Sir Henry Thompson appeared in the 'Lancet' for Jan. 13th, 1872. He recommends Friedrichshall water in persons of "a uric acid diathesis." Many patients, however, perhaps chiefly women, find they do better with Hungarian bitter water, Hunyadi Janos; and many others have no reason to forsake the cheaper saline laxatives of Epsom, Cheltenham, Seidlitz, or Rochelle.* Whichever is selected, it should be taken before breakfast in not less than a tumbler of warm water.

Regulation of the diet is of the utmost importance, particularly as regards alcoholic liquids. The stronger wines, champagne, and malt liquors should be prohibited; and the patient should be limited to a very moderate allowance of a light but sound Bordeaux or Rhine wine with his principal meals, or of brandy or whisky largely diluted with water.

In most cases of this kind the mineral acids (particularly the nitro-hydrochloric), with gentian and taraxacum, appear to be very serviceable; calumba or chiretta are also recommended.

A nitro-hydrochloric acid bath was advocated by Sir Ranald Martin for the "liver cases" of India. It is made by mixing two ounces of strong hydrochloric acid and one ounce of strong nitric acid with two gallons of water in a glazed earthen or wooden vessel, at a temperature of 96° to 98°. The feet only are placed in the bath, while the inner side of the thighs, the right hypochondrium, and inner side of the arms are sponged with the liquid. The process is repeated each night and morning for half an hour at a time.

* Most laxatives are better in combination. This is probably one advantage of natural waters over the sulphate of magnesia, or of soda, or the tartrate of potash and soda, alone. But it is easy to imitate the waters of spas by adding a little carbonate of soda and common salt to the combined sulphates. What is sold as citrate of magnesia often consists of bicarbonate of soda and tartaric acid. The combination known as "Lamplough's Pyretic (or Antipyretic?) Saline" contains the same ingredients with about 2 per cent. of chlorate of potash; "Eno's (so-called) Fruit Salt" adds to these sulphate of magnesia and sugar. They are probably less injurious than patent purgative pills and lozenges and "Indian" tamar, which usually contain aloes, jalapin, or gamboge.

The patient should also be made to take exercise. Of all kinds horse-exercise is the best. Rowing is also excellent, and any exertion which produces deep breathing and free sweating. Walking, however good in other ways, is perhaps least useful for this hepatic form of dyspepsia. A quarter of an hour's game at rackets, for instance, is far more beneficial to most persons than an hour's walk. This advice applies particularly to persons much engaged in business which keeps them on their legs all day. A long walk only makes them more tired, and unable to digest the heavy meal which their sense of exhaustion prompts them to eat; whereas a much shorter time, spent in riding or rowing, exercises the whole body; and after a short rest, or, if needful, a quarter of an hour's sleep, they can enjoy dinner with a zest "that after no repenting draws."

Icterus.*—When the colouring matter of the bile fails to escape by the natural passage, it is carried into the general circulation by the hepatic veins, and is deposited in the skin and other parts, so as to give the patient a yellow colour. This constitutes *jaundice* or *icterus*.

A large number of the diseases of the liver are attended with jaundice, but by no means all. Moreover, it is present when there is no other reason to suppose the liver to be affected: not only in yellow fever and certain remittents, but also occasionally in typhus, in relapsing fever, and in pneumonia.

Jaundice therefore is itself no *disease*, due to a single cause, and capable of being treated with drugs without further investigation of origin. On the contrary, it is one of the best examples of a morbid condition, which appears an entity to the public, as it once appeared to physicians, but which we now regard as only a *symptom*, to be traced to its cause whenever we can do so.

General symptoms.—The parts that most obviously display the yellow colour are of course those which are most exposed to view, and which at the same time are naturally pale. Even in the skin, the redder parts are far less decidedly altered in appearance than those which are less florid, and in the superficial mucous membranes a similar difference is still more striking. For whereas even in extreme degrees of jaundice the lips and lining of the cheeks show comparatively little change, the yellow hue is exceedingly well marked in the conjunctivæ, through which in health the pearly-white sclerotic beneath is plainly visible. Indeed, the natural whiteness of the eyeballs enables us to recognise in them slight alterations of colour which are quite inappreciable elsewhere. Just as we place a sheet of white paper beneath any transparent substance, the colour of which we desire to scrutinise, so we always look to the conjunctivæ for the first signs of icterus, or for the faint indications of retention of bile-pigment too slight to produce general jaundice.

Whenever jaundice is present in any considerable degree of intensity the internal parts are affected as well as those on the surface of the body. All the paler mucous membranes have a marked yellow colour. The same is true of the connective tissue generally and of the serous membranes; and any collections of fluid within the serous cavities acquire a deeper yellow hue. The lungs and the kidneys, and almost all other organs which are not

* Ἴκτερος, the golden oriole, which by the doctrine of similars, was believed to cure a jaundiced person who caught sight of the bird.—*Fr.* Ictère, Jaunisse. In English the disease is popularly, by a natural tautology, called the yellow jaundice.—*Germ.* Gelbsucht.

so red as to conceal their yellow tint, are evidently jaundiced. The liver displays the same colour, often in an extreme degree. It has been said that the brain participates in this change, which its natural whiteness would of course make very evident. But our observations in the deadhouse at Guy's Hospital confirm those of Dr Moxon, who states that the brain shows no abnormal colour, even in advanced jaundice. The transparent humours of the eye are sometimes yellow, and sometimes not.

The colour of the skin in jaundice varies according to its intensity and its duration. If the natural escape of the bile be suddenly completely arrested, the body may quickly assume a deep orange hue. But in many cases this is more gradually developed, the tint being at first a pale sulphur yellow. The whole cutaneous surface does not necessarily present the same depth of jaundice; this is generally more marked in the face and upper parts than in the legs. When jaundice has existed for a considerable length of time the colour frequently undergoes a change; it is no longer yellow, but becomes greenish, and after a time passes into a dark olive colour. This change doubtless corresponds with the alteration which bile itself always undergoes when exposed to the air in assuming a green colour, namely, conversion of bilirubin (the principal colouring matter of the bile) into biliverdin, which again after a time turns brown, passing into what is called choletelin. Persons in whom the skin assumed the dark green colour above referred to were formerly said to have "black jaundice,"* and it was supposed to be an indication that the hepatic disease was of a cancerous nature. We now know that it means only that the jaundice has lasted for a long time, but it is true that such cases are generally cancerous. Probably for the production of a green or "black" tint in jaundice it is necessary that the flow of bile through the ducts should be *completely* arrested. If this be not the case the skin remains yellow, however long the jaundice may last.

It may well be supposed that there is seldom much difficulty in discovering whether a patient is or is not jaundiced. Only a very careless or inexperienced physician would mistake for it the greenish-yellow hue of chlorosis, or the yellowish waxen tint often seen in cancerous diseases of the abdomen, or the dusky sallow look of malaria. Nor ought anyone to confound with the olive-green tint of black jaundice the brown or bronze colour associated with disease of the adrenals, although it must be remembered that before Addison's discovery patients were supposed to be jaundiced. In all the conditions just mentioned, but particularly in the last, the conjunctivæ retain their natural pearly-white appearance. One must bear in mind that the yellow tint of jaundice is invisible by gas- or candle-light.

Cases are, however, sometimes met with in which it is difficult to be sure whether a slight degree of jaundice is or is not present. The doubt then generally lies between jaundice and idiopathic (so-called pernicious) anæmia. In that disease the conjunctivæ often look yellowish, but this, as in other cases, particularly in old persons, is really caused by the presence of a little fat in the submucous tissue. The yellow colour is partial, instead of being uniformly distributed over the whole surface of the conjunctivæ, the shape of the lobules is seen, and the vessels which supply them.

Several of the secretions of the body contain biliary colouring matter in

* In the humoral pathology of Galen, while the yellow bile was secreted by the gall-bladder, the black bile was formed by the spleen. Hence the *dyscrasia* or ill temperament due to excess of yellow bile was called choleric or bilious, that due to excess of black bile, melancholic or atrabilious.

jaundice. The sweat is yellow, so that the patient's linen is often much stained under the armpits and at those parts where there has been much perspiration. The milk was noticed to be yellow by Bright and others. On the other hand, the secretions of the various mucous membranes remain free from bile-pigment. The saliva is colourless, and although the patient not unfrequently complains that he has a bitter taste in his mouth, there is no reason to suppose that this is due to the presence of bile in any of the fluids poured into the buccal cavity. Dr Fenwick has pointed out that in cases of jaundice the sulphocyanide of potassium, normally present in the saliva, is not to be found. Even the gall-bladder and the ducts of the liver itself (as we shall presently see) secrete a colourless mucus. That the intestinal mucus and succus entericus contain no bile-pigment is evident from the fact that the fæces are of a greyish-white colour, or (to use the common expression) "clay coloured."

Wherever any diffusible substance is present in the blood in quantity larger than natural, its removal is effected by the kidneys rather than by any other organ. Hence we might expect than in jaundice the urine would contain more of the biliary colouring matter than any other secretion. The colour of the urine, however, may vary from a yellow, scarcely deeper than natural, to a dark brown, a greenish brown, or a black, so intense that one can recognise its colour only by looking at the margin of the fluid, or pouring some of it out in a thin layer, or making it froth. The presence of bile-pigment in the urine is a necessary part of jaundice. There is, indeed, one condition in which the secretion may for a short time have its natural appearance, although the patient's skin is still of a deep yellow colour. This is when the cause of the jaundice has been suddenly removed, particularly if it has lasted for some time. The bile-pigment then ceases to circulate in the blood, and the kidneys no longer excrete it in any quantity; but the skin does not at once give up all the colouring matter that had been deposited in its tissues, and it remains for a few days yellow. With this single exception the urine contains bilirubin when there is jaundice. Indeed, we obtain sometimes more delicate indications of slight icterus from the urine than from the conjunctiva. When jaundice is commencing, the colouring matter may be detected in the urine before the skin or the eyes betray its presence.

Test for bilirubin.—The presence of bile-pigment in the urine is not to be assumed from the colour alone. We have a chemical test which is capable of detecting it in the most minute proportion. This is known as Gmelin's test. It consists in the addition of fuming nitric acid (containing nitrous acid) to a small quantity of the urine. This causes a beautiful play of colour if bile-pigment be present. A good way of employing the reagent is to pour a drop or two of the urine on the flat surface of a white plate, and then carefully to add to it a single drop of the nitric acid. Round the drop of acid a series of colours is developed, rapidly passing through the shades of green, blue, and violet, into red, and finally becoming a dirty yellow. Neubauer and Vogel recommended, for the detection of small quantities of bile-pigment in urine, that the nitric acid (which must not contain too much nitrous acid) should be poured about an inch high into a conical glass, and that a little of the urine should then be carefully spread over its surface by means of a pipette. The play of colour begins where the fluids come into contact with a beautiful green ring, which gradually extends upwards, and at its under surface exhibits a blue, violet, red, and lastly a yellow ring. Mere darkening

of the urine by oxydation of the natural urobilin of urine into a reddish-brown colour is a familiar effect of nitric acid quite distinct from that just described.

When nitric acid is added to urine in the way described above, red and violet rings may be produced by another substance, of which a small quantity is present even in healthy urine, and which is increased under various pathological conditions. This was once called uroxanthin, but a better name is *indican*, for it is the same principle which, when obtained from the indigo plant, has long been known as the mother substance of the indigo pigments.*

The most delicate of all methods of applying Gmelin's test for bile-pigment is to shake large quantities of the urine successively with chloroform. This extracts any of the pigment that may be present, and when nitric acid is afterwards spread over the chloroform, the reaction takes place.

Test for bile-acids.—There has been considerable difference of opinion with regard to the question whether the glyco- and tauro-cholic acids of the bile acid are excreted by the kidneys in jaundice, and whether this is the case in some forms of jaundice and not in others.

The principal chemical test for the biliary acids, or rather for the cholic acid which they both contain, is known as Pettenkofer's. It consists in the admixture of a few drops of syrup, or a few grains of sugar (either sucrose or glycose) with the liquid suspected to contain biliary acids, and the subsequent addition of strong sulphuric acid, precautions being taken to prevent the development of too great heat. A beautiful violet colour appears if the acids of the bile are present. But Pettenkofer's test cannot always be satisfactorily applied to urine unless the urinary pigments are first separated, for if this be not done the sulphuric acid may blacken the liquid, so that no violet colour can be seen. Still there is no great difficulty in applying it to demonstrate the presence of cholic acid when bile has been purposely added to urine. It is very doubtful if the presence of the bile acids has ever been observed in urine without their having been so added.

Moreover, there are theoretical considerations which have been overlooked, and which render it improbable that any considerable quantity of the biliary acids should be found in the urine in any case of jaundice. It seems to be forgotten that in health comparatively small proportions of those acids are discharged from the body. According to Bischoff not more than a quarter of the amount of biliary acids poured into the intestine by the liver passes away in the fæces, and even this has undergone important chemical changes. Evidently, therefore, the biliary acids subserve some further purpose in the economy, and it is probable that they are reabsorbed into the blood. But if this be the case we cannot expect to find them excreted by the kidneys as waste products in a case of jaundice.

Simple or Idiopathic Jaundice.—In a large proportion of cases of jaundice one can ascertain scarcely anything, whether by examining the patient or by asking him questions, beyond the facts that his skin and conjunctivæ are of a deep yellow colour, that the urine contains much bile-pigment, and that

* Many observers have endeavoured to attach a clinical significance to the presence of indican in excessive quantity in the urine, but their labours have not as yet been successful, and therefore it will not be necessary to make any further allusion to this substance. It is frequently present in excess in cases of intestinal obstruction, also in cancer of the liver or other abdominal viscera, and in certain forms of dyspepsia accompanied by constipation.

the fæcal evacuations are clay coloured. There is not, nor has there been, any pain or uneasiness in the region of the liver. Very often the patient says that he feels perfectly well, and would not know that anything was wrong with him but for seeing his yellow face in the glass. After a variable period the jaundice subsides. The first sign of improvement is generally that the motions return to their natural colour; the urine then soon ceases to contain bile-pigment, and a few days later the skin and conjunctivæ regain a healthy appearance. This favourable change often takes place about the twenty-first day; but in some cases it occurs earlier than this, and in other cases very much later. At no period of the complaint can one generally make out that the liver is enlarged, whether by palpation or by percussion; from beginning to end there is nothing to throw light on its cause. For a case of this kind "simple," "primary," "idiopathic," or "benignant," seem to be suitable adjectives.

Slow pulse.—There is, however, one symptom which is sometimes observed in this but in no other form of jaundice—an extreme slowness or infrequency of the beats of the heart. The pulse occasionally falls to 50, 40, or even 20 in the minute. It was found in some experiments upon animals that the pulsations of the heart were much reduced in frequency by the injection of the salts of the biliary acids into the circulation.* It was therefore supposed that the blood perhaps contains these acids in the cases of jaundice under consideration. But chemists have hitherto failed to discover these substances in the urine, whether in this or in other forms of jaundice; and since they are readily diffusable, one cannot suppose that it is possible for them to accumulate in the circulatory fluid in sufficient quantity to affect the heart, without being freely excreted by the kidneys. A very important question in regard to cases of jaundice in which the pulse is slow is whether the prognosis should be modified on that account. One is apt to be alarmed at it, and to dread the supervention of certain dangerous cerebral symptoms. But there appears to be no ground for those apprehensions.

Pathology.—This is still obscure. The complete absence of bile from the fæces in this form of jaundice affords a presumption that there is some obstacle to its flow; and the common theory is that simple jaundice depends upon *catarrh* of the larger bile-ducts. It is believed that their lining membrane is swollen, and that mucus is secreted, which obstructs the channel. One difficulty is that patients never die from, and rarely during, this idiopathic and benignant form of jaundice, so that we have little or no knowledge of its morbid anatomy. Another is that we have no corresponding instances of spontaneous catarrh of a duct with obstruction and reabsorption of the secretion in the case of the ureter or urethra, the salivary ducts and other glands. A more probable suggestion is that catarrh of the duodenum obstructs the oblique and narrow passage of the duct through the walls of the gut, but here the difficulty is that the jaundice does not more constantly follow what is probably a frequent disorder. Moreover, it would have seemed probable that chronic catarrh would have produced permanent jaundice, as chronic obstruction in the nasal duct produces permanent epiphora. Again there is no evidence or likelihood of catarrh of the gall-ducts when icterus accompanies pneumonia.

Moreover, in acute yellow atrophy of the liver the ducts are always

* Röhrig, 'ü. d. Einfluss d. Galle auf. d. Herzthätigkeit,' 1863. See also a paper by Dr Legg ('Proc. Royal Soc.,' 1876) with references to Traube's observations, and a fuller account with tracings in his work on 'The Bile, Jaundice, and Bilious Diseases,' p. 204.

found after death to be pervious and empty. But it is difficult to suppose that when a case of this kind begins as one of simple jaundice it is at first due to a catarrhal inflammation of the ducts, and that this afterwards subsides without leaving any indication of its having ever existed.

Lastly, icterus sometimes follows directly upon the shock of some mental emotion, which is generally of a depressing character. Sir Thomas Watson mentions the case of a young medical friend of his who became jaundiced in consequence of his anxiety about an approaching examination before the Censors' Board of the College of Physicians; and he refers to another case in which an unmarried woman, on its being accidentally disclosed that she had borne children, became in a very short time yellow. Almost every medical man has seen similar instances. Murchison places cases of this kind in an entirely different category from those which he attributes to catarrhal inflammation of the larger bile-ducts; but beyond the fact that the former are caused by mental emotion, while the latter are not, no differences can be found between them, either in their symptoms or in their course.

The fact that simple jaundice is frequently the direct result of mental emotion presents a difficulty in the way of accepting the current theory of its pathology. This question, however, is one which may be most conveniently discussed further on.

It has occasionally happened that an affection having the characters of simple jaundice has prevailed *epidemically*. Several instances of this were collected by Frerichs. Murchison mentions a remarkable outbreak of the same kind which occurred at Rotherham. In 1862 this town was visited by enteric fever, which proved very fatal. Early in the following year jaundice became epidemic. It is said that in February no fewer than one hundred and fifty persons were suffering from it, and there was this curious circumstance, that none of those who were attacked had passed through the fever.

Diagnosis.—The characters which distinguish "simple" jaundice from idiopathic cases in which jaundice is a symptom of some grave malady are chiefly negative; and one might expect that the diagnosis would be somewhat uncertain, since in cases of cirrhosis, of cancer, and even of gall-stones, jaundice is not very rarely the first symptom, preceding all the more especially characteristic symptoms of these several diseases. But whereas they seldom occur in young subjects and in those who have hitherto enjoyed good health, simple jaundice seems particularly apt to attack such persons. Hence it does not often happen that a mistake is made, and that jaundice, supposed to be idiopathic and benignant, proves due to a more serious affection. Practically the only serious difficulty to be considered is the possible, though happily very rare, supervention of acute atrophy of the liver. This important disease will be considered in the next chapter.

Symptomatic Jaundice.—As a secondary complication in some of the *specific fevers* jaundice is not infrequent. This is well known to be the case in "yellow fever" (vol. i, p. 358). Again, in "relapsing fever" jaundice is a frequent and not necessarily an unfavourable symptom (*ibid.*, p. 155). It sometimes, though very seldom, occurs in typhus, and in this disease almost every patient who becomes jaundiced dies. In enteric fever and scarlatina jaundice is extremely rare.

Another disease, attended with fever, in which jaundice may occur as a complication is *acute basal pneumonia*. This has been supposed to be due to an extension of inflammation through the diaphragm to the upper surface of the liver,—an almost absurd notion, for there is no sign of inflammation of the diaphragm, pleura, or peritoneum between liver and lung; pneumonia cannot "extend" to anything but pulmonary tissue, and even if inflammation does attack the liver, it does not in itself cause jaundice. At least one case of this kind has been observed at Guy's Hospital in which the *left* lung was the seat of the inflammation.

General pyæmia is often accompanied with jaundice; indeed, a slight yellowness of the skin is one of its most frequent symptoms. Some years ago Dr Wilks investigated the question whether those cases of pyæmia in which abscesses occur in the liver are or are not particularly liable to be accompanied by jaundice, and he came to the conclusion that the local disease had nothing to do with the production of this symptom. Indeed, in most of the febrile diseases of which jaundice may be a symptom, it is probably dependent upon changes in the blood or elsewhere, and not upon any morbid state of the liver.

Portal pyæmia.—Besides these general maladies, there are certain diseases of the liver itself which are attended with fever, often with rigors, and local pain with tenderness, and which may also give rise to jaundice. Icterus is a rare and accidental symptom in cases of tropical abscess. But it often accompanies a special form of suppuration in the liver, which has been termed purulent *pylephlebitis*, or inflammation of the branches of the portal vein and of the connective tissue in which they are embedded. The following is a striking case of this kind. A man aged thirty-seven was admitted into Guy's Hospital under the care of the late Dr Barlow, exceedingly ill with jaundice, fever, and delirium; and he died in two days. He had been quite well a week before, except that he suffered from stricture of the rectum. The liver was very large, and its tissue was suppurating throughout. The branches of the portal vein were all distended with soft thrombi of a brownish colour. The main trunk of the vein contained a dirty-looking fluid. The inflammation of the liver was evidently due to the absorption of some unhealthy material by the veins ramifying in the coats of the rectum, which was extensively ulcerated. Not rarely suppuration of the portal canals throughout the liver is excited by the presence of gall-stones in the ducts. The possibility of this occurrence must never be forgotten in any case of biliary colic in which febrile symptoms show themselves, although (as will presently be stated) a high temperature may be present without any other evidence of inflammation having been set up.

Other causes of suppurative pylephlebitis are:—Ulcerative affections of the stomach or bowels suppuration of the spleen, suppuration of the mesenteric glands, and the penetration of one of the veins which go to form the portal trunk by foreign bodies; in a case quoted by Frerichs, a fishbone had entered the inferior mesenteric vein.

There may be not only jaundice in pyæmia without hepatic abscesses, but also hepatic abscesses without jaundice. Thus it was frequently absent in thirteen cases of "Multiple Small Abscesses of the Liver," recorded by Dr Carrington in the 'Guy's Hosp. Rep.' for 1882, p. 375, ten of which were due to pylephlebitis. In only two was it a marked symptom.

An inflamed hydatid cyst in the liver may set up suppuration along

the portal canals within the organ, which, in this case, however, seems to depend upon inflammation of the branches of the bile-ducts rather than those of the portal vein. The explanation of this lies in the fact that a suppurating hydatid very generally communicates directly with a branch of the bile-duct, often of considerable size. Hence membranous portions of the hydatid, or of its capsule (detached by sloughing), often enter the bile-duct in these cases and may obstruct it. An interesting case of this kind occurred in Guy's Hospital a few days later than the case of pylephlebitis mentioned just now. The liver was found after death to contain a suppurating hydatid cavity which held three pints of fluid. The bile-ducts throughout the organ were suppurating, and the main canal was obstructed by a large piece of detached membrane rolled up into a cylinder.

Tubercle.—Another but a very rare cause of febrile jaundice is *acute tuberculosis* of the liver. An instance of this occurred to Murchison in a woman forty years of age. Another case was observed some years ago at Guy's Hospital. A man, aged thirty-seven, died with febrile symptoms of typhoid character. The history was imperfect, but he was stated to have had jaundice only five days. After death the liver was found to be full of tubercles, there being as many as fifty to the square inch. There was also pneumonia of the left lung.

Jaundice from cirrhosis of the liver.—Writers generally state that jaundice is seldom caused by cirrhosis, and it is the fact that in the majority of cases this affection runs its whole course without marked icterus. But there is, nevertheless, a considerable minority in which jaundice is present, and not rarely it constitutes the chief symptom of the disease. Among one hundred and thirty cases occurring in the *post-mortem* room of Guy's Hospital, in which the liver was found after death to be cirrhosed, jaundice existed in thirty-four, and in ten it was deep or intense. During this period, however, there were examined in the same place only some sixty other cases in which jaundice was a principal symptom. Thus, among the causes of jaundice, cirrhosis of the liver is far from taking the insignificant place usually assigned to it.

The fact is that the frequency with which cirrhosis of the liver itself occurs is far in excess of that of the other serious diseases that give rise to jaundice; and, consequently, although jaundice is not a very common result of cirrhosis, cirrhosis is by no means an uncommon cause of jaundice.

Jaundice due to this disease has some peculiar characters. It is frequently gradual in its onset. It is often slight in degree. There is seldom, perhaps never, complete absence of bile from the fæces. It is unattended with pain, and so differs from jaundice due to gall-stones or cancer. On the other hand, it is very often associated with the other symptoms of cirrhosis which will hereafter be described, and particularly with ascites. In fact, the concurrence of jaundice with ascites is met with in scarcely any disease excepting cirrhosis and cancer.

Icterus gravis.—Jaundice is an early and constant symptom of the remarkable disease known as *acute yellow atrophy*. This will be fully treated in the next chapter.

Other causes of jaundice.—Icterus is often due to *cancer* either of the liver itself or of the biliary passages, and will come under notice as a symptom of malignant growths in and about the liver.

Deep and permanent jaundice may be caused in infants by a congenital

obliteration of the common bile-duct, apparently resulting from intra-uterine perihepatitis; several instances of this have been recorded. The jaundice has appeared a few days after birth; it has been attended with hæmorrhage from the bowels and skin, and especially from the umbilicus. This last has sometimes been the cause of death, which in other cases has been due to progressive atrophy, attended with vomiting and diarrhœa; in two instances the child has lived as long as six months.

Jaundice in infants (*icterus neonatorum*) is also said to arise from plugging of the common duct by inspissated bile, as in a case quoted by Murchison. But in many cases of supposed jaundice in newly-born children, the yellow tint is the result of changes of the blood in the over-congested skin, "the vivid redness of the newborn baby" (to use Murchison's expression) "fading, as bruises fade, through shades of yellow into the genuine flesh-colour."

Another very rare cause of permanent jaundice is *simple stricture* of the common duct, exactly resembling an ordinary stricture of the urethra. This condition is generally supposed to be the result of ulceration, itself set up originally by a gall-stone.

Permanent jaundice may also be caused by an *external tumour*, of any kind, pressing upon the hepatic or common duct. Even tuberculous glands in the portal fissure have occasionally obstructed the flow of bile. Again, an abdominal aneurysm, particularly one affecting the hepatic artery, has sometimes proved to have been the cause of jaundice. Such cases have generally been attended with severe paroxysmal pain, like what would be produced by gall-stones.

JAUNDICE FROM GALL-STONES.—Lastly, a frequent and important cause of jaundice is obstruction of the common bile-duct by gall-stones. Here the impediment to the flow of bile into the duodenum is not hypothetical and uncertain, but obvious and demonstrable.

Gall-stones are principally of two kinds. Some of them consist almost entirely of bile-pigment; others are made up mainly of cholesterine with but little pigment, and this may be altogether absent from the more superficial parts of the calculus.

The former kind are small, of a dark reddish, greenish-brown or almost black colour, irregular in outline, and so soft that on pressure they break down into a gritty substance. They are small and multiple, often counted by scores or hundreds.

The latter kind are hard and smooth on the surface; they split with a semi-crystalline fracture, displaying lines radiating from their centre, with the glistening aspect of cholesterine. In size and colour they are very variable. Some are three and a half to four inches in circumference; a gall-stone of this size is generally single and fills the whole gall-bladder, so that it has one rounded end answering to the fundus, and another tapering, which corresponds with the cystic duct. Others are quite small, of the size of marbles, peas, or scarcely larger than pins' heads. Several of them are often found in the same gall-bladder, and sometimes an enormous number—but this is not so frequent as with pigment calculi. Their surface is commonly, but not always, white or stone coloured, but their interior has generally a more or less deep yellow or brown hue from bile-pigment; and this is often arranged in concentric layers. Probably the centre of every gall-stone consists of biliary pigment.

The immediate origin of gall-stones is apparently a little mass of mucus.

To this bilirubin is attracted, and so a nucleus is formed, upon which cholesterine is slowly deposited.

Age and sex.—The liability to the formation of gall-stones increases greatly as persons advance in years. In the majority of cases, patients who have them are over fifty years of age, and many are sixty or seventy years old. Sometimes, however, they are found in those who are only twenty-five or thirty; and Cruveilhier and others have recorded their presence in children.

Women are more liable to gall-stones than men, in the proportion, according to Hein, of three to two. Perhaps this is due to the fact that females over the age of forty are particularly apt to gain flesh and to lead sedentary lives.

Symptoms.—Gall-stones are often found in the dead bodies of those who during life had not complained of any symptoms that could have suggested a suspicion of their presence. The gall-bladder is sometimes closely contracted over them, and (as its duct is generally obstructed by one of the concretions) it may contain beside only a very small quantity of mucus or pus. In such cases no clinical interest appears to attach to their presence. But sometimes when the cyst appears to be thus blocked, the walls of the gall-bladder go on pouring out mucus. It may then become much enlarged and form a tumour in the abdomen below the liver, the real nature of which is not always easy of determination. It may, for instance, be mistaken for a pendulous hydatid cyst.

A person who is attacked with jaundice from gall-stones generally experiences an agonising *pain*, which at once distinguishes this form of icterus from those that we have hitherto been considering. The pain begins unexpectedly and quite suddenly, often soon after a meal or after some muscular effort. It may be so excruciating that the patient is bent double by it and rolls upon the floor, uttering piercing cries. Epileptiform convulsions are seen to have been caused by this pain. After a while its intensity becomes somewhat lessened, and it is then replaced by a constant dull aching which continues until the more acute pain returns. The seat to which these agonising sensations are principally referred is the right hypochondrium, but they generally also shoot into the right scapular region and back; and they often seem to spread over a large part of the abdomen.

Another very marked symptom is *shivering*. The face is pale, the skin cool, and the whole body often covered with a cold sweat. The *temperature* is as a rule normal. Dr Duckworth has, however, seen several cases of biliary colic in which there has been pyrexia, in one of which the temperature rose nearly to 104°, and remained high for several days. Murchison also mentions that pyrexia is not uncommon ('Dis. of Liver,' p. 340). The *pulse* is much reduced in force and volume; sometimes it is slow, but more generally it is rapid and very weak. There is great exhaustion, the patient may swoon away, and it is said that fatal collapse has been known to set in. *Vomiting* is very frequent, and hiccough not uncommon.

Jaundice is not one of the earliest symptoms of an "attack of gall-stones." Indeed, it is evident that, until the calculus has passed from the cystic into the common duct no jaundice will arise. Generally speaking however, after a few hours, or at the longest a couple of days, the patient's urine begins to contain bile-pigment, and jauudice shows itself a little later.

It is often supposed that an attack of gall-stones (or "biliary colic," as it is often termed) is necessarily and invariably attended with pain. But a few years ago a case occurred in Guy's Hospital in which a man died in a

surgical ward of hernia who had previously had jaundice, which (it is expressly said) was unattended with pain. The gall-bladder contained numerous gall-stones; and the common duct was dilated so that the finger could be introduced into it.

There are several different ways in which an attack of gall-stones may terminate. Most commonly the jaundice sooner or later subsides. Its duration is then very variable. Sometimes it passes off in three or four days, somerimes it lasts several months. Indeed, even the shorter period exceeds the limit within which an attack of gall-stones may sometimes run its course. It may terminate within twenty-four or thirty-six hours; but in that case it is unattended with jaundice, which, as we have seen, seldom appears until the pain has lasted for some time. A good example of protracted jaundice from this cause is given by Murchison. It is that of a man who was jaundiced continuously for more than six months. Even in this case, however, the pain was not constant, but repeatedly went away for a week at a time. At last the jaundice disappeared, and the man returned to work.

When an attack of jaundice from gall-stones subsides, it is usually because the calculus has passed into the duodenum. The next thing is for it to be voided in the fæcal evacuations, in which (if it be looked for) it may often be found without much difficulty. Formerly writers said that if water is added to a stool containing a gall-stone, the latter will rise to the surface, from being lighter than the liquid. But it is now known that this is a mistake. When first voided, gall-stones have really a higher specific gravity than water; it is only when they have been dried that they float. The best way to detect a gall-stone in the fæces is that recommended by Murchison, namely, to mix up the evacuation with water and to pass the whole of it through a sieve, or a piece of muslin. In some cases, however, after the subsidence of an attack, no gall-stone can be detected. Possibly it is retained for a time within the intestine, or it may have undergone disintegration, particularly if it was one of those friable calculi which consist almost entirely of bile-pigment; or, again, it may never have escaped into the duodenum, but may have slipped back into the cystic duct or the gall-bladder. The last alternative is the one that comes most naturally into one's mind when a patient has very numerous and transitory attacks of biliary colic in quick succession, and when yet no calculus can be found in the evacuations. One is tempted then to suppose that all the attacks are caused by a single gall-stone, slipping to and fro in the duct. On the other hand, it must not be forgotten that a very large number of calculi are sometimes present in the same gall-bladder, and that a great many have been found in succession in the fæces. Sir Thomas Watson relates the case of a patient who collected fifty-five calculi from his stools within a space of five weeks. The discovery of the concretion after an attack of jaundice is not only important as verifying the diagnosis, but it may also lend some help as regards prognosis, in reference to the question whether it is likely that the complaint will recur. If the gall-stone was alone in the gall-bladder, its form is rounded; but if it was one of several it is very likely to show flat surfaces or facets where it touched the stones in contact with it.

Results.—As above stated, attacks of gall-stones have been reported, so severe as to prove directly fatal. But it is doubtful whether any well-authenticated case can be adduced in which a *post-mortem* examination has shown

that death has actually been due to this cause, independently of morbid change in the ducts. What does sometimes, although rarely, occur is that a gall-stone causes ulceration in the gall-bladder or in one of the ducts, which ulceration reaches the peritoneal surface, allows bile to escape into the serous cavity, and sets up fatal peritonitis. Some years ago a case of this kind occurred in Guy's Hospital. A woman had for some few years had repeated attacks of jaundice. For four or five weeks she suffered continuously from this symptom and from pain in the abdomen, which became more severe, until she died. At the *post-mortem* examination acute peritonitis was discovered, which had been caused by the escape of bile through an ulcerated opening in the hepatic duct. The common bile-duct was obstructed by a gall-stone. Murchison relates the case of a lady who died in about a week from a second attack of jaundice, and in whom the fatal result was due to peritonitis set up by perforating ulcers in the fundus of the gall-bladder, themselves caused by gall-stones. In other cases the ulceration of the gall-bladder or biliary passages caused by gall-stones has set up a local pyæmia, attended with the formation of abscesses in the liver, and leading within two or three weeks to a fatal result. Cases of this kind are distinct from those already referred to, in which diffused suppurative pylephlebitis is developed as a result of gall-stones.

Allusion has already been more than once made to the fact that biliary colic frequently occurs over and over again in the same individual, and a patient who has had this complaint once should therefore always be warned that he is likely to suffer from it again. Sometimes, but not always, the first attack is the most severe. When a concretion of some size has once passed through the common duct into the duodenum it is, of course, easier for another calculus of the same size to perform the same journey. The cases in which biliary colic terminates rapidly within a few hours, and even without causing jaundice, are chiefly those in which several previous attacks of the same complaint have occurred. These instances of repeatedly recurring biliary colic are often very trying, both to the patient and to his medical attendant; but in the majority of cases the attacks sooner or later cease to return. At any rate, in several instances of this kind the patients, even when advanced in years, have afterwards enjoyed excellent health, and have ultimately died of some other disease.

Sometimes, on the other hand, the jaundice persists until death. The termination of such a case may be due to some complication. In one instance it resulted from erysipelas of the face; in another from the supervention, at the same time, of acute endocarditis and acute meningitis. In another case, again, the patient fell into a comatose state and died in a fortnight after the commencement of his last attack of jaundice; and a fourth case was probably similar, of which no history is preserved beyond the facts that it proved fatal a few days after admission to hospital, and that the body was well nourished, as in death from some acute disease.

During the twenty-one years from 1854 to 1874 inclusive no case is recorded in our pathological reports at Guy's Hospital in which death occurred with chronic exhaustion and wasting as the simple result of the jaundice due to impaction of a gall-stone in the common bile-duct. The twenty-five volumes of the 'Pathological Transactions,' again, contain only two such cases; and even these may be said to have proved fatal by complications. One is recorded by Murchison; the patient, who had for many years been subject to attacks of gall-stones, died after six months of

jaundice, having suffered during the last three weeks from greatly increased pain and vomiting, hæmorrhages from mucous mombranes, &c.; the common bile-duct was obstructed by a large cylindrical gall-stone, which was ulcerating into the bowel by the side of the orifice of the duct. The other case came under the observation of Dr Wale Hicks; the patient died seven months after the attack commenced, but the jaundice, instead of being persistent, gradually faded, and at last entirely disappeared, and the hepatic tissue was found to have broken down into granular matter and oil; moreover, the obstruction in this case was not complete.

But the way in which death is most frequently brought about in persons who have gall-stones is by the development of cancer of the gall-bladder or the bile-ducts. Within the period of twenty-one years already referred to there have been at least twelve cases in which, gall-stones being present, there has been likewise malignant disease of these structures. In some instances the clinical history has pointed distinctly to the view that the jaundice was originally due to an ordinary attack of biliary colic, and that the development of the cancer was secondary; indeed, one case seems to admit of no other interpretation. A man aged forty-five died of jaundice which had lasted four months. Dr Moxon found that the gall-bladder was very large, containing hundreds of gall-stones; the common bile-duct at its commencement was greatly narrowed, and its walls were thickened by a cancerous growth; *below* the narrowed spot it contained three or four faceted gall-stones, just like those in the gall-bladder. This part of the duct also was dilated, and had evidently been accustomed to the passage of gall-stones before the cancer had begun to form. But in the great majority of the cases in question no gall-stone has been impacted in the duct at the seat of the cancer; the concretions have been found in the gall-bladder itself, which has often been contracted round them, and empty, or containing only a little purulent mucus. Thus it appears probable that if the malignant growth had not developed itself, all the symptoms would have subsided, and the health of the patient would have been restored.

Some observers doubt whether cancer of the bile-ducts in association with gall-stones arises as the secondary result of this irritation. It has been urged that gall-stones are often discovered in the bodies of those who have died from cancer of the breast or of other organs, but this may be merely a coincidence, for both cancer and gall-stones are apt to occur in persons advanced in years. However, even if some deeper relation than at first sight appears probable should be proved to exist between the formation of biliary calculi and the development of cancer in the body generally, this would not do away with the clinical significance of the facts stated above; it would still remain true that when a patient who has had attacks of biliary colic dies of protracted jaundice the ducts are almost invariably found to be affected with cancer.

Anatomical effects of permanent jaundice.—Whatever its cause, jaundice with obstruction of the ducts leads to a definite series of further changes. The gall-bladder and all the biliary passages become greatly dilated and distended, at first with bile, afterwards with a mucoid liquid, which may be of some shade of green or perfectly colourless. The gall-bladder may thus come to contain many ounces, or even (it is said) several pints, and it can be felt as a rounded or pear-shaped mass below the liver. Very often there are gall-stones in it as well as the fluid, and it is stated that their presence may

sometimes be detected by palpation, and that they yield a peculiar crackling sensation, which has been compared to that produced by grasping a bag of hazel-nuts or rolling pebbles about in the mouth. Sometimes the gall-bladder in these cases suppurates and points externally. The result of this is the production of a fistulous opening, through which, after a time, green bile is discharged in quantities of from eight ounces to two pints daily. The jaundice may then subside; but this change is of no benefit to the patient, who becomes rapidly reduced in flesh and strength, and before long dies exhausted. The hepatic ducts, again, become dilated in cases of permanent jaundice; they may even become larger than the branches of portal vein with which they run, and may be visible on the surface of the liver as cylindrical tubes or sacculated pouches. The enlargement of the ducts appears to be the cause of the fact that the liver as a whole is larger than natural in the early stages of this form of jaundice. But after a time the organ begins to shrink, and at length it becomes considerably smaller than in health. Another change in the liver consists in its assuming a dark olive-green colour, which all writers describe as darker than that of other parts of the body. Doubtless this is due to the oxidation of the bile-pigment contained in the hepatic cells, converting it into biliverdin, exactly as in the skin. The connective tissue in the portal canals becomes thickened when the common bile-duct is obstructed. This has lately been dwelt upon by Dr Wickham Legg, who has shown that in the lower animals the operation of ligaturing the bile-duct is quickly followed by an overgrowth of connective tissue as great as in intense cirrhosis. My observations lead me to believe that a similar change occurs very frequently in cases of obstructive jaundice in man, although not to the same degree.

The liver-cells appear not to undergo any change beyond being somewhat reduced in size. In 1843 Dr Thomas Williams related a case in which jaundice was caused by malignant disease of the head of the pancreas, and in which almost all the cells of the liver were found to be broken down, fatty globules and granular matter being present in their place. But no one has since recorded a similar observation, except, indeed, when before death cerebral symptoms had been present of such a kind as to justify the opinion that the disease which caused the jaundice had ultimately become complicated with acute yellow atrophy. This occurred in a case which Murchison has placed on record in the 'Pathological Transactions' (vol. xxii, p. 159).

Fatty stools.—Certain minor results, which sometimes follow jaundice, demand a brief notice. In 1832 Bright recorded some cases of jaundice in which the alvine evacuation contained a substance like fat, which either passed separately from the bowels or soon divided itself from the general mass, and lay upon the surface; "sometimes forming a thick crust, particularly about the edges of the vessel, if the fæces were of a semi-fluid consistence; sometimes floating like globules of tallow which had been melted and become cold, and sometimes assuming the form of a thin, fatty pedicle over the whole, or over the fluid parts in which the more solid figured fæces were deposited." This state of the fæces was sometimes so marked as to have been noticed by the patient before Bright saw the case. The oily matter had generally a slight yellow tinge and a most disgustingly fœtid odour. Bright was himself disposed to regard this peculiar condition of the evacuations as due to disease of the head of the pancreas and of the duodenum, and he seems to have thought that the presence of jaundice in

his cases was accidental. Later writers also have generally attributed the symptom under consideration to obstruction of the pancreatic secretion, supposing that it prevented the fatty matters taken in the food from being digested and absorbed. But this appears to be too narrow a view of the subject, for Bidder and Schmidt found that in dogs, in which the bile-duct was ligatured, the amount of fat that could be absorbed from the intestines was reduced to less than one half, and sometimes even to as little as one fifth or one seventh of the quantity that the animal could digest before. These experiments suggest a doubt whether the presence of fat in the fæces in Bright's cases was not caused by the obstruction of the common bile-duct rather than of the duct of the pancreas. However this may be, the symptom is a rare one.

Emaciation.—Dogs in which a biliary fistula has been formed require a larger quantity of food to maintain their nutrition than before. This corresponds well with the circumstance that in all protracted cases of jaundice the patient becomes exceedingly thin and emaciated; although a few cases of jaundice are entirely free from other conditions which might also account for the wasting.

Flatulence, &c.—Another result of the absence of bile from the bowels in jaundice is that their contents undergo putrefactive changes, the occurrence of which is prevented under normal conditions by the powerful antiseptic properties of bile. Hence the evacuations often have a very fœtid odour, and gases are generated which cause tympanitic distension of the abdomen. The contents of the intestines may probably in this way acquire irritant properties and so set up diarrhœa. But, on the other hand, obstinate constipation is very often present in jaundice, and this is commonly accounted for by the hypothesis that the bile is the "natural purgative" or stimulant to the peristaltic action of the bowels.

Pruritus.—Again, jaundice is sometimes attended with itching of the skin. Now, in certain persons papules develop themselves whenever the skin is scratched. Hence when they are jaundiced they often present an eruption of pimples, the summits of which become quickly destroyed by the finger-nails. This rash has been mistaken for scabies. According to Graves, urticaria may develop itself under the same circumstances. This accomplished physician also pointed out the fact that itching of the skin sometimes precedes jaundice by a considerable interval; in one of his cases this was a period of ten days, in another of two months. The late Dr Addison used to teach the same fact; and said that he had once suggested beforehand the possibility that an attack of jaundice might be impending when a patient complained of itching, for which no explanation could be found, and that his prediction had been justified by the result.

Xanthelasma.—Persistent jaundice, from whatever cause, is apt to lead to a remarkable affection of the eyelids and other regions called originally "Vitiligoidea" by Addison and Gull ('Guy's Hospital Reports,' 1851 and 1887), but now known as xanthoma or xanthelasma. It will be described in a future chapter among diseases of the skin.

Theory of jaundice.—We must now revert to a question of great importance, which I have already alluded to, namely, whether there are two distinct forms of jaundice, in the one of which the pile-pigment is secreted by the liver as usual, and afterwards reabsorbed into the blood; while in the other the secreting action of the liver is suppressed, so that any bile-

pigment that may be present must have been formed by some other organ or in the blood itself. We have seen that in certain cases of jaundice the ducts are mechanically obstructed, while in other cases they are patent. The question is whether the jaundice is essentially different in its origin in these two classes of cases. To use the phraseology of modern German writers, must we admit that there are two kinds of icterus—the one hepatogenous, the other hæmatogenous?

One distinction between these two forms of jaundice has been supposed to be that bile acids are present in the urine in cases of obstructive jaundice, and are absent when the bile-ducts are free. This distinction we have seen to be untenable. Nor does the state of the fæces afford such a criterion. In acute yellow atrophy, in which the ducts are unobstructed, the motions are sometimes, if not always, free from bile; and, on the other hand, we had in Guy's Hospital a case in which jaundice was due to obstruction of the common bile-duct by a cancerous growth, which, however, only partially occluded its channel, so that the fæces remained of their natural colour.

The theory of a hæmatogenous jaundice might seem to be sufficiently refuted by the fact that it is inconsistent with the physiological doctrine that the bile-pigment is in health formed by the liver, and does not pre-exist in the blood. But a glance at the evidence on which this doctrine is founded will show that the question cannot be thus summarily dismissed. This evidence (so far as it is independent of pathological observations, and from direct experiments on frogs, which are scarcely to the purpose) is chiefly based on the fact that no bile-pigment can be detected in the blood in health. But it is quite conceivable that, even though the bile-pigment is formed in the blood, it may never accumulate in sufficient quantity to be detected by chemical tests, if we suppose that the liver, with its large and active circulation, is engaged in removing it from the circulating fluid as fast as it is produced.

The remaining evidence in regard to this question is pathological, and is liable to be misunderstood. Thus, Murchison lays stress on the fact that the gall-bladder and bile-ducts are sometimes found after death to contain only a grey mucus, although during life there had been no jaundice. This would be a striking argument if the liver were always diseased in such cases. But Dr Moxon found that of four instances of this kind, one only was a case of fatty liver accompanying phthisis, the others being cases of pyæmia or pneumonia. It is evident that these cases merely show that under certain circumstances the formation of bile-pigment may be arrested. They have no bearing on the question whether the bile-pigment is formed by the liver or elsewhere.

Murchison, although a firm believer in the doctrine that the bile-pigment is formed by the liver, nevertheless does not adopt the opinion that the presence or absence of obstruction of the ducts makes any important difference in the way in which jaundice is produced. He points out that the osmotic currents between the blood and the contents of the biliary passages and intestines are extraordinarily active, and that even in health much of the bile which is secreted by the liver is probably reabsorbed into the blood. He argues that we have only to suppose that this reabsorption is excessive, or that the reabsorbed pigment fails to be properly got rid of, and we have at once an explanation of the occurrence of jaundice with patency of the ducts. But, as Dr Moxon has pointed out, this theory, that jaundice is in all cases due to reabsorption, is entirely inconsistent with the fact that in

jaundice the biliary passages are almost always found to contain, not bile, but an almost colourless mucus. This is the case, not only in acute yellow atrophy of the liver, but also when the ducts are permanently obstructed by cancerous growths, gall-stones, &c. As Dr Moxon remarks, the contrast is at first sight very astonishing between the deep yellow fluid found in distant serous cavities in such cases and the clear liquid which is present in the ducts of the liver itself, and which is sometimes so completely devoid of bile-pigment as to yield no reaction with Gmelin's test. The absence of bile from the biliary passages is, however, easily explained. The secretion of bile takes place under very low blood-pressure. Hence, when the common duct is obstructed, the entrance of bile into the biliary passages is probably arrested almost instantaneously. But the liquid poured out by the walls of these passages themselves and by the gall-bladder can undoubtedly continue to be formed under a much higher resistance. Consequently it soon displaces the last trace of bile; and like all mucous fluids, it is itself unstained by bile-pigment, even when jaundice is present.

This explanation evidently assumes that a colourless mucus will be found in the bile-ducts in cases of obstructive jaundice only when the obstruction is complete. And a search through the pathological records of Guy's Hospital for the last twenty years fails to discover any exception either to this rule or to that from which it is derived: on the one hand, there is no case of long-continued complete obstruction in which the ducts contained a liquid highly charged with bile-pigment; on the other hand, there is no instance of partial obstruction in which they contained a colourless mucus.

Certain writers have endeavoured to account for the presence of a fluid unstained with bile in the biliary passages in cases of jaundice, by supposing that the smaller ducts are plugged with inspissated bile, which thus cuts them off from the canals into which they should open. But it is surely inconceivable that all the smaller ducts throughout the organ should at the same time be obstructed in this way. For there would be no alternative but to suppose that in all cases of complete obstructive jaundice the bile-ducts up to the very point where their radicles meet the walls of the hepatic cells are plugged with a non-diffusible mucous fluid. This evidently brings obstructive jaundice into very close proximity with that form of jaundice in which there is no obstruction, and it can hardly be doubted that they both arise in essentially the same way.

The question as to the origin of jaundice is now reduced within very narrow limits. Yet we must still leave it unanswered. Is the bile-pigment first formed by the hepatic cells from materials which the blood brings to them, and is it immediately afterwards given up to the blood again? Or is it formed elsewhere in the body, so that the healthy liver-cells secrete without producing it and have their functions abolished from the time that jaundice appears?

In favour of the latter view is the fact that in protracted jaundice the liver-cells contain bile-pigment which has stagnated in them so as to have become green by oxidation. Moreover, it is difficult to see why jaundice should ever occur without obstruction of the ducts if the former view is correct. It is more easy to believe that in acute yellow atrophy the function of the liver is completely arrested than that the liver-cells pour back the bile-pigment into the blood as soon as it is formed by them, the ducts being perfectly patent. On the other hand, acute yellow atrophy itself

affords one argument of great power in favour of the view that the bile-pigment is formed by the liver. This lies in the fact, pointed out by Murchison, that as the destruction of the liver-cells progresses, the amount of pigment that is excreted by the kidneys diminishes.

Treatment of jaundice.—The *idiopathic* form is so uncertain in its course that it might well appear a hopeless task to determine whether remedies are capable of abridging its duration. But in many cases the disease subsides so quickly after the commencement of certain plans of treatment that it is difficult to resist the conclusion that these are possessed of very decided curative powers. On the Continent the most efficacious remedy is believed to be the administration of certain mineral waters: those of Vichy, Ems, Kissingen, Marienbad, and Carlsbad. Of these Vichy is the one most strongly recommended by French physicians, while German writers speak most highly of Carlsbad. Since all these springs contain a considerable quantity of the salts of soda (especially the sulphate and carbonate) it is interesting to find that the same salts are believed in this country to be useful in the treatment of jaundice. It is true that we seldom give the salts of soda alone, but rather combine them with remedies such as taraxacum and rhubarb, which are either thought to exert a specific action on the liver, or are regarded as useful by regulating the bowels.*

With regard to the general management of the disease, the patient should not be kept in bed, nor even within doors. He should be allowed to take moderate exercise, and to have his usual diet, from which, however, fat, pastry, and malt liquors, should be excluded.

When the cause of the jaundice has been removed, the fading of the yellow colour of the skin may, according to Murchison, be hastened by warm baths, by giving the patient diureties and diaphoretics, and by the administration of benzoic acid in four-grain doses three times a day.

With regard to the treatment of the various diseases which may give rise to *jaundice with pyrexia*, there is but little to be said; they are almost

* That in this way it is possible to bring an attack of jaundice to an end I feel confident, and I think that the following cases which came under my observation go far to prove it. A man aged fifty-nine came to me on January 9th, 1874, suffering from jaundice, which was not very deep, but had already been of two months' duration. There was some tenderness and fulness over the liver. I ordered him to take half a drachm of spiritus ammoniæ aromaticus in a mixture of rhubarb, soda, and calumba. He came again on the 16th, and said that for five days his motions had continued to be clay coloured, but that on the 14th they began to contain bile, and that they were now quite dark coloured. On testing his urine I found that it contained very much less bile-pigment than before. His jaundice was much diminished, and in the course of another week it entirely disappeared. In the next case there was some doubt as to the exact cause of the jaundice, but this does not affect the question of the value of the treatment. A bargeman came to me on February 23rd, 1872. Ten months before he had become suddenly jaundiced without pain. A month afterwards he had been seized with excruciating pain over the liver, lasting some hours, and he had since had three similar attacks. He had been under treatment both in Guy's Hospital and at King's College, but without result. He remained jaundiced the whole time. Having ascertained the treatment that had been previously employed in Guy's, I ordered him to take ten grains of carbonate of soda, with a scruple of extract of taraxacum, and (as he had some dyspeptic symptoms) half a drachm of tincture of hop three times a day, and a grain of opium at night. On March 1st he came to me again, and assured me that he was very much better, having lost the pain and sickness. On March 4th he noticed that his motions resumed their natural appearance, and before long he was well. I do not suppose that all cases of simple jaundice will subside equally rapidly under such treatment, but I think it certainly deserves a fair trial. Another remedy which is often prescribed for this complaint is the dilute nitro-hydrochloric acid. I have seen simple jaundice quickly pass off in patients who have been taking this remedy.—C. H. F.

more invariably fatal than acute atrophy itself. The administration of quinine and stimulants may doubtless somewhat lower the pyrexia, and if the issue should be at all doubtful, may perhaps incline the balance in the patient's favour.

When *permanent* jaundice has once declared itself, and the obstruction of the common duct is complete and irremediable, it is no longer advisable to prescribe carbonate of soda, or dilute nitro-hydrochloric acid, or taraxacum. The more faith we have in the efficacy of these remedies in simple jaundice, the more we shall fear that they may now do harm. Regulation of the diet is perhaps the most important part of the treatment. It has been shown experimentally, by Bidder and Schmidt in Germany, and by Bennett and Rutherford in this country, that dogs with artificial biliary fistula may live for years provided they are supplied with and will take a sufficiently large quantity of food. It is true that in those animals the escape of bile through the fistula causes a drain which is wanting in jaundice in the human subject, but the experiments at least suggest the conclusion that a large supply of nutriment should if possible be maintained. At the same time its quality should be carefully attended to. In dogs whose common duct has been tied the daily quantity of fat that can be absorbed from the food is greatly diminished. Evidently, therefore, oleaginous and fatty articles of diet should be taken very sparingly, if at all, by persons with permanent jaundice.

Something may be done to counteract the absence of bile in the intestines by the administration of the purified bile of the ox or pig. Murchison recommends that this should be given in doses of from three to six grains, about two hours after meals, in capsules or pills coated with a solution of tolu in ether, so that they may pass through the stomach unaltered. The pancreatic emulsion may possibly do good in such cases.

Oxgall has the further advantage of taking up the antiseptic function of the natural bile. With this object, as well as that of relieving flatulence, creosote, turpentine, vegetable charcoal, &c., may likewise be prescribed with benefit. Occasional laxatives are generally required, and the milder ones should be preferred.

The *itching* caused by jaundice is sometimes so troublesome as to require special treatment. Warm baths have sometimes proved serviceable, and, according to Niemeyer, especially alkaline baths. The use of the flesh-brush is recommended by Murchison, and the internal administration of the bicarbonate of potass. He mentions that the last-mentioned remedy did some good in one case in which opium and morphia had failed. Very often, however, this symptom is one which baffles all efforts to relieve it. Belladonna internally and a hydrocyanic acid lotion externally are probably the best means of relief. Opiates may certainly aggravate the irritation.

Treatment of gall-stones.—In an attack of "biliary colic," the patient should first be placed in a hot bath. Fomentations and poultices are then to be applied to the abdomen. If there be much tenderness on pressure in the right hypochondrium, a few leeches may often be used with great relief to this symptom, according to the testimony of Murchison and other writers. But these measures will not suffice for the relief of the agonising pain without the administration of opium or morphia in full doses frequently repeated. In a patient previously in good health two grains of opium are not too much to begin with, followed by a grain every two or three hours, until ease or sleep is

obtained, it being of course understood that the case is carefully watched. Very often the stomach is too irritable to retain the anodyne, and then the subcutaneous injection of a quarter of a grain of morphia may be resorted to with signal advantage. Another antispasmodic, of which Murchison speaks highly, is the extract of belladonna, which he gives for this purpose in half-grain doses. The inhalation of chloroform has sometimes proved very effectual, and is certainly the speediest and most effectual way of relieving the pain when it is at its worst. Another plan, which was highly recommended by Dr Prout, is the administration of large draughts of hot water, containing one or two drachms of the bicarbonate of soda to the pint. Dr Prout speaks of this as acting like a fomentation to the seat of pain; and even when the stomach rejected the first portion of the fluid, he used to persevere, believing that it diminished the severity of the retching. If, however, the vomiting be very violent, it should be checked by effervescing draughts, dilute hydrocyanic acid, and the like. Many of the older physicians, and even Bright, prescribed antimony in the treatment of biliary colic with the hope of relaxing spasm, and so facilitating the expulsion of the calculus, but this remedy is now justly discarded on account of its tendency to aggravate the sickness. A mixture of turpentine and ether is a once famous remedy for biliary colic. It is usually rejected by the stomach.

There are no means at present known of *preventing* the formation of gall-stones. Active exercise, spare diet and occasional cholagogues are believed to be of some service, and the sulphate and phosphate of soda are often prescribed. Chloroform is a chemical solvent of cholesterine and of bilirubin; hence it has been given when gall-stones are suspected, to prevent them becoming impacted; five or ten up to thirty drops may be taken in spirits and water. The succinate of the peroxide of iron also has been recommended as a solvent, but without even theoretical probability of its being useful.

When the *gall-bladder* is distended by an impacted calculus, it occasionally becomes adherent to the abdominal parietes; an abscess may then be developed which points externally, and when it breaks or is opened by a surgeon, gall-stones are discharged with the pus. The site of the external opening is by no means always directly over the gall-bladder; it may be at the umbilicus, or even in the left side of the abdomen; nay, a case has been recorded in which two biliary calculi made their way into the connective tissue of the vagina. It is important to note that in cases of this kind there are (or may be) no symptoms directly suggestive of the presence of gall-stones until they are found in the discharge. The common bile-duct is often quite free, while the cystic duct is completely closed; and thus, neither is there any jaundice nor does any bile enter the gall-bladder and mix with the pus. Hence, the abscess is often supposed to be seated in the abdominal walls, or, again, in the substance of the liver, and months or even years may pass before the real nature of the case declares itself. In the meantime the patient has a fistulous opening in the side, which, however, need not prevent the enjoyment of good health; and when all the gall-stones have come away, it may, at length, heal up.

The treatment of gall-stones and of a suppurating gall-bladder by operation (cholecystotomy) has been followed in recent times by some remarkable successes: see Dr Marion Sims' case, 'Brit. Med. Journ.,' June 8th, 1878 (p. 811); and Mr Lawson Tait's, 'Med.-Chir. Trans.,' 1880, and subsequently. References to earlier proposals of the operation, and isolated

cases of its execution, will be found in the 'London Med. Rec.,' April 15th, 1881, p. 153.

When the gall-stone escapes by ulceration into some part of the intestine, it may be voided per rectum; and since a concretion which takes this course is often very large, its passage through the anal orifice may be attended with severe pain and violent straining, the cause of which cannot be explained until the gall-stone is discovered. Probably when a large stone thus makes its way out of the body, it has passed from the gall-bladder directly into the hepatic flexure of the colon; but more often, when the stone is of moderate size, it is with the duodenum that an ulcerated gall-bladder communicates. As mentioned in a previous chapter (p. 415), the gall-stone may then fail to pass through the small intestine, and becomes impacted in the jejunum or the ileum, so as to set up obstruction of the bowel.

The recognition of this condition is, as we saw, difficult, and rests usually on probability derived from the age and sex of the patient and previous accounts of jaundice and pain.

The treatment is by opium, so as to relax the grip of the bowel on the gall-stone. When after abdominal section for obstruction, the cause has been found to be an impacted calculus, it has sometimes been possible to manipulate it through the ileo-colic valve and safely leave it there. In other cases the gut has been opened and the stones extracted with complete success. Crushing the calculus by means of padded forceps without wounding the intestine, and breaking it up by means of a needle, are methods which have been suggested but not as yet carried out.

INFLAMMATORY DISEASES OF THE LIVER

ACUTE SUPPURATIVE HEPATITIS—ABSCESS OF THE LIVER.—*Geographical distribution—Ætiology—Relation to dysenteric ulceration—Morbid anatomy—Characters of the pus—Direction of rupture—Symptoms—Physical signs—Diagnosis—Abscess between the liver and diaphragm—Prognosis—Treatment by drugs—Paracentesis.*

CHRONIC INTERSTITIAL HEPATITIS—CIRRHOSIS OF THE LIVER.—*Anatomy—Ætiology—Effects upon the portal circulation—Early symptoms—Jaundice—Cerebral symptoms—Hypertrophic cirrhosis—Ascites—Diagnosis—Perihepatitis—Simple chronic atrophy—Syphilitic hepatitis and gummata of the liver—Cancer—Adhesive portal phlebitis—General prognosis and treatment of cirrhosis.*

ACUTE ATROPHIC HEPATITIS—ACUTE YELLOW ATROPHY OF THE LIVER.—*History — Anatomy — Symptoms—percussion—the urine—jaundice—cerebral symptoms — Ætiology and Pathology—Diagnosis—Prognosis—Treatment.*

IN passing from disorders of the functions of the liver and interference with the due excretion of bile to the structural diseases of this important organ, we will take first the subject of hepatic inflammation. The liver is not liable to ordinary simple, primary, or idiopathic inflammation as the result of cold, like bronchitis and pleurisy, nor to acute inflammation secondary to a definite preceding cause, as endocarditis to rheumatism. Hepatitis occurs in the three following very different clinical forms:

1. Acute suppuration, which we may compare with abscess of the brain.

2. Chronic interstitial inflammation with subsequent fibrous change, contraction, and hardening, leading to atrophy of the glandular parenchyma; a change closely analogous in its pathology to sclerosis of the spinal cord, to chronic interstitial pneumonia, and to the most chronic form of Bright's disease. It is from this analogy that we commonly speak of cirrhosis of the lung and cirrhosis of the kidneys, while some pathologists describe the corresponding hepatic disease, not as cirrhosis, but as sclerosis of the liver.

3. There remains the most rare, most obscure, and most remarkable disease of all that affect this organ, an acute affection leading to the most rapid atrophy, and wanting in many of the characteristics of inflammation. It is open to question whether it should be regarded as hepatitis at all. But it appears to bear some distant analogy to acute pneumonia on the one hand and to acute Bright's disease on the other; and unless we recognise it as a parenchymatous inflammation, peculiar to the liver as pneumonia is to the lung, it is altogether unique in pathology no less than in its clinical aspect, and we must either treat of it apart, or name it by one of its most striking features as Icterus gravis and group it (as was done in the first edition of this work) with jaundice from obstruction. On the whole it appears best to place it

near abscess and cirrhosis; but the arrangement is mainly one of convenience, and each of the three sections of the present chapter will be treated independently.

Acute Suppurative Hepatitis—Abscess of the Liver.—We have already seen that a large number of abscesses of the liver are often found in cases of pyæmia, general or portal, and in the latter affection pus is found in the portal canals throughout the organ (p. 498). In neither of these conditions, however, is any considerable quantity of matter developed at one spot.

We have now to consider a very different class of cases,—those in which a single abscess (or a limited number of abscesses) arises in the substance of the liver.

In England this disease rarely occurs. Indeed, some writers have stated that it is never seen, except in those who have previously lived in a hot climate, and the name of "tropical abscess of the liver" has therefore been assigned to it. This statement, however, is not altogether accurate. Between 1860 and 1880 there have been in Guy's Hospital fifteen cases in which death was caused by the formation of a single large abscess (or in one instance two large abscesses) in the liver. Five of these cases occurred in persons who had come from China, or India, or the West Coast of Africa, but in ten of them there was no such history, and in the majority of them it is positively stated that the patients had never been out of England. Still it is undoubtedly true that this form of hepatic abscess seldom arises here, whereas in India and other hot climates it is very common, and indeed takes a most important place in the European death-rate. In the West Indies it is said to be comparatively rare.

Ætiology.—It has long been known that abscess in the liver and dysentery are often associated, and various opinions have been held as to the connection between these two diseases. Annesley supposed that sometimes, as the result of an hepatic abscess, the bile acquires peculiarly irritating properties, and thus sets up inflammation and ulceration of the intestine. On the other hand, Dr George Budd in 1842 propounded the theory that dysentery is the earlier of the two occurrences, and that abscess in the liver is the result of the absorption of some morbid product from one of the intestinal ulcers; in other words, that the pathology of the so-called tropical or single abscess of the liver is essentially the same as that of the multiple abscesses with which we are familiar as arising from portal pyæmia. Dr Budd's view for some time received general acceptance; but it is rejected by almost all recent writers who, from their experience of tropical diseases, speak with special authority upon this subject.

Some of the arguments which these writers use are not of great weight. Thus it is said that if dysenteric ulcers in the colon were the cause of abscess of the liver, the same result ought to follow other forms of intestinal ulceration, such as occur in phthisis or in enteric fever. But it is well known that in all inflammatory affections the liability to the production of pyæmic infection depends essentially upon the character of the original mischief; and in dysentery this is just such as would be likely to set up blood-poisoning, which is not the case in either tubercular or typhoid ulceration.

Another argument is that many cases of hepatic abscess, in which recovery takes place, run their whole course without the patients having any

symptoms of dysentery; and another, that when such symptoms do occur they often seem to follow rather than to precede those which indicate the occurrence of suppuration within the liver. But it is a question whether dysentery does not frequently begin insidiously, or even remain altogether latent (cf. p. 398). In England this is often the case, and it very probably is so in India likewise.

Again, it is said that abscess of the liver does not occur in all epidemics of dysentery, and is comparatively uncommon in certain countries where dysentery prevails. Aitken speaks of it as being very rare in China, as compared with India. Dr Baly did not meet with it in the epidemic of dysentery at Millbank. An answer to this objection might perhaps be found in a more careful study of the conditions under which dysentery and hepatic abscess are found to coexist. It may be that a certain length of time is required for the development of suppuration in the liver, and thus abscess may necessarily be wanting in rapidly fatal epidemics.

But the main strength of those who deny that abscess of the liver is secondary to dysentery lies in the fact that many cases have been placed on record in which, after death has occurred from tropical abscess, the intestines have been examined and found to present no sign of past or present inflammation. Murchison met with a case of this kind in a European soldier in Burmah. The man had never had dysentery, although it may be noted that while he was under observation he suffered from persistent diarrhœa. He died, and an enormous abscess, holding four quarts of pus, was found in the liver, but neither the small nor the large intestines nor the stomach presented any cicatrices or trace of recent ulceration. Again, Mr Waring collected 204 cases of abscess of the liver, in exactly one fourth of which the intestine is said to have been perfectly healthy, and Dr Morehead mentions that he has notes of twenty-one similar cases.

Among fifteen fatal cases of large abscess of the liver which occurred at Guy's Hospital there were three in which no sign of ulceration was found in the intestines; one of these was a tropical case. In eleven others it is expressly stated that the bowels were or had been diseased. In one case which came under Dr Moxon's observation there was only a minute cicatrix in the gut, so small that it might very easily have been overlooked. Now, we surely cannot accept this as accounting for the occurrence of suppuration in the liver, particularly as such an admission would take away the ground from beneath our feet in maintaining that tuberculous and other ulcers cannot be expected to give rise to it. It is perhaps better to defer the expression of an opinion with regard to cases like this in which no indication of intestinal mischief has been discovered, until further researches shall have shown whether or not dysentery is apt to occur in a latent form in the East. If this should prove to be the case, there would be no difficulty in accounting for an occasional case of hepatic abscess with no *post-mortem* evidence of ulceration of the bowel, by supposing that the latent inflammation subsided before any actual breach of surface occurred, which undoubtedly is often the case.

It seems therefore premature to give up the theory that abscess of the liver is secondary to dysentery, until fresh facts shall have proved that this theory is untenable. The positive observations which support it are very strong; in this country dysentery and hepatic abscess are each so rare that their frequent association would be a most extraordinary circumstance, unless they are more than casually connected; and it is the rule that the ætiology of a

disease can best be studied, and its origin best unravelled, in the countries where it is not too common.

In India the current opinion at the present time would appear to be that dysentery and abscess of the liver are really common results of the same causes. It is supposed that inflammation extends from the mucous membrane of the alimentary canal to the largest gland which opens into it, just as it does from the urethra to the testes in cases of gonorrhœa. The principal objection to this view appears to be that, while it would account for the occurrence of general hepatitis, it affords no explanation of the formation of a circumscribed abscess.

If pre-existing ulceration of the intestine be rejected as a constant cause of hepatic abscess, it is very doubtful whether any other cause can be assigned in its place. Elevation of the temperature is a supposed cause, and Dr Morehead thinks that it may sometimes explain the occurrence of hepatitis during the hot months of the year in plethoric individuals who have not long arrived in India. But it appears from the statistics of the European General Hospital in Bombay that the admissions of patients with hepatitis are relatively more numerous during the months which follow the cold season, and during the cold season itself, than during the hot months. Hence if heat be concerned in production of abscess of the liver, it can be only as a predisposing cause ; while exposure to cold may possibly act as an exciting cause. There is no reason to believe that intemperance has anything to do with the causation of the disease.

Local injury—such as a blow in the right hypochondrium—has sometimes been accused of having set up suppuration in the liver, and probably with justice, but this explanation is applicable to very few cases. A case in point occurred at Guy's Hospital in 1876. A drunken woman was run over, but the ribs were not fractured. She died with symptoms of pneumonia of the right base, and after death this was found to be consecutive to a large abscess in the right lobe of the liver, which had not perforated the diaphragm.

In another still more remarkable case which happened in 1881, an abscess in the liver was caused by the perforation of a large phthisical vomica in the base of the right lung which opened into the liver. In a third case which occurred at Guy's Hospital in the same year, two acute abscesses of the liver in a boy of ten were found after death to have been caused by a pin lodged in the appendix cæci : a similar example was recorded in the ' Pathological Transactions ' for 1870.

Excluding cases of general pyæmia, suppurating hydatid cysts, and tropical cases in soldiers and sailors from abroad, the hepatic abscesses have been found due to portic pyæmia : from dysentery, ulcerative colitis, fistula in ano, gastric ulcer, or typhlitis.

Anatomy.—The pathological processes which are concerned in the production of abscesses of the liver have been minutely studied by German observers, and are fully described by Rindfleisch. He distinguishes a "thrombotic" from an "embolic" variety. In the former, the inflammation is said to occupy especially the walls of the interlobular branches of the portal vein. These, and the sheath of connective tissue round them, are swollen by an infiltration of leucocytes, until the columns of hepatic cells become compressed and perish. The adjacent masses of infiltrated connective tissue then come into contact, and they form small white granules, which are very similar in size to lobules, and which, indeed, might

be mistaken for them. These "pseudo-lobules" in their turn melt away, and an abscess cavity is formed. But fresh pseudo-lobules are constantly making their appearance at its periphery and give a ragged character to its inner surface. In the "embolic" variety, on the other hand, the portion of hepatic tissue which corresponds to the distribution of the plugged vessel becomes intensely congested, the circulation in it is entirely arrested, and it sloughs *en masse*. The lobules round it undergo reactive inflammation, they become enlarged, and those nearest the sloughing part are permeated by numerous pus-corpuscles which lie outside the secreting cells, between them and the capillaries. The hepatic cells appear to take no active share in the formation of pus. It must be added that the observations on which these statements rest appear to have been made in cases of the small multiple abscesses of pyæmia, which are not now under consideration; but it is probable that they may be applied generally.

The characters of abscesses of the liver vary greatly in different cases. When of recent formation and rapid growth they may possess no limiting membrane whatever, the pus lying in an irregular cavity formed by reddened and softened hepatic tissue. If they have been of somewhat longer standing they are lined with a layer of opaque yellowish material, the formation of which has been described above. Very old abscesses have a dense fibrous wall, which may be three or four lines in thickness, and so hard as to feel like cartilage, or it may even be partly calcified. Sometimes, in making an autopsy, one finds that such an indurated wall has become more or less detached from the surrounding tissues, and lies bathed in pus which has passed through it, so as to be limited only by the substance of the liver. In such cases, no doubt, the abscess has been of long standing, but has undergone rapid extension shortly before death.

There have been some discrepancies in the statements of writers as to the appearance of the pus in an hepatic abscess. Dr Budd states that it is usually white or yellowish and free from odour; and no doubt this is correct. But the records of autopsies at Guy's Hospital state that in three cases it was greenish and either mucoid or curdy, and in two other instances it was of a reddish or reddish-brown colour. This is of some interest, because Budd asserted that the pus of an abscess in the liver is never red, so long as it is confined within the cavity of the abscess itself, but acquires such an appearance only when it is expectorated through the lung. He regarded this kind of expectoration as affording conclusive evidence of the existence of an hepatic abscess. But Dr Morehead, who at one time held the same view, says that in more than one case of asthenic pneumonia he has observed sputa having precisely the same characters. In one case of hepatic abscess that came under the author's care in Guy's Hospital some years ago, the fluid removed by the trocar was of a brickdust colour, looking not unlike anchovy sauce; and it had a most peculiar and nauseous odour.

An abscess of the liver may sometimes attain an extraordinary size. We once found one to hold six pints; but Maclean mentions one which contained altogether nineteen.

As might be expected, when the pus has reached the surface of the liver, it may make its way in various directions. Thus it may point externally in the right hypochondriac and epigastric regions, the serous surface of the liver having first become adherent to the wall of the abdomen; or it may rupture into the peritoneum; or, again, it may discharge itself into

the stomach, duodenum, or colon. In this last event the patient may vomit a considerable quantity of matter, or pass it per rectum. But in many cases the pus cannot be traced. Sometimes, again, it burrows towards the lumbar region. Again, hepatic abscesses not infrequently perforate the diaphragm, so that their contents are evacuated through the lung. Dr Morehead says, however, he has seen three cases in which a patient suffering from this disease expectorated pus, but no perforation could be discovered after death.

Symptoms.—In many cases abscess of the liver remains entirely *latent* and is found after death in the bodies of those who had never been known to suffer from any symptoms of the disease. Cases of this kind are mentioned by all writers on tropical diseases, for it is chiefly in persons who have been in hot countries that latent abscess of the liver has been met with. The author once examined the body of a gentleman who died of protracted diarrhœa a few years after his return from China. There was an abscess the size of a walnut in the posterior part of the liver, although careful inquiry during the patient's life had failed to elicit any evidence that the liver was otherwise than perfectly healthy.

Again, an abscess of the liver, which up to a certain point has gone on increasing in size without affecting the patient's health, may suddenly give rise to the most serious symptoms. Maclean mentions the case (occurring in the Mauritius) of a man, apparently in good health, who had walked seven or eight miles in search of employment, when he complained of pain at the pit of the stomach and in a few hours died; an abscess of the liver, lined with a dense fibrous membrane, had burst into the pericardium. Another case is that of a man who had been invalided from India on account of "chronic hepatitis," but who when he arrived at Fort Pitt had apparently recovered so completely that he was sent to the depôt for duty. Some weeks afterwards, while he was straining at stool, "something gave way;" and this proved to be an hepatic abscess, which likewise had ruptured into the pericardium. Maclean supposes that the thick capsule which most of these latent abscesses possess prevents their causing constitutional irritation, by forming a barrier, so to speak, against the disturbing influence of the pus. But the growth of a dense capsule is of course the work of time; and the abscess in these cases is latent from the very first, before any capsule exists.

In many cases, however, abscess of the liver gives rise to symptoms which are strongly indicative of its presence; and the results of physical examination of the hepatic region may be almost conclusive. The symptoms are said to be a sense of fulness and weight, or even pain, in the right hypochondrium, pain in the right shoulder, inability to lie on the right side, fever, disturbance of the digestive organs, and cough.

The *pain* which attends abscess of the liver is exceedingly variable in degree. It is often much more intense in the shoulder than in the hypochondriac regions. Budd mentions a case in which the pain in the shoulder was for a long time most severe; but when the abscess was opened the pain became relieved. The occurrence of pain in this spot has been said to be a proof that the disease is situated in the *right* lobe of the organ. The pain over the liver itself is often increased by pressure, and also by the patient drawing a deep breath, or turning over on to the right side.

In cases of hepatic abscess *cough* is often present. It is generally short and dry; and is no doubt due to reflex-irritation.

It might be expected that *pyrexia* would be a marked symptom; and Maclean states that in every case of suppuration of the liver that had recently been under his observation at Netley the thermometer showed a rise of from one to three degrees. But these were, no doubt, cases in which the existence of abscess was suspected on other grounds; there appears to be no reason for supposing that thermometric observations would often reveal its presence if it were undiscoverable by other means. In the case above mentioned of hepatic abscess from a pin in the appendix cæci, the temperature rose to 106°, and this, with the great increase of hepatic dulness, local tenderness, and absence of other causes of fever, led to the diagnosis of suppuration of the liver.

Dr Morehead believes that *rigors* are not of much value as indicative of suppuration in the liver; they may be present when there is no abscess; and, on the other hand, they are often absent when an abscess is forming.

Jaundice appears never to arise in the disease under consideration, unless by accident, from the occurrence of pressure upon one of the main ducts. With multiple pyæmic abscesses it is frequent (cf. p. 498).

The *physical signs* of hepatic abscess depend mainly upon its position and its size. Twining, of Calcutta, believed that even a deep-seated abscess often indicates its presence by producing a peculiar rigidity of the upper part of the rectus abdominis muscle on the right side; and there is no doubt that this condition of the muscle is an important sign of disease in some one of the viscera immediately subjacent; but later observers have by no means confirmed the opinion that it points especially to the disease now under consideration. The existence of enlargement of the liver is of much more diagnostic value when present; particularly if the outlines of the organ be altered, if there be bulging or tumefaction in one particular direction, and if any spot can be detected which is soft and fluctuating. Where enlargement of the liver can be clearly made out, there is generally much tenderness on pressure in the hepatic region; and round any fluctuating point there is almost always a marked inflammatory induration of the abdominal parietes.

These decided indications of hepatic abscess are of course absent in all cases in which the back part of the liver is the seat of the disease. Even then, however, there may be an increase in the area of hepatic dulness, which, if well-marked symptoms be present, may make the diagnosis sufficiently clear.

Diagnosis.—That the detection of hepatic abscess is often very difficult may be inferred from the statements that have already been made as to the vague character of the symptoms which alone are present in many cases.

According to Maclean, there is another affection of the liver which is common in India, and which is very apt to be mistaken for suppurative hepatitis, viz. acute inflammation of the capsule of the liver, or *acute perihepatitis*. The symptoms of this disease, however, are said to resemble those of pleurisy rather than those of suppuration in the parenchyma of the liver. The pain is sharper and more acute than in the last-named disease, and it is more decidedly aggravated by pressure, or by a full inspiration, or by movement of the patient's body. Acute perihepatitis is probably not very uncommon in this country, for the liver is often found after death to be fixed to the diaphragm by adhesions, which appear to correspond with those which would be left by an acute, rather than with those

that would have resulted from a chronic, inflammation; but it is doubtful whether this affection is at present capable of clinical recognition. Sometimes, however, a peritoneal *rub* may be heard over the surface of an enlarged liver.

Even where there are distinct signs of the existence of an abscess in the right hypochondrium, one must not take for granted that this is seated in the interior of the liver itself. It may be embedded in the substance of the *abdominal walls*. Again, in the 'Guy's Hospital Reports' for 1874, a series of cases, six in number, was published by the author, in which a collection of pus existed in the *right hypochondrium*, between the upper surface of the liver and the diaphragm. This affection is most frequently caused by direct injury to the part, or by extension of inflammation from disease of one of the other abdominal viscera. In these respects it differs from abscess of the liver itself, which, however, in all other points resembles it very closely, and probably is not capable of being distinguished from it by physical examination. A correct diagnosis between these two diseases is not a matter of merely scientific interest; for a subdiaphragmatic abscess can probably be more readily cured by evacuation of its contents than one embedded in the liver, since its walls can more easily come into apposition.

Again, a *suppurating hydatid* of the liver may easily be mistaken for simple abscess, if the case be first seen when inflammation has already been set up within the capsule of the cyst, and if the existence of a painless tumour previously should have escaped the observation of the patient. Indeed, in such cases, it is really impossible to determine the nature of the disease until the contents of the abscess are discharged, so that they can be submitted to careful examination. The treatment is the same as for a single large hepatic abscess, and the result is equally successful, so that the question of diagnosis is not of great importance.

Lastly, it is said that a *suppurating gall-bladder* might be mistaken for an abscess situated within the substance of the liver; but this applies only to abscesses situated in a particular region.

Prognosis and treatment.—The treatment of a case of acute hepatitis, in which suppuration is feared, must vary according as there is or is not reason to believe that pus has already been formed within the liver.

Writers of large experience in India concur in stating that in the early stage of the disease ipecacuanha is a very potent and valuable remedy. Maclean says that this drug is nearly as efficacious in suppurative inflammation of the liver as in tropical dysentery, and that it should be given in the same manner, viz. in doses of from fifteen to twenty grains, repeated at intervals of five, six, or eight hours. Antimony is also used by some medical men in India, either in addition to the ipecacuanha or separately; and stress is also laid upon the importance of relieving pain and giving sleep by the hypodermic injection of morphia. Fomentations or poultices are kept applied to the hepatic region.

It appears to be established that under such treatment patients often quickly recover after having had symptoms exactly like those which are recognised as the early symptoms of hepatic abscess; but can one be certain that suppuration would really have occurred in these cases if left to themselves?

When there are grounds for believing that an abscess has actually been formed in the liver, the main question as regards the treatment is of course whether or not the pus should be evacuated by the surgeon. The answer to

be made to this question must depend upon the results of experience with regard to the natural course of the disease when left without interference. Now, it is certain that hepatic abscesses sometimes subside spontaneously. In making *post-mortem* examinations dried-up abscesses have sometimes been found with cheesy contents. This, however, occurs more rarely than was formerly supposed; for, on the one hand, dead hydatids have probably often been mistaken for cured abscesses; and, on the other hand, it is certain that the same view was generally taken of syphilitic growths in the liver before their real nature was understood. So that the spontaneous subsidence of a hepatic abscess without discharge of its contents can scarcely be anticipated in any case in which well-marked symptoms of suppuration have once shown themselves; and particularly if its site is indicated by definite physical signs.

Cases in which an hepatic abscess has discharged its contents spontaneously not infrequently terminate favourably, the cavity after a time closing and pus ceasing to be formed. It would appear that the prospect of such a termination has hitherto been greatest in those cases in which the abscess has made its way through the lung. Dr Stovell is said to have reported nine recoveries out of sixteen cases of this kind; but according to Morehead, there is reason to believe that the abscesses were small. Again, recovery is by no means infrequent when an abscess discharges into the stomach or intestine. Morehead had five cases which took this course; and three of them terminated favourably. Rupture into the peritoneum or pericardium is almost invariably fatal within a short space of time; but fortunately abscesses of the liver very seldom take either of these directions.

Again, when an abscess points towards the exterior of the body, and presently breaks and discharges its contents through the skin, the patient sometimes ultimately does well. Maclean says that this is much more likely to occur when the point at which the abscess reaches the surface is near the ensiform cartilage than when it is in the right hypochondrium, or in an intercostal space. It appears that Morehead was the first to draw attention to this distinction, which apparently depends upon the circumstance that in the former case the collection of pus is usually a small one, being seated in the left lobe of the liver.

No better indication could be given of the extreme divergence of opinion which has existed with regard to the propriety of surgical interference in cases of hepatic abscess than the fact that some authorities, including Budd, have expressed the opinion that even when an abscess is actually pointing it should be allowed to break of its own accord.

Within the last twenty years, however, the whole aspect of this question has been changed by the introduction of the aspirator of Dieulafoy, and of the various appliances of the antiseptic method. No better illustration of the advantage of treating cases of hepatic abscess with the aspirator could be given than a case which is reported in the 'Medical Times and Gazette' for April, 1874, from the Madras Hospital.

An Englishman was admitted with a tender swelling extending from the hepatic region downwards to the level of the umbilicus. He had daily accessions of fever, and other symptoms indicative of deep-seated suppuration. As there was fluctuation in the centre of the swelling, this was tapped with the aspirator, and four ounces of pus were withdrawn. The relief, however, was but partial; and when the operation was twice repeated, only a small quantity of matter came away each time. The patient's condition kept getting

worse. The existence of a second abscess was therefore suspected; and as there seemed to be a slight bulging of the right lower ribs, it was determined to make an exploratory puncture in this position. The needle of the aspirator was introduced, and the syringe was soon seen to fill with pus. Forty ounces were withdrawn; and in a few weeks the patient left the hospital well.

Such a case as this contrasts strikingly with those which are given by the older writers on the diseases of India as showing the danger of opening hepatic abscesses. In that country the practice of thrusting trocars into the liver is of ancient origin, and even when no pus is thus obtained it appears seldom to be dangerous and often beneficial—possibly in preventing the formation of an abscess. With antiseptic precautions and better means of diagnosis, surgeons in India and China now aspirate hepatic abscesses with boldness and success. Usually, after once tapping, an incision is made and a drainage-tube inserted. Some surgeons provide for safe adhesions being formed by previously stitching the liver to the abdominal walls. If the aspirator should not be at hand, an ordinary trocar might probably be used quite as safely, provided that the carbolic spray were used, and the other details of the antiseptic method were attended to. It is now fully established that the introduction of an instrument of this kind into the substance of the liver is not of itself attended with any risk. When an opportunity arises of examining the parts soon after the performance of such an operation one can scarcely ever discover the track of the trocar. But a strong objection to puncturing an abscess without an aspirator is that the pus is very likely to refuse to flow through the cannula.

This occurred in a case which was under the author's care in 1875; and it was not until suction was made that it began to escape. Eleven ounces of viscid reddish pus were then drawn off; the man had not the slightest elevation of temperature afterwards, and was kept in bed only as a matter of precaution. The tumour altogether disappeared. He had been in foreign service as a mariner off the Indian and West African coasts. But he had bought his discharge four years before he came into the hospital, and since then had been working as a carman. It appeared that he had suffered severely from diarrhœa, but had not had dysentery.

Even when the trocar does not reach any pus, marked relief may be afforded by its introduction into an inflamed liver. In a case of Dr Ralfe's at the Dreadnought Hospital ('Lancet,' ii, 1876), in which hepatitis came on in the course of an attack of dysentery, only a small quantity of blood flowed into the aspirator syringe, but the patient declared that he felt instantly relieved, and did in fact experience no more pain in the right hypochondrium, where it was before severe; and his temperature, which had ranged from 99·4° to 102·2°, fell in two days to normal.*

Chronic Interstitial Hepatitis, or Cirrhosis of the Liver.—In strong contrast to the acute suppuration of the liver just described, is an insidious and very chronic process of inflammation, which is unattended by fever, and tends, never to suppuration but to the production of fibrous tissue, which shrinks and thus strangles the secreting parenchyma. The condition in question is that known as *cirrhosis*, or granular disease of the *liver;* or more familiarly as "gin-drinker's" or "hobnailed" liver.

Anatomy.—A liver affected with cirrhosis presents very remarkable

* See the papers by Dr Harley and Messrs Willett, Marsh, Thornton, and Lawson Tait in the 'Brit. Med. Journ.' for Nov. 13th, 1886.

characters. Instead of being red, it is pale, and mottled with grey and yellow tints. It is exceedingly tough and hard, so that one may be unable to crush it by the pressure of the finger; its tissue may even creak when cut with a knife. Its external surface is not smooth and even, as in health, but presents numberless round elevations of all sizes, from small granules up to the size of peas, or even larger. On section similar islets are found closely packed throughout its substance. They are often of a bright yellow colour, and on this account Laennec invented the name of cirrhosis for the disease (κιῤῥός, yellow). At this time, indeed, they were regarded as morbid products, and Morgagni and others spoke of them as "tubercles." The microscope, however, shows that they consist of liver-substance, which is scarcely distinguishable from the tissue of the healthy organ. The material which is really morbid is that which lies around and between the yellow bodies. This is a greyish, somewhat translucent substance, which under the microscope is found to consist of cellular elements and of fibrous tissue, in different proportions in different cases. Sometimes, but very seldom, it is made up entirely of cells, constituting what is termed embryonic tissue. This is in the earliest stage of the disease, when death rarely occurs. In the great majority of instances it consists of well-developed fibrous tissue, which may contain small aggregations of similar cells, or be separated by a definite layer of them from the healthy liver-substance. The presence of cellular elements is a proof that the disease was still advancing at the time of death.

It is this fibrous material which gives the cirrhotic liver its peculiar characters. The early embryonic tissue lies in the angles between the hepatic lobules, around the terminal branches of the portal vein. As it undergoes development its separate portions coalesce. Thus they gradually form fibrous rings, surrounding the hepatic lobules, or groups of lobules. But (like all young connective tissue) the new material has a strong tendency to contract. It therefore compresses the secreting cells contained in the lobules. Some of these undergo absorption, allowing the adjacent fibrous rings to coalesce. They may thus form extensive tracts of a whitish-grey colour, containing only here and there a few isolated groups of hepatic cells. Other lobules, again, become squeezed up into the rounded yellow granules or nodules above described. The reality of the compression is evident from the fact that, when a section is made through a cirrhosed liver, the yellow masses at once rise and project above the cut surface; the secreting cells in the yellow masses do not lie in definite columns, but are disarranged, so that it is usually impossible to determine how many original lobules each mass contains.

The distribution of the blood-vessels in a cirrhosed liver is very remarkable. If the hepatic artery be injected, one finds that the new fibroid tissue, which looks quite bloodless, is abundantly supplied with vessels from this source. But the branches of the portal vein often appear to be almost completely obliterated. Rindfleisch says that in one case he found it impossible to force injection beyond the three or four main divisions of its trunk. He therefore supposes that in this disease the bile is mainly elaborated from arterial blood.

As a rule, the hepatic ducts are but little obstructed in cirrhosis. But isolated nodules are often found to be of a dark yellow or green colour, which is evidently due to local jaundice, caused by obstruction of the corresponding ducts.

Size of the cirrhotic liver.—In advanced cases the liver is generally smaller than natural, and sometimes it is very greatly reduced in weight. The *post-*

mortem records of Guy's Hospital record several instances in which the organ has weighed as little as thirty-two or thirty-four ounces, and cases have been recorded in which it has been still smaller. But not uncommonly a cirrhosed liver is found after death to be of the natural size, or even above it. Whenever considerable enlargement is present, I believe that the organ is almost always also loaded with fat. We have had one remarkable instance in which a liver, which was of gristly hardness, weighed nine pounds; it contained so much fat that it would actually float in water.

It is, however, a question whether a minor degree of enlargement of the liver does not constantly occur at an earlier stage of cirrhosis. That this should be the case is evidently quite consistent with what we know to be the pathology of the disease. Unless the wasting of the hepatic cells goes on from the very first, *pari passu* with the growth of new fibroid tissue, the organ cannot but increase in size. Bright long ago stated that he had traced the enlargement of the organ when cirrhosis was beginning, and its gradual diminution towards the more confirmed stages of the affection. But the physician very rarely has an opportunity of observing this. Cirrhosis can seldom be diagnosed with certainty until it has reached an advanced stage; and the abdomen is then often so distended as to prevent an accurate determination of the size of the liver.

It has not been proved that an appreciable enlargement of the liver is always present at the commencement of the disease; while, on the other hand, it is certain that fatal effects often manifest themselves at a time when the organ is still not much below the natural size, and even when it is far larger than natural. Hence, when one finds a patient with a large granular liver, it is a grave error to suppose that his prospects are necessarily better than if the liver were small.

Ætiology.—The main *cause* of cirrhosis of the liver is almost universally believed to be the abuse of alcoholic liquors. The terms "hobnailed liver" and "gin-drinker's liver" have long been accepted in this country as synonymous. Dr Dickinson found that this disease was present in 22 out of 149 persons whose trade it had been to make or sell or carry stimulants, and who died in St George's Hospital; while it occurred in only 8 out of 149 other persons who had been unconnected with the liquor traffic, and who therefore may be presumed to have included a smaller number of the victims of intemperance. It has been objected that the disease is sometimes seen in children; but recent observations appear to show that this may really be an argument in favour, rather than against, its alcoholic origin. Dr Wilks some time since had a little girl, eight years old, under his care at Guy's Hospital suffering from what proved to be a very small hobnailed liver; she had been fed on gin, and had taken as much as half a pint daily. Niemeyer quotes from Wunderlich two very similar cases which occurred in sisters, aged eleven and twelve years respectively, who had each drunk spirits to excess. Moreover, the disease is seldom or never met with among those who are known to have been temperate in their habits. It has been supposed that other ingesta besides alcohol may set up this form of chronic inflammation in the liver; and the excessive use of spices and other condiments has sometimes been charged with causing it. Niemeyer records a case in which he supposed that it was set up by the presence of gall-stones in the hepatic ducts; and persistent jaundice from obstruction of the biliary passages appears more or less constantly to lead to an overgrowth of the connective tissue in the portal canals—in fact, to a slight degree of

cirrhosis. Obstructive heart-disease, again, was once believed to be a cause of cirrhosis; but it is exceedingly doubtful whether this ever by itself gives rise to the affection, although, by retarding the circulation through the liver, it may probably enable comparatively moderate quantities of alcohol to produce it. Persistent intermittent fever is another condition that has been supposed to be followed by cirrhosis of the liver, but Frerichs seems to be right in thinking that this cause does not operate by itself. The idea that syphilis may be one of the causes of the affection may now be dismissed; for the hepatic disease which results from syphilis is really quite different, as we shall presently see.*

In stating that excessive indulgence in alcoholic liquors is the principal cause of cirrhosis, however, one cannot deny that the affection is occasionally met with in children and others who are very unlikely to have been intemperate. And the late Dr Anstie, in investigating certain other effects of chronic alcoholism, found that in the immense majority of his cases there was no marked symptom of disease of the liver, nor did such symptoms show themselves, although he watched the patients for a considerable time. He therefore inferred that the abuse of stimulants could not be the cause of cirrhosis. But in drawing this conclusion Anstie cannot have allowed sufficient weight to the fact, of which he was doubtless aware, that this disease often reaches an advanced stage without manifesting itself by definite symptoms. At Guy's Hospital, the liver is accidentally found cirrhosed (in persons who have died of injury or of some other disease) once for every two cases in which cirrhosis has been the cause of death. In many of these cases, the organ is indurated in an extreme degree, yet the patient certainly suffered from none of the more marked symptoms of such an affection; so that there can be little doubt that in many of Anstie's cases the liver was really more or less cirrhosed.

The frequency with which cirrhosis of the liver is thus latent is, in fact, one of the most remarkable features of the disease; but there has been hardly a single instance in which the organ has been found, under such circumstances, to weigh less than fifty ounces. This might be taken as showing that the affection was not really so advanced as it appeared to be, and that if life had been prolonged the characteristic symptoms would have developed themselves; but the cases appear to be too numerous to admit of such an explanation. Moreover, on casting up the ages of persons in whose bodies cirrhosis of the liver was discovered without there having been marked symptoms during life, it results that the average age was higher by about five years than that of those persons who died of the effects of the disease. If this fact can be relied upon, and if it should be confirmed by a wider experience, it would prove that cirrhosis is not always (as we have been inclined to suppose) a progressive disease, but rather that after having reached a certain degree of development it often remains stationary, and remains so during the rest of the patient's life.

As regards the *age* of patients who die of the effects of cirrhosis of the liver, it appears from the records at Guy's Hospital that between forty and fifty years of age there is a larger number than in any other decennial period. The proportion of males to females was 102 to 26.

Effects.—Even when cirrhosis of the liver is about to give rise to effects that will rapidly endanger life, its early symptoms are exceedingly indefinite.

* H. Mayer, Wickham Legg, and Simmonds have produced cirrhosis in rabbits and cats by ligature of the common bile-duct.

They are chiefly those which have already been described as indicative of "congestion" of the liver (p. 489); with the addition of the other symptoms of chronic alcoholism; restlessness at night, tremor of the tongue and hands, irritability of the bowels, and nausea. A knowledge of the patient's habits often suggests a suspicion that the liver may be cirrhotic at a time when there is no positive proof of it.

Portal congestion.—The digestive disorders which thus usher in the more serious effects of cirrhosis may be in part due to the impairment of the functions of the liver. But another and perhaps a more important cause of these symptoms is the interruption of the flow of blood through the portal system of vessels which results from the presence of the new fibrous tissue which is developed in the substance of the organ.

Pathologists have long found a difficulty in explaining how the blood returns from the chylopoietic viscera when the liver is affected with any considerable degree of cirrhosis. Some of it no doubt escapes through the anastomoses which exist at the upper and lower limits of the distribution of the rootlets of the portal vein. Thus, the œsophagus is often found to be surrounded by a plexus of dilated vessels, which had carried upwards a part of the blood from the stomach; and hæmorrhoids are very frequently present, which may be taken as an indication that some of the blood from the rectum had passed away into adjacent branches of the iliac veins. But these communications seem quite insufficient to make up for the great obstruction that must exist in many cases of cirrhosis. Rindfleisch states that in one extreme instance, which he investigated, the portal blood passed directly into the inferior cava through a number of dilated anastomoses between the mesenteric and spermatic veins. Frerichs lays stress upon the existence of vessels in the newly-formed adhesions between the liver and the diaphragm and abdominal wall. He also adopts the statements of Sappey with reference to certain accessory branches of the portal vein, the chief of which run along the round ligament of the liver to reach the under surface of the diaphragm. In two cases of cirrhosis Sappey found this vessel distended to the size of the little finger. Some years ago we observed a large vein in this position when attempting to inject the portal vein in the body of a patient whose liver was hobnailed.

Whatever may be the precise course taken by the portal blood, there seems to be no doubt that much of it gets into the veins which ramify over the abdominal walls, and pass upwards into the internal mammary veins. For the superficial vessels of the abdomen become greatly over-distended in cases of cirrhosis, and this can be explained in no other way, unless it could be shown that the trunk of the inferior cava itself were greatly compressed, and of this there appears to be no evidence whatever.

The congestion of the portal system of vessels which thus results directly from cirrhosis of the liver accounts for the fact that after death from this disease the stomach is generally found to be reddened and lined with mucus, and that the spleen is often enlarged. There has, indeed, been some difference of opinion about the state of the spleen. According to some writers it is almost invariably increased in size; whereas others have said that this is seldom the case. Frerichs found that the spleen was enlarged in exactly half his cases; and this statement has been adopted by most subsequent writers. Another frequent effect of the portal congestion in cirrhosis of the liver is hæmatemesis (cf. p. 339).

It must be added that congestion and dilatation of blood-vessels is not

limited in this disease to the radicles of the portal vein, but occurs likewise in distant parts of the body, where it is very much less easy of explanation. Thus a very frequent, and really a valuable, symptom of cirrhosis of the liver is the presence on the patient's cheeks of a number of minute red lines and points, consisting of minute cutaneous vessels that have become varicose—not any form of the affection known as acne-rosacea, although this too is often the result of intemperance, but what are termed "stigmata." Beside the face, they may often be found on the chest and abdomen. Hæmorrhages from the different mucous membranes and purpuric spots upon the skin are also frequent effects of cirrhosis of the liver.*

But the most important effects of cirrhosis are three : jaundice, cerebral symptoms, and ascites. Of these, ascites is by far the most frequent and important.

Jaundice, as produced by cirrhosis of the liver, has already been mentioned at p. 499, where it was stated that among 130 cases in which the liver was found after death to be hobnailed, there were thirty-four in which more or less jaundice existed, and nineteen in which it was intense. This statement, however, hardly does justice to the frequency of icterus, in comparison with the other clinical symptoms of cirrhosis, for in more than forty of the 130 cases the cirrhosed state of the liver was accidentally discovered in the *post-mortem* room. This would leave less than ninety cases in which the cirrhosis produced marked effects during life ; and among these the proportion of cases in which some jaundice was present would be more than one in every three. The liver is generally enlarged in these cases. In nearly half the instances which occurred in Guy's Hospital, with intense jaundice, the organ weighed more than seventy ounces ; once as much as 131 ounces. It almost always contained much fat.

Cerebral symptoms, especially drowsiness and coma, have been mentioned as frequently ushering in the fatal termination in cases of cirrhosis which produce jaundice. They are also common in cases which give rise, not to jaundice, but to ascites. Moreover, when, by means of diuretics and purgatives, one is able to clear the peritoneal cavity of its fluid, the successful action of such remedies seems often to avail the patient very little, for he presently becomes stupid and unconscious and dies, although his abdomen may be perfectly flat and empty. The question has even been raised whether the removal of the fluid by medicines may not have been sometimes concerned in the production of the fatal issue. Dr Carrington has pointed out that pyrexia is not infrequently present.

In addition to the drowsiness and coma which are the chief symptoms observed in cases of this kind, Frerichs mentions noisy delirium and (in one instance) spasmodic contractions of the left side of the face. We had one patient who, although he could be partially roused, seemed to be quite unaware of being in bed and in the hospital, and, when asked where he was, always named some street in the city, where he had previously lived. This man lay for two or three weeks in a semi-comatose condition.

The cause of these cerebral symptoms is obscure. It has been supposed by some writers that it results from a disintegration of the secreting cells of the liver, like that which occurs in acute yellow atrophy. But I have several times carefully examined the tissue of the organ in such cases, and have always found numerous liver-cells in an apparently unaltered state.

* Dr Carrington observed pyrexia in eighteen out of forty-four cases of cirrhosis ('Guy's Hosp. Rep.,' 1883).

The microscopical characters were, in fact, undistinguishable from those of any other cases of cirrhosis. Frerichs, however, states that in his cases a large quantity of leucin separated from the organ, and that the bile-ducts contained only a small quantity of pale bile. He therefore describes the condition in question as one of "acholia." Another theory, which has been propounded by Dr Austin Flint (of New York), is that the cerebral symptoms in these cases, as well as in those of acute yellow atrophy, are due to the accumulation of cholesterine in the blood. He supposes that in health one of the functions of the liver is to eliminate from the blood cholesterine; and in a case of cirrhosis which terminated by coma, he found that there was a large increase in the amount of cholesterine contained in the blood. He has therefore invented the name "cholestearæmia" for the state in which such symptoms are developed.

Hypertrophic cirrhosis.—Charcot believes that when these cerebral symptoms are present, with jaundice and without ascites, the liver will be always found enlarged; and that the cirrhotic change has then begun, not around the lobules in the portal canals, but within the lobules. He also believes that this intralobular hypertrophic cirrhosis is not, like the ordinary hobnailed liver, due to drink.

It is no doubt a true clinical observation that cases of cirrhosis, in which there is much icterus, run a more rapid course as a rule, although one such patient in Guy's Hospital had persistent jaundice for seven years, and died at last of hæmatemesis and not of cholæmia. It is also true that early and extreme ascites accompanies the more atrophied and contracted conditions of the liver. But certainly we meet with livers which are above normal weight in persons who have been intemperate, and who suffer from ascites with little or no jaundice.

Dr Price, now of Reading, collected the cases of cirrhosis which occurred in Guy's Hospital from 1875 to 1883 inclusive ('Guy's Hosp. Rep.,' xlii, p. 295). They were 142 in number: 108 men to 34 women. There was admitted intemperance in half the cases. In only nine was there evidence of syphilis, and in six of these there was also evidence of drink.

The weight of the liver was less than sixty ounces in 29 cases only, and in two of these the patient was below adult age (16 and 14). Excluding accidental causes of enlargement, there were 63 cases above sixty ounces. In 33 cases the weight varied between fifty and sixty ounces. Ascites was present in 58 out of 72 cases, and more often when the liver was below than when it was above the normal weight. Jaundice was present in 30 out of the 72, and more often when the liver was above than when it was below the normal weight. When death was preceded by coma, jaundice was almost invariably present. Granular kidneys were more often found in association with hypertrophic than with atrophic cirrhosis.

Charcot described the formation of new biliary ducts within the lobules in cases of hypertrophic or "biliary" cirrhosis. That these exist is confirmed by other observers, but Dr Saundby has shown that these new-formed ducts may be present when there is no jaundice, and absent when jaundice is well marked.

Ascites.—This is the most constant effect of cirrhosis of the liver. It is usually abundant and gives all the physical signs described in the preceding chapter (p. 480). It is probably a purely passive effusion.

There are, however, other affections of the liver which resemble cirrhosis in producing ascites; and since some of these require separate description, it will be convenient to give it now.

1. *Chronic inflammation of the capsule of the liver*, or, as it is often termed, *perihepatitis*, is perhaps the most frequent of these. In this affection the organ is remarkably deformed; it no longer has a sharp edge, but is converted into a rounded mass. Its capsule is opaque, and often forms a separable layer, which, when stripped off, leaves a smooth surface, just like that of the healthy peritoneum. The alteration in the form of the liver is in part caused by the contraction of this thickened capsule; but very commonly its anterior edge is also folded over on to the dorsum in a way that is difficult of explanation. In a case of this disease the margin of the liver thus touched a part of the convex surface that should have been four and a half inches distant in a direction from before backwards; and when the capsule was removed, the organ returned to its natural shape.

The weight of a liver affected with perihepatitis is generally about the same as that of a healthy organ. Its tissue is commonly soft, and is very often loaded with fat. It is seldom cirrhotic, but there is sometimes an excess of white fibrous tissue in the course of the larger portal vessels. Perihepatitis appears to be a frequent cause of ascites. At Guy's Hospital there is one fatal case of it for every five of dropsy from cirrhosis of the liver; and this proportion would be greatly increased if we were to take into account those cases in which thickening of the capsule of the liver is merely a part of a general chronic peritonitis. Unlike cirrhosis, perihepatitis seems to be very rarely found in the bodies of persons who die of other diseases or are killed by accident; whence it appears that it never remains latent, but always advances until it causes ascites. Again, in cirrhosis the kidneys are generally healthy, but in the majority of cases of perihepatitis they are diseased. It follows that, if in a case of ascites the urine be healthy, there is but little likelihood that the cause is inflammation of the capsule of the liver; and, on the other hand, that when in a case of renal dropsy the abdomen is filled with fluid to a degree disproportionate to that of the serous effusion in other parts, this is probably due to perihepatitis rather than to cirrhosis.

With regard to other than renal causes of perihepatitis, very little is known. It is often a locally exaggerated form of a case of general chronic peritonitis.

2. *Simple chronic atrophy* is another affection of the liver which may cause ascites. The 'Pathological Transactions' contain two very striking cases of this kind, in which the abdomen contained a large quantity of fluid. In one of these cases, which is recorded by Dr Cayley (1868), the liver weighed only twenty-two ounces; the left lobe had almost disappeared, being only an inch wide. In the other case, one of Murchison's (1867), the organ weighed twenty-five ounces; its margin was thin and flat, forming a kind of rim, which consisted only of connective tissue and vessels enclosed between the two layers of the capsule. This rim measured in one place an inch across. In both cases the substance of the liver was of a dark colour, and quite free from induration. Minor degrees of atrophy of the liver are by no means uncommon, particularly in old people and in those who die of wasting diseases, such as cancer of the stomach or œsophagus.

This affection seldom gives rise to symptoms. In twenty years we seem to have had at Guy's Hospital only one fatal case in which ascites could be said to have depended entirely upon simple atrophy of the liver.

3. *Syphilitic affections of the liver* sometimes cause effusion into the peritoneal cavity. These present very different characters in different cases.

Sometimes gummata are scattered through the hepatic tissue, which is in other respects healthy. This condition is generally unattended with any symptoms, but it may happen that one of the gummata is so placed as to obstruct the circulation through the organ, and thus causes ascites. A case of this kind occurred at Guy's Hospital, in which one of the hepatic veins, close to the inferior vena cava, was so narrowed that it would only just admit a probe. More often the gummata, instead of being embedded in the hepatic tissue, lie in the middle of broad fibrous bands, which traverse the liver from one surface to the other, forming deep notches or depressions, or cutting off large masses from their continuity with the rest. Again, beside containing gummata and fibrous bands, the liver is very often lardaceous; and ascites is especially apt to occur in the cases last mentioned. The organ may then reach a very great size, weighing from six to seven pounds, while the capsule is generally thickened and adherent to adjacent parts. A striking case, which appears to have been of this kind, was recorded by Dr Grainger Stewart. A patient had ascites, for which she was tapped twenty-one times, the enormous quantity of 12,120 ounces being removed in the course of these operations. At first the paracentesis had to be repeated every fortnight, but the intervals gradually became longer, until at length she regained tolerable health. In twenty years (1862–82) there occurred in Guy's Hospital about six cases of fatal ascites due to this kind of disease. In several of them the liver could be felt during life to be enlarged and adherent to the parietes, with an uneven and nodular surface, and these characters more than once enabled a correct diagnosis to be made.

4. *Carcinoma of the liver* is another disease that may give rise to effusion of fluid into the peritoneal cavity, but comparatively seldom in large quantity. The presence of malignant growths in the substance of the organ itself is to be distinguished from the cases in which cancer merely involves the different structures in the portal fissure. Both affections will be discussed in the following chapter (*v. infra*, p. 538).

5. In another class of cases, the trunk of the portal vein, not its branches within the liver, is obstructed by thrombosis as the result of plastic inflammation—*pylephlebitis adhesiva*. This, however, is a very rare condition, and is seldom seen except in association with advanced cirrhosis or capsulitis of the liver, by which the circulation through the vein had been evidently greatly obstructed before death. Frerichs states that thrombosis of the portal vein may be suspected when ascites develops itself very rapidly, particularly if the fluid should reaccumulate quickly after tapping. But the reports of cases given by this writer fail to establish this statement. Indeed, it appears that the flow of blood through the portal vein is often arrested by cirrhosis of the liver as completely as it could be by an actual obliteration of the vein; and it is certain that the fluid may collect again with remarkable rapidity after paracentesis when cirrhosis is the cause of ascites, without there being any further obstruction from thrombosis of the vessel. A case of ascites from adhesive portal phlebitis occurred many years ago in a boy of ten at Guy's Hospital: no other lesion was found.

The *prognosis* of cirrhosis is always very grave, though we have some evidence that it may be latent or possibly may be arrested (*supra*, p. 525). But when it has caused ascites the fatal result is well-nigh inevitable (p. 484).

Its *treatment* is first that of hepatic dyspepsia (p.491), and afterwards

that of ascites (p. 485). The patient must at any stage, except perhaps the latest, be induced, if possible, to give up the use of all intoxicating liquors.

ACUTE YELLOW ATROPHY OF THE LIVER.*—For many years it has been known that cases of apparently idiopathic jaundice occasionally do not run a favourable course, but develop hæmorrhages and cerebral symptoms, and end in death by coma. These were called *icterus gravis*, or malignant jaundice. But the first clear and complete cases were published by Bright in the first volume of the 'Guy's Hospital Reports' (1836), under the title "Intense Jaundice without Mechanical Obstruction, apparently depending upon Inflammatory Action in the Liver." The following is his account of these two typical cases of acute yellow atrophy (Nos. 5 and 6, pp. 624—630).

(1) A woman aged twenty-eight, of dissolute habits, while taking mercury, was attacked by abdominal pain, and jaundice quickly followed. Dr Bright saw her on the third day; the urine was then bile-stained and the fæces clay coloured; on the twenty-first there was drowsiness, followed by delirium. She died comatose on the twenty-third day of the jaundice.

At the autopsy there was intense staining of every tissue with bile; the organs of the head, chest, and abdomen were healthy except the liver, which weighed only thirty-seven ounces, and was "soft or flaccid to the touch," with no trace of peritoneal inflammation.

(2) A German girl aged eighteen, was admitted into Miriam Ward, January 11th, 1832, labouring under icterus. "The skin was of a brilliant yellow, and the cheeks, which were flushed, were the colour of a very ripe apricot." She had probably been ill for nearly four weeks, and the jaundice had gradually deepened to its present tint. She had lately sat by the fire in a kind of doze. That evening she vomited, and "lay in a perfectly torpid state the whole night, apparently suffering no pain; but towards the morning became delirious, so that it was with difficulty she could be restrained in her bed." Dr Bright ordered "two grains of calomel every two hours and the ammonia julep (Mist. Ammoniæ Co. of the present Guy's pharmacopœia) every four hours, besides wine if she became more depressed." Purging enemata and a blister over the liver were also ordered; the head was shaved and mustard was applied to the feet. She continued, however, completely comatose all the following night and died at ten in the evening of January 13th.

At the autopsy, excepting a universal and deeply jaundiced tint, the brain, heart and lungs, stomach and intestines, pancreas, and kidneys were found normal. The spleen was soft. "The liver was unusually small, and for the most part of a brightish yellow colour, with portions marked with purple or deep brown."

Dr Bright remarks: "In this case, as in the last, no obstruction could be discovered in the ducts which could have prevented the flow of bile from the liver." "The immediate cause of death in this case, as in the last, was the poisoning influence of the bile on the system." "The bile must have been rapidly absorbed into the system almost at the moment of its formation, and its profuse mixture with the blood seems to have acted as a poison, and hence the immediate cause of death. I am inclined to

* *Synonyms.*—Icterus gravis—Icterus typhoides (Lebert)—Irish yellow fever—Ictère malin—Ictère hémorrhagique essentiel—Die acute Schmelzung (Rokitansky)—Atrophia hepatis flava sive acuta (Frerichs)—Hepatitis parenchymatosa diffusa.

consider this as the result of a decidedly inflammatory state of the organ." Again, he remarks that, in these severe cases of jaundice with nervous symptoms, "the tendency to hæmorrhage comes on very early and is excessive."

A few years later, in 1843, Graves, of Dublin, published in his famous 'Clinical Lectures' (2nd ed., 1848, vol. ii, p. 255) three cases of jaundice in sisters. One, a girl of seventeen, was attacked in July, 1840, with vomiting and icterus, followed by hæmatemesis, violent delirium with convulsions, coma, and death on the seventh day. No autopsy was permitted. The second, aged eleven, died in March, 1841, with similar symptoms on the fifth day. After death, the liver was of "natural size," dull yellow with dark spots, and bile in the gall-bladder; the brain (examined first) was much more vascular than usual; the thorax was not examined. The third sister, aged eight, was taken ill with jaundice and vomiting the following June. She was actively treated by bleeding, leeches, and calomel, with James's (the compound antimonial) powder; the alarming symptoms disappeared after three days, and she recovered from her jaundice in about three weeks more.

Graves's cases were afterwards referred to by Budd and by Trousseau, as well as by Frerichs in his well-known treatise on disease of the liver. But there is no evidence that the last was anything more than ordinary icterus; the first is incomplete, and in the second there was no atrophy of the liver discovered.*

Acute yellow atrophy of the liver is not a common disease. Murchison says that although delirium and a brown tongue constituted a certain passport into the London Fever Hospital, only one case occurred among 3000 patients admitted in a period of six years. In Guy's Hospital there are notes of the inspection of eight cases in twenty years, 1864—84.

Anatomy.—The first thing that strikes one, in making a *post-mortem* examination in a case of this kind, is the diminution in the size and weight of the liver. The organ forms a thin flaccid mass, which lies at the back of the abdomen, hidden by the ribs and by the intestines, which last are commonly over-distended. Instead of weighing from fifty to sixty ounces—the usual weight of a liver at the period of life at which acute atrophy commonly occurs—it weighs perhaps thirty-two ounces, thirty ounces, or even as little as twenty-three ounces. When cut into, it looks as though it were softened; but the finger is found not to penetrate it more readily than the healthy organ. Although it is so flabby, it is not really deficient in firmness. Its colour is greatly altered. Most of it has a bright orange-yellow tint; but some parts are dark red or purple. Sometimes roundish masses having this red or purple hue are scattered through the substance of the organ; sometimes one part, generally the left lobe, is almost entirely red, while the rest of the liver is mainly of a gamboge-yellow hue. To the naked eye it appears as though the red parts were less altered than the yellow, but the microscope shows that this is not the case. In both parts the hepatic cells have undergone destruction, and are replaced by a mass of granules and oil-globules; but in the red parts the destruction is complete, whereas in the yellow parts some of the secreting cells still remain visible, and towards the centres of the lobules may even retain their columnar

* Isolated cases of rapid and fatal jaundice which may with more or less probability be referred to yellow atrophy, have been quoted by Frerichs and Trousseau from Rubæus (1660), Boerhaave and Morgagni.

arrangement. Among the remnants of the hepatic tissue are often to be seen crystals of leucine (amido-caproic acid) and tyrosine (amido-sulpho peruvic acid), both products of albuminous decomposition. The former substance presents the appearance of rounded, flat discs, generally marked with concentric rings; the latter occurs in bundles or globular masses of needle-shaped crystals.

In some cases it has been thought that a pellucid nucleated material could be detected, supporting the detritus of the cells in acute atrophy, and this was believed to be the case in two cases examined by the writer. Lastly, Waldeyer and Klebs have described, in the reddened parts, cells resembling those of the epithelial linings of the biliary ducts, arranged in regularly branching lines and tubes which seemed to have cæcal terminations.

The bile-ducts are found to be empty; their mucous membrane is unstained by pile-pigment. The gall-bladder is either empty or contains a few drachms of grey mucus or of a pale yellow or greenish fluid.

The kidneys can very generally be shown by the microscope to have undergone morbid changes. The epithelium of the tubules is granular and may be very fatty.

Nervous symptoms.—In addition to the jaundice, these are mainly cerebral. Headache and intolerance of light are often first complained of. Before long the consciousness is more or less impaired. The patient now becomes very restless, screaming and tossing about from one part of the bed to another. Violent convulsions sometimes follow. Ultimately a state of complete coma is developed; the pupils become widely dilated and insensible to light; the urine and fæces are passed involuntarily; the breathing is stertorous; and the scene ends in the death of the patient, almost always within five days from the commencement of the characteristic symptoms. It is, indeed, said that acute yellow atrophy has sometimes destroyed life in less than twenty-four hours. According to Niemeyer the majority of cases end fatally on the second day, but this is certainly not the case.

Physical signs.—It is possible by means of *percussion*, to trace from day to day the gradual diminution of the liver. From a normal measurement of four inches vertically in the right mammary line—reaching from the fifth intercostal space downwards to the costal margin—the hepatic dulness may be watched as it undergoes reduction to three inches, two inches, and one inch, until at last it disappears entirely. One must not, however, suppose that the diminution of dulness over the liver is necessarily due solely to the wasting of the organ. As we have seen, the liver in acute yellow atrophy is remarkably flaccid and falls backwards away from the ribs, so that the intestines, if distended, ride over it. Indeed, this source of fallacy is not confined to cases in which the liver is really affected with acute atrophy. In all forms of jaundice the bowels are apt to become inflated with gas, and the consequent enlargement of the abdomen may cause the right hypochondrium to become gradually more and more tympanitic, and the area of hepatic dulness to diminish from day to day. This has led to the error of supposing the liver to be in a state of acute yellow atrophy, when such was not the case.

State of the urine.—This does not generally contain a large quantity of bile-pigment. It does not look black when in bulk, nor in a thin layer has it so intense a saffron-yellow colour as in some other forms of jaundice. It is said that Gmelin's test may give an imperfect reaction, or fail altogether to indicate the presence of the colouring matter of bile. The principal

change in the urine is of a kind which at first sight would appear to have no relation to the disease in which it occurs. The urea and uric acid, and also the chlorides, sulphates, and earthy phosphates are greatly diminished in quantity or are altogether absent; and in their place are found two new substances—*leucine* and *tyrosine*—the same which have already been described as being present in the substance of the liver itself. There is generally no difficulty in detecting these substances. They sometimes form a distinct deposit when the urine is left to stand for a time, or, if this is not the case, they may be made evident by evaporating a few drops of it on a glass slide. Tyrosin is easily recognised by its taking the greenish yellow colour of the urine. In some cases, however, they cannot be discovered, at least without the adoption of a more complicated procedure. The urine is, as a rule, albuminous; but it is uncertain whether this should be regarded as a symptom of a constantly concomitant nephritis (as Grainger Stewart believes) or as "febrile," or as the result of the presence of leucine and tyrosine.

Hæmorrhage.—In some other forms of jaundice there is a tendency for the blood to escape from the vessels, and this is especially marked in acute yellow atrophy. Very frequently the patient vomits a dark fluid resembling coffee grounds, and containing altered blood. Petechiæ are often developed in the skin, and almost invariably, towards the end of the case, the evacuations have a dark brown or a tarry black colour, which is really the result of hæmorrhage.

The stools.—With regard to the colour of the fæces in acute yellow atrophy different writers have made different statements. Murchison says "the jaundice appears to be due to a poisoned condition of the blood, and consequently bile is still found in the stools." The remark has often been made that it is a good sign for the motions to be clay coloured in jaundice, there being then less danger of the supervention of cerebral symptoms. But it is at any rate certain that towards the end of a case of acute atrophy no bile enters the intestines, for after death the ducts and gall-bladder are found to contain an almost colourless mucus. Moreover, Frerichs says that in this form of jaundice the stools are dry and clay coloured; and more than one case is recorded at Guy's Hospital in which such was the case. The question is not so easy of determination as might at first sight appear, on account of the great frequency of intestinal hæmorrhage in this disease. When the stools have been supposed to contain bile in acute atrophy of the liver, their dark appearance has generally been due to altered blood. We must, however, remember that the disease does not affect the whole substance of the organ uniformly, but attacks some parts earlier than others. Thus, at its commencement bile very possibly continues to enter the intestines from those portions of the liver which have not yet become diseased.

The patient soon falls into a "typhoid state." His tongue is almost always dry and brown, and his lips and teeth are encrusted with sordes. According to Niemeyer and other writers, the temperature of the body is raised considerably above the normal. But in several of the cases that have occurred at Guy's Hospital the temperature was normal. In one instance it was below the average four days before death, but it began to rise two days later, and while the patient was dying it was found to be 101·6°. Dr Duckworth noted the absence of pyrexia in three cases that were observed in St Bartholomew's Hospital. Frerichs says that in his cases the skin was usually cool, dry, and inactive, and he quotes Bright and

Addison as having made particular mention of the same circumstance. The pulse is almost always accelerated, but in one of Dr Duckworth's cases it was on two days about 50. Towards the end it becomes very small and intermittent.

When acute yellow atrophy occurs in a pregnant woman, *abortion* or miscarriage almost always precedes the patient's death.

The course and event of acute yellow atrophy is not absolutely constant. A few instances of recovery have been recorded. Not long ago Dr Wilks had at Guy's Hospital a fatal case in which there was a distinct history of a previous attack that had been recovered from. The patient had become delirious and had such severe hæmatemesis that it was thought he would die in a few hours. However, he rallied and lived two months longer, at the end of which he again became delirious. Leucine and tyrosine were found in the urine. He died a fortnight later. The liver was found by Dr Moxon to weigh forty-seven ounces. The left lobe and the adjacent part of the right lobe were small and dark looking. The lobules in them were distinct, but scarcely any hepatic cells were to be seen. The rest of the right lobe formed a soft, yellow, rounded, projecting mass. The marked contrast appeared to justify the supposition that the left lobe had become atrophied at the time when the cerebral symptoms first arose. Another case of recovery followed by a second fatal attack was recorded by the late Dr Frank Smith, of Sheffield, 'Path. Trans.,' 1877, p. 236.

Ætiology.—The origin of the disease is exceedingly obscure. There are several different conditions, each of which has appeared in certain cases to be its exciting cause, and its occurrence seems to be favoured by other conditions, which must therefore be regarded as causes predisposing to it. Among the exciting causes, mental emotions seem to take a foremost place. It has already been stated that fear or grief is a frequent cause of simple jaundice, and cases arising in this way may prove fatal by the supervention of cerebral symptoms. Again, more than one instance has been recorded in which it has followed directly upon a drunken debauch; and in several cases it has set in during the secondary stage of constitutional syphilis. It might be argued that one cannot in either of these conditions exclude the possibility that the jaundice was really due to mental anxiety or remorse. Persons affected with syphilis in particular often undergo great mental torture which they carefully conceal. Pregnancy is another cause to which the disease is occasionally ascribed, and apparently with justice, for out of twenty-two female patients referred to by Frerichs one half were pregnant. But here again mental influences may possibly come into operation.

Sex and age.—Even apart from pregnancy, acute yellow atrophy of the liver is more common in women than in men.

It occurs principally at an early period of life. Five times out of six the patient is under thirty years of age. It is, however, rare in childhood, although Dr Goodhart had under his care a typical case in a boy only two and a half years old.

Pathology.—Some of the earlier writers on acute yellow atrophy of the liver, having found after death that the larger bile-ducts were free from obstruction, conceived the idea that the minute channels which issue from the secreting lobules of the organ might have undergone compression in consequence of swelling of the cells forming the periphery of the lobules themselves. And they supposed that the jaundice was really due to reabsorption of bile secreted by the cells forming the centres of the lobules.

Rokitansky even imagined that the breaking down of the hepatic cells was due to a solvent action excited by the retained bile. These views, however, can be controverted by evidence of great weight, and at the present day pathologists are pretty well agreed in believing acute atrophy of the liver to be *parenchymatous inflammation* (in Virchow's sense of the term), although one must admit that no precisely analogous disease can be found among the affections to which other organs are liable (cf. p. 513). This, we have seen, was the view originally taken by Bright (p. 532).

Some uncertainty still prevails with regard to the origin of the leucine and tyrosine, which, as we have seen, are excreted in the urine in this disease. Most authorities suppose that in acute atrophy of the liver the chemical changes which should be undergone by albuminous substances in the blood are incomplete, so that instead of urea and uric acid the new bodies in question are formed, and this view accords well with the fact that urea and uric acid are more or less completely wanting. But other writers, basing their opinion on the fact that the healthy liver during decomposition contains leucine and tyrosine, think that these substances are the direct products of the disintegration of the hepatic tissue.

Again, there is a doubt whether the granular and fatty changes in the epithelium of the renal tubules are the result of the disease of the liver, or whether both these conditions do not rather depend upon some common cause. The former view appears to the author to be the correct one, for the morbid changes in the kidneys are often comparatively slight.

What is the cause of the cerebral symptoms which form so striking a feature in acute atrophy of the liver? Frerichs thought that they depended upon the presence of leucine and tyrosine in the blood, but experimenters have hitherto failed to verify this supposition. Rokitansky started the theory that these symptoms were really uræmic and dependent on the renal changes. But the character of the cerebral symptoms in acute atrophy of the liver is not the same as in uræmia.

It is possible that the disease may prove to be "specific;" Waldeyer and other pathologists have found microphytes in the liver, but they appear not to be constant. See Dr Cavafy's case ('Path. Trans.,' 1883).

Diagnosis.—This is not difficult in most cases, if attention be paid to the various points referred to in describing the disease: particularly the rapidly diminishing liver dulness, the hæmorrhages, the delirium, the stupor, and the presence of albumin, leucine and tyrosine, as well as of bilirubin in the urine.

The affection which most closely resembles acute yellow atrophy is one produced by *poisoning with phosphorus.* Within the last few years it has been shown that the toxic effects of this substance are by no means limited to the vomiting and purging which immediately follow its ingestion. In a few hours these generally pass off, and often the patient appears to be perfectly well for three or four days; but at the end of this time jaundice sets in, followed by delirium and coma, and these symptoms ere long prove fatal. According to some observers, the liver is then found to be altered exactly in the same way as in cases of acute yellow atrophy; they therefore speak of phosphorus-poisoning as one of the causes of this disease, but probably the appearances are always distinguishable from those seen in acute yellow atrophy. In some cases of poisoning by phosphorus at any rate, it is certain that the liver presents characters which are very different. It is larger than natural, of normal shape, and of a pale buff colour, mottled with numerous ecchymotic spots. Under the microscope the principal change is the

presence of an immense quantity of fat, in large drops as well as in minute granules, within the hepatic cells, the walls of many of which are indistinct. One may therefore at first feel disposed to think that they have undergone destruction; but there is often a similar difficulty in detecting the hepatic cells in other cases of fatty liver in which there was every reason to suppose that their walls are simply obscured by the fatty globules.

Clinically also the effects of poisoning by phosphorus appear to be different from the symptoms of acute yellow atrophy. In the former affection leucine and tyrosine are not found in the urine. The liability to hæmorrhage, however, forms a prominent feature in both diseases. After poisoning by phosphorus the uriniferous tubules are loaded with highly-refracting granules, like those in the hepatic cells, and the fibres, both of the voluntary muscles and of the heart, are found to have undergone a granular fatty degeneration. Thus the morbid state produced by phosphorus appears to be an acute steatosis of the liver, kidneys, and muscles. In England such cases are rarely seen, but in Germany they are far from uncommon. Persons who wish to commit suicide there seem to use the heads of a bundle of lucifer-matches, just as among us they employ white arsenic for the same purpose.

In a case of poisoning which occurred at Guy's Hospital, the temperature of the body was very low, at least for some hours before death, the thermometer standing in the axilla at 96·8°, and afterwards at 91·5°.

In another case under the editor's care a woman destroyed herself and her child (of five years) with phosphorus. The latter perished quickly as if by exhaustion, with little vomiting and no pain; but the mother lived for several days, and at first appeared to be recovering. There was no marked jaundice, no hæmorrhage, and no cerebral symptoms, but she sank rapidly into coma. After death the liver was found in a state of fatty degeneration, and the same process had affected the kidneys and the heart, but there were no appearances like those of acute yellow atrophy. In the child the liver was very fatty, but the heart and kidneys were unaffected.

Treatment.—No remedies have yet been shown to be efficacious. Our recent cases of this most singular disease have not been actively treated, for it was taken for granted that they must terminate fatally. However, there are, as we have seen, a few exceptions to this rule; and, since the disease seems not to attack the liver as a whole, but generally to spread through the organ from the left lobe, there appears to be no reason why it may not be opposed by medicines. Dr Budd recommended a mixture containing a drachm of the sulphate and fifteen grains of the carbonate of magnesia with half a drachm of the spiritus ammoniæ aromaticus three times daily. This advice seems to have been founded upon the brilliant results which certain Irish physicians formerly obtained from purging in similar cases of icterus gravis. The most striking examples were the cases recorded in the year 1834 by Dr Griffin, of Limerick. Four children of the same parents were attacked within a few weeks by jaundice, with cerebral symptoms. Two of them died, but two recovered after having been in a state of almost complete coma. The treatment was the same which failed in the hands of Dr Bright —bleeding, blistering, and active purging.

NEW GROWTHS, DEGENERATIONS AND PARASITES

ATTENDED WITH ENLARGEMENT OF THE LIVER

Malignant disease—Carcinoma hepatis—its anatomy and histology—its rarity as a primary disease and its most frequent antecedents—its symptoms and course —Cancer of the biliary passages.

Hypertrophy of the liver—Lymphatic overgrowth—leuchæmia hepatica—The fatty liver—general obesity and its treatment—Malarial enlargement—Lardaceous disease—Hydatids of the liver—and of other organs—Other parasites.

CARCINOMA.—Malignant disease of the liver has long been known to pathologists and is a very frequent form of disease. But it is rarely primary, almost always following either cancer of the stomach, rectum, or some other distant organ, or else cancer of the gall-bladder or ducts.

Anatomy.—The usual form of cancer of the liver is that of numerous nodules scattered irregularly through its substance; but almost constantly some of them reach the surface. They may be seen as minute white or yellow points not bigger than hepatic tubercles, but some are almost certain to be larger than this, and they grow from the size of peas to large masses as big as one of the lobes of the liver. In fact the heaviest livers recorded are cancerous.

These lumps are usually soft (*encephaloid* or *medullary*), and yield an abundant white juice on scraping. They often undergo caseous degeneration in the centre, and still more frequently are so vascular that hæmorrhage takes place into their substance, so as to give rise to the term formerly applied to them, as to the vascular excrescences of mammary cancer —*fungus hæmatodes*—and occasionally they become almost cavernous in structure. As the result of central softening, the great tubers which are seen on the surface of the liver are marked by a depression in the middle which gives them a very characteristic umbilical aspect, like the leaves of navel-wort or the seeds of nux vomica.

In exceptional cases, the cancerous nodules are much slower in growth, harder in texture, and more uniform in size. The disease has then been called scirrhus (*carcinoma fibrosum*), and sometimes distorts the liver so uniformly and renders it so tough, that to sight and touch it exactly resembles the cirrhotic livers with the larger and less regular kind of "hob-nails." So close was the resemblance in a case lately under the writer's observation that it was a surprise when the microscope showed the true nature of the transformation (Guy's Path. Museum, prep. 1922[31]).

Whatever the form, the histological structure of the cancerous tumours of the liver is almost invariably the same, that of typical glandiform carcinoma. Cylindroma is occasionally seen, epithelial (corneous) cancer still less frequently, and most rarely of all, colloid carcinoma.

In most of the cases formerly described as primary cancer of the liver, the growth probably began in the gall-bladder, gall-ducts, or in the portal fissure.

When the disease is truly primary, it sometimes assumes a remarkably infiltrating character. In a case of this kind under the writer's care in 1878 and 1879, the liver was enlarged to the enormous size of 200 ounces. There was no other trace of cancer in the body, and other remarkable features of the case were its long duration, its painlessness, and the youth of the patient, who was a boy of only thirteen.*

When the organ is affected with this diffused form of cancer, the appearance of its section is very peculiar. The lobular markings are everywhere plainly visible, but they are coarser than is natural. The substance of the liver is of a greyish colour, or even white; all parts of the cut surface yield a milky juice, and the microscope shows that the cells in the lobules have the character of cancer-cells, although they are arranged in radiating columns, occupying the meshes of the blood-vessels, like the secreting cells of the healthy organ. It is probable that these cells are directly derived from those of the pre-existing hepatic tissue.

The presence of numerous and large nodules greatly increases the size of the liver, so that the largest livers observed are those affected with cancer. Two have been observed at Guy's Hospital which weighed each 18 lbs. In a case recorded in vol. xxiii of the 'Pathological Transactions' a liver, which was full of cancerous tubera, weighed 19½ lbs.; another case is there alluded to in which the weight was 24 lbs., and Dr Arthur Jones, of Northampton, met with a case in which the weight of 28 lbs. was reached.

Signs and symptoms.—Many cancerous nodules may be scattered through the liver without enlarging it enough for its edge to be felt below the ribs. But when the tubera are grown bigger, they may reach such a size that they can be felt through the abdominal walls, or may be seen to rise and fall each time the patient breathes. They are generally firm to the touch, sometimes of apparently stony hardness; but occasionally they feel very soft, so that one might imagine fluctuation in them. Indeed, their centres may really become hollowed into cavities containing fluid, as in an instance which occurred at Guy's Hospital: a cancerous tuber formed a cyst that would have held a cocoa-nut; it was so near the surface of the organ that it might have yielded fluctuation; and it was filled with a clear straw-coloured liquid. Sometimes cancerous nodules can be felt to have a central umbilicus; a sign of importance, for it is not observed in any other affection.

Instead of several distinct nodules or tubera, cancer of the liver may be felt as a single large rounded mass, projecting from the right or left lobe downwards into the abdomen, or upwards towards the chest. Sometimes, again, a cancerous liver is enlarged, without its shape being in any way altered, even when it has reached an enormous size.

* The distinction between primary and secondary carcinoma is often difficult, even in the *post-mortem* room. All authorities are agreed that scattered nodules and tubera are almost invariably secondary. Some observers think that even the largest solitary masses, and the diffused forms of cancer of the liver, are very rarely primary. But my own impression is that such a view may be stated too absolutely. It is true that a *post-mortem* examination sometimes reveals the presence of a primary carcinoma in the intestine or the vertebræ of the os innominatum, which had been before unsuspected. But it is also true that in other cases no primary disease outside the liver can be discovered on the most careful examination. One source of fallacy may be mentioned, which is, that cancer of the gall-bladder growing into the hepatic tissue has sometimes been mistaken for a primary cancer of the liver. The cavity of the gall-bladder may in such a case be so small that, lying in the centre of the tumour, it is easily overlooked.—C. H. F.

As above stated, cancerous tumours of the liver are often exceedingly vascular, and their vessels have very thin walls, so that hæmorrhage into the substance of the nodules is far from uncommon. According to Frerichs, such hæmorrhages may be so copious as to give rise to clinical symptoms—a perceptible increase in the size of the tumour, and even decided anæmia. Sometimes, again, when a vascular cancerous growth is situated just beneath the surface of the liver, the serous membrane covering it gives way, and blood escapes from its substance into the peritoneal cavity. A remarkable instance of this once occurred in our theatre, in which a large clot covered the surface of the organ. One can seldom determine, in cases of this kind, what quantity of blood has exuded, for much of it is mixed with the ascitic fluid; but it would seem that the fatal issue is sometimes more or less directly due to the rupture of the tumour, for patients in whom this has occurred have been observed to fall into a state of collapse some hours, or even as long as three days, before death.

It was shown by Frerichs that cancerous growths in the liver derive their vascular supply mainly from the hepatic artery, and that they receive very little blood from the portal vein. In proportion as they increase in size, the trunk of the hepatic artery becomes enlarged, while the area of distribution of the portal vein is diminished. The growth, however, not infrequently penetrates into the interior of one of the branches of the last-named vessel, and may then extend along its channel so as to obstruct the flow of blood through a large part of it. These facts doubtless explain the frequent occurrence of *ascites* in cases of hepatic cancer; but probably this is sometimes the result of chronic peritonitis which started from the serous covering of the organ. Thirdly, it is frequently caused by cancerous chronic peritonitis with effusion, as described above (p. 478).

Another symptom of the disease is *pain*, which is often severe, and generally accompanied by marked tenderness on pressure in the right hypochondrium. *Jaundice* is comparatively seldom present, or shows itself only when the case is about to terminate fatally. In this respect there is a wide difference between cancer of the liver itself and cancer of the structures in the portal fissure, which is a frequent cause of jaundice. There is sometimes slight pyrexia (100° or 101° Fahr., and in one case of the editor's 104·6°), for which no other cause can be found after death.

Diagnosis.—Cancer of the liver, when at all advanced, is usually easy of detection. The liver is enlarged, painful, and irregular on its surface. There is sometimes jaundice, and frequently ascites. The patient is at or beyond middle age, and usually shows signs of grave disease in loss of flesh, anæmia, and a sallow complexion. When such symptoms are present we should examine the rectum, the uterus in a woman, and the testes in a man, with a view to discover the primary seat of cancer.

Sometimes, however, there is considerable difficulty in diagnosis, and this turns almost always upon the question between cancer and cirrhosis. We have seen (*supra*, p. 524) that in cases of cirrhosis the liver is often enlarged, and can be felt below the ribs; its surface may be uneven, it is often tender to the touch; jaundice is very frequently present (p. 527); and ascites may be moderate and late in making its appearance. Moreover, intemperance in liquor does not preserve a man from cancer, and cirrhosis may develop itself in or even after middle age. The question therefore is sometimes extremely difficult, perhaps insoluble; and, as we have seen, even after death it can sometimes only be decided by the microscope (p. 538).

The *prognosis* is of course hopeless when the nature of the case is clear The only chance for the patient is in the possibility of an error in diagnosis. Treatment can only be directed, and often with considerable success, to relieving distressing symptoms. It is remarkable how long such cases linger, and even revive for a time, when apparently at the point of death.

Cancer of the biliary passages.—The exact locality and extent of the growth vary widely in different instances. In many of them examined at Guy's Hospital its original seat appears to have been the head of the pancreas; in others its starting-point was the pylorus, or the first part of the duodenum. In some it seems to have commenced in the walls of the gall-bladder, and to have passed downwards until it invaded the common bile-duct. In other instances, again, the cancer has affected the glands about the portal fissure, and then there has sometimes been primary cancer of some distant part of the intestine. The extent of the cancerous disease, again, is very variable in these cases; there may be nothing more than a small nodule, no larger than a hazel-nut, involving the walls of the common duct. When this is the case, gall-stones are generally likewise present. Or all the parts in the portal fissure may be involved in an immense mass of cancer, which may extend to the peritoneum as scattered nodules, or lead to the formation of large and numerous secondary growths in the liver. In some of these cases also, gall-stones have been found.

Malignant disease of the biliary passages is almost always true carcinoma. Sometimes, indeed, the growth looks hard and dry, and yields little or no juice on scraping. But in one case, although the growth in the portal fissure looked as if it were composed of a fibroid material rather than of true carcinoma, the liver contained large secondary nodules, the character of which was unmistakeable.

Obstructive jaundice is a constant result of this form of malignant disease, and forms one of its most striking symptoms.

When there is a mass of cancer about the portal fissure, or in the lesser omentum, the portal vein is almost always pressed upon, and ascites follows. Thus, the association of ascites with jaundice is strongly suggestive of malignant growth outside the liver; indeed, with the exceptions of cancer of the substance of the liver and of cirrhosis, this is the only disease in which these symptoms are often found together.

Sarcoma is excessively rare as a disease of the liver. When present it is probably always secondary, and often melanotic. Cavernous angioma has been observed, usually as new growths the size of a marble. Wilks and Moxon regard it as non-malignant.

The remaining structural diseases of the liver are degenerative or adventitious. Like cancer, they do not as a rule interfere with the physiological action of the liver except by accidental mechanical pressure on its duct, and are for the most part of pathological rather than clinical interest; or, if clinically important, it is because a recognition of their nature often leads to the diagnosis of a primary or concomitant lesion elsewhere. In all of them the liver becomes much larger than natural; and this increase in size is often the only indication that the organ is otherwise than healthy. In particular these affections are unattended with pain, and Murchison therefore conveniently grouped them together as "painless enlargements" of the liver.

Simple Hypertrophy.—The author once made a *post-mortem* examination of a case in which death occurred three weeks after an accident. The liver was found to project four inches below the ribs, and it weighed 130 ounces—fully double its normal weight. No morbid change could be discovered in the hepatic tissue, so that the case was set down as one of simple hypertrophy of the organ. This affection is recognised by writers on diseases of the liver, but at present nothing definite is known about it.

In diabetes the liver is usually found larger than natural, sometimes considerably so, but it never approaches the bulk just described.

Leuchæmic Enlargement.—In leuchæmia and splenic anæmia the liver often becomes considerably enlarged, owing to the overgrowth of the lymphatic tissue which forms the portal canals. The spleen is enlarged at the same time. This affection will be described in a subsequent chapter.

The Fatty Liver.—Another condition in which the liver becomes enlarged, without pain or other marked symptoms, is that in which its cells are loaded with fat. In one of our cases of this kind the organ weighed 112 ounces, in another it weighed 155 ounces, or about three times its normal weight. The 'Pathological Transactions' contain a case in which it weighed twelve pounds. A cirrhosed liver often contains much fat, particularly when it is increased in size, and then the nodules on its surface can often be felt through the abdominal walls. But primary idiopathic fatty degeneration leaves the organ perfectly smooth. Its edge is somewhat thick and rounded. In the dead body it is found to be anæmic, having a more or less bright yellow colour, but, as Rindfleisch remarks, one must not suppose that it has the same appearance during life, for it can be injected without the employment of any great force, so that the pressure of the blood probably suffices to overcome the resistance excited by the distended cells so long as the heart is beating. The organ is soft and tears very readily beneath the pressure of the finger. Its specific gravity is diminished, sometimes even to such an extent that it floats in water. When it is cut into, it greases the knife, especially if this be warmed. Fragments held in a spirit lamp will sputter, and then burn brightly.

The microscope shows that the accumulation of fat takes place within the hepatic cells, and especially in those which lie towards the periphery of the lobules. These often contain drops of oil, so large as to obscure their walls, and an inexperienced observer may suppose that the cells themselves had undergone destruction. This, however, is not the case. The oil can be extracted by ether, and the shrunken nucleated cells remain in their natural relation. In fact, the fatty liver of pathologists is only the fat-stored liver of the physiological absorption which follows every full meal; but what in health is intermittent becomes in disease constant and excessive.

As might be expected, a fatty liver can often be easily detected at the bedside. It may be found as low as the umbilicus, or even lower, and the smooth, even surface and the soft doughy feel of the edge distinguish this from other enlargements of the organ. The deficiency of resistance may, indeed, be so great that the liver slips away beneath the hand; one may then have great difficulty in feeling it, even though the parietes may be perfectly soft and yielding, and percussion may indicate that the organ is

much increased in size. This very difficulty, however, points to fatty disease of the liver as the cause of the enlargement.

Ætiology.—The conditions under which the liver is apt to become loaded with fat are numerous, but they may be divided into two main classes, strikingly opposed to one another. In one of these an excess of fat is present in the body generally; in the other there is emaciation, often in an extreme degree.

The first kind of fatty liver occurs, along with general obesity, chiefly in persons who lead sedentary lives, and who eat large quantities of rich food, particularly if they also indulge freely in stimulants. This is evidently analogous to the affection that is artificially produced in geese by the purveyors of the *pâté de foie gras.* The birds are kept in a dark place, with but little space to move in, and are crammed with a farinaceous paste. The consumption of fat within the body is thus reduced to a minimum, while its formation is increased. It first accumulates in the blood, and then is deposited in the hepatic cells.

The other kind of fatty liver cannot be so easily explained. Cases of pulmonary phthisis are those in which it most frequently occurs. This was first noticed by Louis, who found it in one out of every three bodies of those who had died of consumption. At first sight one might be inclined to attribute this to deficient oxidation of fat from interference with the respiratory function. But if this supposition were correct, the liver ought to become fatty in cases of asthma and of emphysema likewise, whereas the other diseases in which it really becomes so are such as resemble phthisis in being attended with wasting, namely, cancerous affections, ulcer of the stomach, chronic dysentery, &c. One is therefore driven to assume that in the course of progressive emaciation the blood becomes in some way overloaded with fat, which is forthwith stored up in the liver. Dr Wilks is inclined to connect the affection under consideration with the circumstance that the patient has generally been bedridden for a long time before death. According to Larrey, it is possible by keeping geese shut up in close, hot cages, without food, to induce a fatty enlargement of the liver while the birds themselves become greatly wasted.

Symptoms.—A fatty liver produces neither pain nor jaundice. It is, indeed, said that when this affection is present in an extreme degree the fæces are pale, that the amount of bile secreted is diminished, and that a sensation of fulness in the right hypochondrium may be experienced by the patient. Many years ago Addison expressed the opinion that a symptom, suggestive if not pathognomonic of the affection, was a peculiar state of the skin, which he described as looking semitransparent and pale, somewhat like polished ivory, and as feeling smooth, so as to resemble the softest satin ('Addison's Works,' New Syd. Soc., p. 102). This is observed in the form of fatty liver which accompanies emaciation. On the other hand, when the patient is the subject of obesity, the skin acquires a shining, greasy appearance, apparently due to an excessive secretion of fat by the sebaceous glands. It is said that when such persons sweat, the fluid is unable to wet the skin, and runs off in large drops.

Hebra noticed that habitual spirit drinkers have usually a soft, smooth, and clear skin with free and active sebaceous secretion. It may be that this condition is only found when the liver also is fatty, or both conditions may be the result of alcohol.

Some writers have said that diarrhœa frequently depends upon fatty

disease of the liver. It has, no doubt, been shown that both these conditions are together present in many cases, but perhaps they are both effects of one cause.

Significance.—In a case of phthisis or other wasting disease, the detection of a fatty liver does not affect the treatment, except that probably cod-liver oil and the like should no longer be given. It even influences the prognosis but little, since such cases are generally fatal, and the most that can be inferred from it is the fact, already sufficiently apparent, that the patient's nutrition is greatly damaged. On the other hand, when the same affection is part of general obesity, it is this, and not merely the state of the liver which calls for interference on the part of the physician.

Obesity in general.—This opportunity, however, is the best that offers for discussing the treatment of obesity, a condition in itself of grave import. No good insurance office will accept at ordinary rates the life of a man whose weight bears more than a certain proportion to his height. It is notorious that such persons bear even slight accidents badly, and succumb to illnesses that would be unattended with danger in healthy subjects. After death their tissues are found to be soft and flaccid, and to break down under pressure much more readily than usual; and decomposition often advances with undue rapidity. The omentum, the mesentery, and the sub-peritoneal tissue generally, are loaded with fat. The large size of the abdomen in fact presses up the diaphragm during life, and hampers the play of the lungs. The heart also is commonly covered with fat, and its substance is soft and lacerable, so that it readily tears and has been compared to wet brown paper.

The most important part of the treatment of obesity consists in the regulation of the diet. Some years ago popular attention was strongly drawn to this subject by a pamphlet published by a Mr Banting, who, in less than a year, had reduced his weight from 202 to 156 pounds. He was at that time sixty-six years of age, and his height was five feet five inches. Before he began to diet himself he had great difficulty in stooping, was compelled to go downstairs slowly backwards, and used to puff and blow with every exertion, beside being liable to fainting. The articles which he specially avoided were bread, butter, milk, sugar, beer, sweet wines, and potatoes. He took a liberal supply of animal food. He says that when he had lost his excess of fat he felt better than he had done for twenty years, and the fainting-fits altogether ceased.

Such a change of diet should not be made without supervision on the part of a medical man, for in some persons it may doubtless be attended with risks of its own. But the dangers which obesity brings with it far outweigh them. Habits of early rising and of active exercise are useful in preventing the deposition of fat, but active exercise is beyond the power of those who are already corpulent. Even hard riding does not prevent increasing obesity. Liquor potassæ and other alkaline remedies have been recommended in the treatment of this condition, but they often prove altogether useless. The tendency is sometimes hereditary and insuperable, but walking, moderately restricted diet, avoidance of beer, and the free use of water, with occasional purgation and more frequent sweating, will in most cases succeed in diminishing the patient's bulk and relieving his discomfort.

The Malarial Liver.—Under the influence of repeated attacks of ague, the liver as well as the spleen may become enlarged so as to become palpable

below the ribs. In such cases there is frequently some degree of jaundice present, and constantly the yellowish earthy pallor so characteristic of paludal cachexia (cf. vol. i, p. 345). The condition is apparently one of frequently recurring congestion leading to permanent enlargement. It may result from remittent fevers or (it is said) from residence in a malarious district, even when no febrile symptoms have followed.

THE LARDACEOUS LIVER.—Prolonged suppuration, syphilis, and perhaps other causes lead to the conversion of certain organs and tissues into a peculiar translucent material, which is known by the epithets lardaceous, waxy, albuminous, or amyloid. Its chemistry and pathology will be described in the chapter on Bright's disease. Of these organs the liver is one.

A lardaceous liver often reaches a considerable size. In one case it weighed more than eight pounds, and Wilks speaks of another in which it reached fourteen pounds.

One of the most striking characters of a lardaceous liver in the *post-mortem* room is its greatly increased density. Wilks mentions an instance in which the specific gravity was found to be 1084. It is also extremely hard. Its cut surface looks dry and bloodless, smooth, shining, and translucent. It can be cut into thin slices much more readily than in health. The lobular markings are unduly distinct. Iodine gives its characteristic reaction, as does also methyl-violet.

The microscope shows that the hepatic cells themselves are converted into lustrous shapeless masses of the lardaceous material. The cells earliest affected are those which lie at about the middle of each lobule. This position corresponds with the distribution of the ultimate branches of the hepatic artery in which the degeneration begins. After a time the change extends inwards to the centre of the lobule, and last of all outwards to its periphery.

Dr Duckworth has recorded cases in which the lardaceous liver became reduced in size. In one case, after reaching to the iliac fossa and nearly to the pubes, it became in fifteen months about half the size it was, and at the autopsy weighed only 117 ounces ('St Barth. Hosp. Rep.,' vol. x, p. 57, 1874).

Symptoms of lardaceous disease of the liver are almost absent. As with fatty and leuchæmic enlargement, there is neither pain nor ascites nor icterus.

Its diagnosis depends on its physical signs. It often reaches down to the level of the umbilicus. During life the edge can generally be felt very distinctly, and is more readily discovered than that of a fatty liver. It is firm and resisting. The surface of the organ is perfectly smooth, unless perihepatitis or some other disease be also present. But such a combination is sufficiently common to need to be borne in mind, particularly (as we shall see presently) in reference to the diagnosis of hydatids of the liver.

But in diagnosing a lardaceous liver we are practically guided by our knowledge of its ætiology. We expect to find it in a case of disease of the bones or of the lungs accompanied with long-continued suppuration, and particularly in syphilitic cases. And we are confirmed in our conclusion by finding evidence of the same degeneration affecting the spleen, the kidneys, or the intestines.

HYDATIDS OF THE LIVER.—In all the forms of painless enlargement of the liver that have hitherto been mentioned, the organ is *uniformly* increased

in size. In this respect they differ altogether from a hydatid tumour, which is a rounded elastic swelling, occupying only part of the liver It may reach a considerable size before its presence is detected, and it often causes not the slightest inconvenience or discomfort to the patient.

Natural history and development.—It has been stated in a former chapter (p. 436) that every tapeworm, in the course of its development, passes through a very remarkable phase, in which it forms a bladder, embedded in the substance of one of the higher animals, and filled with a transparent fluid. Now, a hydatid of the liver really represents this stage in the development of a little tapeworm, which in its mature form inhabits the intestine of the dog, and is called *Tænia echinococcus.* When fully grown this measures at most four millimetres or about the sixth of an inch in length, and consists of only three or four segments, of which the last alone contains developed sexual organs. It is very common in London dogs and is often present in large numbers in their intestines. Its ova are discharged with the fæces of the host, and in some way obtain an entrance into the human stomach, being probably carried there either in drinking-water or upon the leaves and stems of raw vegetables. When an ovum of the echinococcus tapeworm has thus found its way into the alimentary canal of a man who is henceforth to be its host, it at once enters upon a wonderful career of development. The first change appears to be due to the action of the gastric juice, which dissolves its shell, and liberates the embryo, larva, or *scolex*, as it is called. This possesses six little hooks, arranged in two rows; and being capable of active movement, it probably at once sets to work and bores its way through the walls of the stomach—or of the small intestine, if it has passed through the pylorus with the chyme. Its further course is somewhat uncertain. Sometimes it appears to reach the serous surface, and may either remain and develop itself within the peritoneal cavity, or perhaps strike across into one of the solid viscera. If, however, this were its usual course, the hydatid tumour which is the result ought to be met with in the other abdominal viscera almost as frequently as in the liver. But the fact is that hydatids are very much more commonly found in the liver than anywhere else. It can hardly be doubted that the cause of this is that the embryo, in piercing the wall of the stomach or intestine, generally gets into one of the rootlets of the portal vein, and is at once washed away by the stream of blood, and carried onwards through the main trunk until at last it is arrested in one of the capillary branches of the portal vein within the liver.

Having thus reached the interior of the liver, the scolex proceeds to develop itself into a hydatid. Perhaps it first bores its way out of the blood-vessel and may travel some distance through the hepatic tissue, as the embryos of other tæniæ are known to do in the organs which they infest. Very soon, however, its movements are arrested. It loses its hooks, grows larger, and from being solid, becomes converted into a vesicle, containing a transparent fluid. It also gives rise to certain changes in the tissues round it, apparently as a result of the irritation caused by its presence. It becomes surrounded by a layer of granular matter, and before long by a distinct membranous investment, consisting of connective tissue, and abundantly supplied with vessels. Henceforth the hydatid is always enclosed in this investment, which grows as it grows, and which may be properly termed its capsule. Thus the capsule of a hydatid is really a structure formed from

the human tissues, with which the hydatid or echinococcus itself lies in contact but possesses no organic connection.

Up to this point the development of the echinococcus is precisely analogous to that of the cystic stage of any other tænia, for instance, of a cysticercus. But the further steps are very different in the two creatures. The cysticercus, in order to complete its development, would only have to form a single "head" or "scolex" in its anterior. This head would grow as a kind of bud or protrusion from one part of the interior of the cysticercus, and would gradually become provided with its two rows of hooklets and its suckers. Now, in most specimens of the echinococcus, instead of a single bud or protrusion, a number of them form at different times from the interior of the animal. And each of these buds does not develop into a head, but itself forms a cystic body which for a time remains attached to the spot where it arose by a pedicle, but soon becomes detached. It is then called a "daughter-cyst," while the original hydatid that encloses it is termed the "mother-cyst." Each daughter-cyst, again, may develop one or more "granddaughter" cysts in its interior. In this way the echinococcus becomes filled with a number of smaller vesicles of various sizes, which may amount to thousands. If any of them contain other smaller vesicles they are sometimes described as "pillbox hydatids," since their arrangement may be said to resemble that of a "nest" of pill-boxes. Sooner or later, the little buds or protrusions cease to form detached vesicles, and develop into very small thin membranous sacs, the pedicles of which are persistent, and which are called "brood capsules," because they give origin to a variable number of "scolices" or "heads," each of which has its row of hooklets and its four suckers, and is capable, under favourable circumstances, of growing into a tænia. These scolices or heads have in England been commonly designated echinococci; and the use of this term accords both with its derivation (ἐχῖνος, hedgehog, κόκκος, grain or berry) and with the intention of Rudolphi, who invented it. But at the present day, the name of echinococcus is commonly applied to the whole animal, with its daughter-cysts, brood capsules, and scolices. It will be observed that a difference between a cysticercus and an echinococcus is that, whereas the former gives rise only to one scolex, and can ultimately form only a single tapeworm, the latter may develop thousands of both (cf. p. 440).

Hydatids do not, however, necessarily pass through all the developmental changes above described. Sometimes they fail altogether to produce scolices, and even daughter-cysts. They are then said to be "sterile hydatids" or "acephalocysts." The term acephalocyst was invented by Laennec, because he believed that scolices were never produced by the hydatid which infests the human subject, whereas he was aware of their presence in hydatids from the lower animals. It is said that Bremser, in 1821, first discovered scolices in hydatids taken from the human body. Bright was one of the earliest English physicians to observe them; he gave a drawing of them in the 'Guy's Hospital Reports' for 1837. However, even after it was universally recognised that the hydatids of man contain scolices, they still were called acephalocysts. The term has never fallen entirely into disuse, and may still be applied to those hydatids which are really sterile. This is said to be more frequent in the case of hydatids infesting the brain than in those of any other organ.

The account given does not, however, exhaust the list of developmental changes of which a hydatid is capable. Sometimes, but in the human

subject very rarely, instead of budding internally to form daughter vesicles, it throws off protrusions externally. In this way the liver may become riddled with hydatids, not contained in any mother-cyst, but penetrating its tissue in all directions, and even invading the neighbouring organs. A very remarkable case of this kind occurred a few years ago in Guy's Hospital, under the care of Dr Rees. A boy was admitted with what seemed to be effusion of fluid into the right pleura, and enlargement of the liver. But when the chest was punctured with a trocar, hydatids escaped. Ultimately he died, and it was found that the liver, diaphragm, and right lung were full of hydatids, which were budding externally in all directions. It has been supposed by some writers that when two or more echinococci are found in different parts of the liver, or in different organs of the body, the one has been derived from the other by the process of gemmation. But it is more probable that in such cases each hydatid was separately developed from a single embryo, the patient having swallowed more than one of the ova of the echinococcus tapeworm.

Multilocular hydatid.—Lastly, brief reference must be made to a very remarkable form of hydatid—developed by this process of external gemmation—which is styled "multilocular" by continental pathologists. It forms a solid globular mass in the liver, as large as a fist or a child's head. Its periphery is well defined, and it can be shelled out of the tissue in which it lies. But on section it is found to be divided by trabeculæ into a number of small cavities of irregular form, each containing a mass of gelatinous material which is made up of hydatid membranes pressed closely together, and small cysts containing scolices. The individual cysts are never larger than peas, and are often as small as millet-seeds. Virchow has suggested that in this form of the affection the parasite occupies the interior of the lymphatic vessels. A multilocular hydatid tumour is always found to have undergone softening in its centre, and to be broken down into a suppurating cavity. Suppurative peritonitis and jaundice are also frequently present at the time of the patient's death. This form of hydatid tumour seems hitherto not to have been observed in England.*

Anatomy.—We must now return to consider the characters of an ordinary hydatid tumour or echinococcus of the liver. This forms a more or less globular mass, varying in size from that of a walnut to that of a cocoa-nut, or even larger; the largest on record is said to be one weighing thirty pounds, which was observed by Luschka. If it is subjected to no pressure in its growth its form is probably always spherical, but if it meets with more resistance on one side than on another, it may be flattened or egg-shaped, or it may even asssume an hour-glass form. The extent to which a hydatid is embedded in the liver varies greatly in different cases, and entails very different clinical features. In some cases it would seem that the six-hooked embryo originally lay just beneath the serous covering of the liver; and the hydatid may then form a globular mass depending from its surface, and having so little obvious connection with it, that one may find great difficulty in determining that the liver is really the seat of the tumour. In other cases a great part of the sphere formed by the hydatid may lie within the hepatic substance; and its presence may be indicated only by a

* Frerichs suggested that a specimen in the museum of Guy's Hospital, which is labelled "colloid cancer of the liver," is perhaps really a multilocular hydatid. But some years ago I carefully examined this specimen, and could not discover any trace of a parasite in it.—C. H. F.

rounded projection from one face of the organ, the curve of which is often little noticeable in proportion as the cyst is large. Sometimes, again, a hydatid may reach both surfaces of the liver at once; and the original anterior edge of the liver may then be distinctly made out as a narrow ridge, passing obliquely downwards and to the right across the rounded tumour, which occupies the epigastric and hypochondriac regions. Lastly, a hydatid may be embedded entirely in the back part of the liver, or reach only that portion of its surface which is in contact with the diaphragm, and covered by the ribs.

Symptoms.—The sensations imparted to the physician by manipulation of hydatid tumours of the liver through the abdominal walls vary much in different cases. The tumour may be quite soft, and fluctuation may readily be detected in it, a wave being transmitted from one part of it to another; or it may be firm and tense, sometimes of stony hardness. In a certain proportion of cases a peculiar sensation may be elicited by percussion over it, to which Briançon first drew attention, and on which French writers generally lay great stress. It is termed the *frémissement hydatique.* The way to detect it is to place three fingers of the left hand upon the tumour, and then to tap the middle finger abruptly with the right forefinger. The other fingers of the left hand may then perceive a peculiar quivering sensation, which was formerly supposed to be due to the vibration of the daughter-cysts contained in the hydatid, but which (it is now known) may occur with cysts in which there is nothing but fluid. It is far from decisive of the cyst being due to an echinococcus.

Diagnosis.—As a rule, the diagnosis of a hydatid tumour of the liver, lying below the ribs, is not difficult. If the cyst should project far from the lower surface of the organ it may be mistaken for a distended *gall-bladder*, and sometimes there may be a doubt whether the case is not one of hydronephrosis. Distension of the gall-bladder, without jaundice—the common bile-duct being patent—is, however, exceedingly rare; and in hydronephrosis the tumour fills the lumbar region to an extent which can be very rarely the case with a hydatid; and, again, the colon is generally to be felt running over it.

A tumour which is distinctly cystic, and which at the same time is embedded in the substance of the liver, can be nothing but a hydatid. Simple *retention-cysts* containing serum are occasionally met with in the liver; but they appear never to reach such a size as would enable them to be detected during life, and are of merely pathological interest.

But when a hydatid is deeply embedded in the substance of the liver, so that it is but little raised above the surface of the organ, and yet causes it to project a long way down into the abdomen, there is often great difficulty in determining the nature of the case. In several instances the diagnosis of a hydatid tumour was given, but the enlargement proved to be due to lardaceous disease of the liver, and the circumscribed tumour to its being intersected by fibrous bands, in connection with *syphilitic gummata.* One diagnostic character of syphiloma of the liver is immobility of the organ during inspiration, due to adhesions of its surface, which are generally present. More or less pain and tenderness on pressure are also common symptoms in such cases. It once happened to the author to direct the performance of exploratory operations in two cases on the same day, and in each of them the tumour proved to be solid; it was probably in both cases a syphilitic and lardaceous liver. These patients did well; but

another patient died from the effects of chloroform while undergoing an operation for a supposed hydatid of the liver, and in this case also the tumour was found on *post-mortem* examination to be a solid mass of the nature just described.

In other cases there may be a difficulty in determining whether a tumour of the liver is a hydatid or a *cancerous growth*. The distinction must be based partly on the physical character of the tumour, partly on the presence or absence of symptoms of constitutional disturbance, particularly pain. It must, however, be mentioned that pain is not invariably absent in hydatid disease of the liver. Frerichs gives a case in which such a tumour was the seat of violent pains after every manipulation and movement, which pains ceased almost immediately upon the removal of a clear watery fluid by tapping. Moreover, the capsule of a hydatid tumour of the liver may inflame and suppurate; and it then gives rise to very great pain. The health of patients harbouring this parasite often appears to be perfect, but they may lose flesh to a certain extent; they may also suffer much inconvenience from the pressure of the tumour on neighbouring organs.

But, as has already been stated, an echinococcus growing in the liver, instead of forming a tumour that can be felt in the abdomen, may project only from the convex surface of the organ, under cover of the ribs. If it should attain a considerable size it may then cause the lower part of the chest to bulge considerably, and the edges of the costal cartilages to form a much more open curve than on the opposite side of the body. The intercostal spaces over the swelling may feel more resistant than usual, and may even project beyond the level of the ribs. At the same time the lower part of the chest yields a dull note on percussion, and the case is very likely to be mistaken for one of *chronic pleural effusion*. Such an error may, however, be always avoided by careful observation of the limits within which the dulness on percussion and the enlargement of the intercostal spaces can alone be detected. In cases of hydatid tumour below the diaphragm the area of dulness is bounded above by a curved line, which descends as it approaches the spine posteriorly. In cases of pleuritic effusion the dulness reaches to quite as high a level in the dorsal region, close to the spine, as in the neighbourhood of the right nipple. A collection of fluid in the right pleural sac might, indeed, be so confined by adhesions as to be undistinguishable from a hydatid in the liver. But this possibility need hardly be taken into account in a statement of the rule that where the physical signs indicate the presence of a collection of fluid limited to the lateral region of the base of the right chest, a hydatid tumour of the liver is present. Some time ago the author saw a case with Mr Durham, which admirably illustrates this principle. The patient, a young lady, had been sent to him by a physician, who considered her to be suffering from a chronic pleuritic effusion, the result of an attack of pleurisy two or three months before. The right lower ribs, in the lateral region of the chest, were bulging, the intercostal spaces were tense, and they seemed to yield a sensation of fluctuation. There was increased dulness on percussion over the same part; but in the back the physical signs were in all respects normal. In spite of the history that was given us we agreed that the case was one of echinococcus in the liver; and the aspirator at once proved this conclusion to be correct.

For whatever doubt there may be as to the nature of a cystic tumour

of the liver, it is quickly set at rest by the chemical examination of the liquid removed from it by paracentesis with an aspirator.

The hydatid fluid possesses characters different from those of any other liquid that is met with in the chest or abdomen, although they are not unlike those which belong to the cerebro-spinal fluid. It is limpid or very slightly opalescent, its sp. gr. is 1007 to 1009, or a little higher; there is no albumen in it, so that it does not coagulate either when boiled or on the addition of nitric acid.* It does not contain common salt with alkaline and earthy phosphates in the amount found in other watery cysts. When a glass containing hydatid fluid is held up to the light one can often see floating in it delicate white bodies, so minute as to be only just visible, which rapidly settle to the bottom of the vessel. These are the clusters of scolices, either still enclosed in their brood capsules or (if the latter are ruptured) kept together by their common stalk. They form beautiful objects for the microscope, appearing as bodies of round or slightly elliptical form, with oval calcareous corpuscles scattered through their transparent substance, and each with its crown of hooklets and its four suckers usually retracted into the interior of the cystic body. Very often they are still alive, and can be seen to perform active movements. It was formerly supposed that the scolices, or echinococcus-heads, became detached from the main wall of the parent cyst in the course of their growth, and that they could swim about in the fluid. But this was a mistake; they are naturally fixed, and are only set free during the operation of paracentesis.

The discovery of a hydatid scolex, or of one of the hooklets, whether in the fluid or the solid contents of a cyst of doubtful nature, is of course conclusive, and the membranous wall of a hydatid cyst also possesses microscopical characters which are entirely different from those of any tissue of the human body. This is the case, at least, with the outer of the two coats of a hydatid, or, as it is technically termed, the "cuticula." It is made up of a number of very thin layers arranged concentrically. The smallest portion of it is seen under the microscope to be marked with delicate parallel lines, having a peculiar finely dotted appearance, which is perfectly characteristic. Another peculiarity which belongs to hydatid membranes is the fact that, when they are lacerated, the free edge always rolls itself up, so that the originally inner surface is outermost. Chemically they consist of a modification of chitin.

Events.—A hydatid tumour of the liver may give rise to a variety of consequences, some of which end in the cure of the disease. It is an interesting question whether there is any natural limit to the life of the echinococcus. To this question no certain answer can perhaps as yet be given, although Reynal is said to have met with an instance in which a tumour of the neck which had existed forty-three years—from the age of seventeen to sixty—when punctured gave issue to an immense quantity of hydatids, all apparently living; and Budd recorded the case of a lady who died at the age of seventy-three, and who was believed to have had two hydatid tumours since she was eight years old. It is certain that in persons who have died at a much earlier age than this, dead hydatids are frequently discovered. Very often their contents are deeply stained with bile. Cruveilhier long ago suggested that the entrance of bile into the capsule by ulceration of some small bile-duct has in such cases been the cause of the death of

* It is said that, like cerebro-spinal fluid, it always contains a minute proportion of grape sugar, and succinate of ammonia has also been found in it.

the parasite, and this opinion has since been very generally adopted. But it seems very doubtful, for more than once two dead hydatids have been found by the author in the same liver, one of them containing bile-stained matters, while the contents of the other were colourless; and it seems unreasonable to attribute the death of the one to the toxic action of the bile, and to leave the death of the latter unexplained. Another supposition has been that hydatids die because their external adventitious capsule is too thick and resistant to allow of their due growth. The fibrous capsule of a dead hydatid is often of cartilaginous hardness, or in great part calcified.*

When a dead echinococcus is found in the liver at an autopsy its capsule is generally found to contain a putty-like substance, made up in large part of calcareous salts, and mixed with the gelatinous relics of hydatid membranes, which often glisten with cholesterine crystals. The putty-like substance is very like that which occurs in a dried-up abscess, and in all probability is transformed pus which had formed within the capsule of the hydatid, as the cause of the death of the parasite or as its consequence.

Suppurating hydatid.—Inflammation within the capsule of an echinococcus is not an uncommon occurrence, and one which is very important, since it modifies greatly both the physical signs of the affection and its symptoms. If the tumour can be seen or felt in the abdomen this becomes painful and tender and hot, and there may after a while be redness of the skin over it. The patient's health begins to suffer; he may have repeated attacks of shivering, and symptoms of hectic may show themselves. In some cases, however, inflammation of the capsule of a hydatid may apparently take place without marked symptoms.

Rupture of the cyst.—A suppurating hydatid may ulcerate and discharge in various directions, just as a living echinococcus may also burst its capsule and pour out its contents. In the latter case suppuration of the cavity follows, and thus, unless the death of the patient should follow directly, one cannot determine whether the creature was alive or not at the time when its capsule gave way.

A hydatid cyst sometimes (but very rarely) makes its way through the abdominal parietes. It is said that this may happen even to a cyst in which the parasite is still alive, the discharge being then clear water. Another direction in which rupture may take place is into the peritoneal cavity, and this is often not a spontaneous occurrence, but the result of some injury to the abdomen, such as the patient's falling downstairs or receiving a severe kick or blow. Fatal peritonitis generally follows quickly upon an accident of this kind, but unless it is known that the patient had a hydatid tumour previously, is must necessarily be impossible during his life to say why the injury caused such severe symptoms. At least two cases, however, have been recorded in which rupture of a hydatid cyst into the peritoneal cavity seems to have taken place without the patient having been much the worse for it, although there was for some time afterwards fluctuation in the lower part of the abdomen, just as in ordinary ascites. In all probability the different

* It is asserted that the mother-cyst (although it may be full of closely-packed daughter-cysts) does not appear to be folded, as though it had ever been of larger size. It is therefore imagined that the death of the hydatid was due to its being prevented from receiving the proper amount of nutriment for the supply of its multiplying daughter-cysts. But I have, in one instance of this kind (in which I paid attention to this point), found that the mother-cyst was much folded.—C. H. F.

results of rupture in different cases depend upon the circumstance that sometimes a large number of daughter-cysts and scolices are effused into the peritoneal cavity (particularly when the tumour is widely ruptured by extreme violence), but that in other instances only the hydatid fluid is extravasated, either from the hydatid cyst being sterile, or from the aperture being small and the escape of the contents gradual.

Much more commonly, a hydatid cyst discharges its contents either into the stomach, or into some part of the intestinal canal. The daughter-cysts are then vomited or discharged per anum; and sometimes air enters the tumour, which thus becomes tympanitic on percussion. The evacuations of membranous portions of hydatids in the fæces sometimes goes on for several weeks or longer; and in the majority of cases the patient ultimately recovers.

The rupture of a hydatid cyst into one of the biliary passages has already been referred to as one of the causes of febrile jaundice (pp. 498-9).

The hydatid cysts which discharge their contents in any of the directions that have been hitherto considered are generally connected with some part of the liver which is within reach of the ordinary methods of physical examination. But often the tumour bursts not downwards, but upwards, into the chest; in these cases, its seat is almost always in the upper and posterior part of the liver, so that very frequently no positive physical signs of its presence can be discovered either before or after its rupture.

Sometimes a hydatid cyst discharges its contents through the diaphragm into the pericardial sac; sometimes into one of the hepatic veins within the liver, the daughter-cysts in the latter case passing straight into the right chambers of the heart, and plugging up the branches of the pulmonary artery. The clinical features in both cases are very similar, consisting in the occurrence of sudden death, or, at least, rapidly fatal syncope, in a person who perhaps has hitherto appeared to be in perfect health.

In rare instances, again, a hydatid tumour has been known to discharge its contents into one of the pleural cavities (generally the right), with the result of setting up a severe and rapidly fatal pleurisy. But in the great majority of cases in which the diaphragm is pierced by a hydatid, the pleura has become adherent before perforation takes place. The consequence is that the parasite makes its way into the substance of the lung, and sooner or later reaches a bronchial tube, into which it opens, so that its contents are expectorated. Cases of this kind are exceedingly interesting, and it is often a long time before their real nature can be made out.

Some years ago a boy, aged six, who was a patient of Mr Fagge, of Hythe, had suffered for about eighteen months a pain just outside the right nipple, and a constant hacking cough, for which all treatment was useless. He became exceedingly wasted, and was supposed to be sinking. One day his cough left him, and he became exceedingly prostrate, but next morning the cough returned, and he spat up a hydatid cyst and a quantity of pus. From that time he began to recover, and his cough gradually disappeared. Ultimately he died of another disease, and it was proved that the hydatid had originally come from the liver.

A young woman was once attending among the out-patients who had been expectorating hydatids for nearly a year when she first came to the hospital. Next week she had a most violent attack of coughing, which lasted three hours, and it seemed she would be choked. But at last she got rid of

a large piece of hydatid membrane, which was apparently a part or the whole of the mother-cyst; for she coughed up no more hydatids, and to a great extent regained her health.

When portions of hydatid membrane from the liver are expectorated they are generally colourless, but sometimes are deeply stained with bile. In many instances the patient regains his former state of health when all the hydatids have been voided, and the capsule has contracted so as to close the cavity. This process, however, is itself not entirely free from risk. In the case of a patient who had spat up hydatids some months before her death, and in whom hæmoptysis at last proved fatal, it was found that the blood came from a branch of the pulmonary vein; this had become obstructed and dilated into a cylindrical tube as large as a lead pencil, and had afterwards opened into the cavity that had before lodged the hydatid, although this cavity was now shrinking. Here the hydatid had originally been seated, not in the liver, but in the lung itself.

Prophylaxis.—Before considering what is the proper curative treatment of hydatid tumours, we must ask what measures can be adopted to prevent the echinococcus from entering the human body. In London hydatids are frequently found in the liver, but in Edinburgh this affection is exceedingly rare. In the United States, again, it is said to be very uncommon. On the other hand, in Australia it is far more frequent than in England, and among the inhabitants of Iceland it causes one seventh of the total mortality. In our own country, it is not likely that people will ever take precautionary measures against infection with hydatids beyond taking care when they eat raw vegetables to have them thoroughly cleansed. But it is very different in Iceland. There prophylaxis against hydatid disease is probably the most important of all sanitary questions.

Now, according to our present knowledge, it is evident that this parasite would soon become extinct in all civilised countries if its cystic form were not liable to infest some other animal than man. Dogs acquire the *Tænia echinococcus* only by eating the flesh of some creature in which the scolex is embedded, and they can very rarely have the opportunity of deriving this from the dead human body. Sheep and pigs are believed to be the chief animals besides man which harbour hydatids. Consequently, dogs should be prevented eating the offal of these animals, and should be excluded from all slaughter-houses. It has also been suggested that the floor of every kennel should be frequently scalded with boiling water so as to destroy any ova of the *Tænia echinococcus* that may have been voided with the fæces of the dog.

Curative treatment of hydatid tumours is believed at the present day to belong to surgery rather than to medicine. At one time it was thought that the internal administration of iodide of potassium, or even of chloride of sodium, would sometimes poison the intruder and lead to the disappearance of a hydatid cyst; and, on the other hand, it was supposed that the operation of paracentesis was attended with great risk to the patient. Neither of these opinions is now accepted, and most observers are agreed that an operation should at once be performed when a hydatid is detected which is above the size of a billiard ball, and which appears to be increasing in size.

The operative procedures that may be adopted for the cure of the affection are the following: first, some of the fluid may be withdrawn either by a fine trocar or by the aspirator; secondly, one or two fine needles may

be inserted into the cyst, and may either be left in it for ten minutes and then carefully taken out again, or they may be connected with a galvanic battery, the current from which is allowed to pass through the tumour for ten minutes before the needles are withdrawn.

The operation last mentioned, that of *electrolysis*, is one which has been practised several times at Guy's Hospital by the author and Mr. Durham, who published in the 'Medico-Chirurgical Transactions" for 1871 the reports of eight cases, in seven of which (if not in all of them) this plan of treatment was completely successful, being followed in most instances by the total disappearance of the tumour. Two electro-gilt needles were used, which were introduced into the cyst at a distance of one or two inches from one another. Care was taken to observe that they had entered the same cavity and could be made to touch one another. They were then attached to wires connected with the negative pole of a galvanic battery of ten cells, while the positive pole was made to terminate in a moistened sponge placed over the surface of the tumour at a little distance from the points of entry of the needles. The current was allowed to pass for about ten minutes, after which the needles were withdrawn.

When this operation was first tried, it was supposed that its success would be attributable to the decomposition of the saline liquid contained in the cyst, by which the hydatid would be killed. But further experience suggested a doubt as to the correctness of this opinion. In several of our cases fluctuation could be detected in the lower part of the abdominal cavity a few hours after electrolysis. In two instances a rash showed itself on the first or second day afterwards, resembling urticaria or scarlatina. Now, Dr McGillivray has recorded a case in which urticaria followed the discharge of fluid from a hydatid cyst into the peritoneal sac. It therefore seemed to us probable that the operation of electrolysis led to a similar escape of some of the fluid, and that this might really be the cause of the death of the parasite.

In fact electrolysis is unnecessary, for the same result follows the introduction of the needles without any galvanic current (*acupuncture*). In two cases at Guy's Hospital this plan was adopted with successful results.

The other operation, that of *paracentesis*, has been especially advocated by Murchison, and is now usually performed with the aspirator of Dieulafoy. If a large part of the hydatid cyst be outside the substance of the liver, the aspirator may with advantage be employed to remove its contents, which this instrument can rapidly extract. But if the cyst be almost entirely buried within the solid tissue of the liver the aspirator is likely to do harm. The withdrawal of a very small part of the fluid contained in a hydatid, by means of a very fine trocar, is often sufficient to destroy the parasite and to set up a process which will ultimately lead to the disappearance of the tumour. To do more than this is generally useless, and to exert forcible suction by means of an aspirator upon a cyst surrounded on all sides by the resistant tissue of a solid organ must involve some risk of setting up inflammation.

For it is of importance to observe that the uncertainty at present existing with regard to the choice of operations for hydatid tumours depends hardly at all upon differences in the risk attaching to these operations in themselves: *all* of them are now known to be capable of curing the complaint in most cases with safety to the patient. The point that remains doubtful is whether one operation is more likely than another to be

followed by suppuration of the cyst, and the necessity for subsequent paracentesis.

It very often happens that some weeks or even months after tapping, the tumour is found to have regained its former size, or even to exceed it. This has generally been supposed to render necessary a repetition of the paracentesis, and when the liquid collects again, it is tapped a third time, and so on. The liquid obtained by these secondary operations differs from hydatid fluid in containing more or less albumen. The augmented size of the tumour is, in fact, due, not to the continued life of the parasite, but to the effusion of serum within its capsule, and after a second or third operation this serum generally contains leucocytes in greater or less numbers, and so passes into pus. On the other hand, if the real cause of the enlargement of a hydatid tumour after an operation be recognised, and if further interference be carefully abstained from, the tumour sooner or later begins to decrease in size again, and after a time disappears. The rule therefore would seem to be that no second operation upon a hydatid cyst should be performed within twelve months, unless there be reason to fear that suppuration has been set up within the capsule.

With regard to the question whether there is less danger of the occurrence of suppuration after acupuncture, or after paracentesis with a fine trocar, we are not yet in a position to speak with certainty. In a series of ten cases treated by the former method (with or without electrolysis, which we have seen to be immaterial) there was not a single instance in which suppuration occurred, or in which any symptoms arose beyond those of very transient febrile disturbance. One patient was up and about the ward on the fourth day and was discharged from the hospital on the tenth day. Such immunity from severe inflammation of the cyst is certainly rare. Dr Duffin has remarked that children have formed the majority of the patients on whom the operation of electrolysis has hitherto been performed, and that this may perhaps be the reason why that operation has not hitherto been followed by suppuration; but hydatid cysts may suppurate, under other conditions, even in children.

Another point that must not be forgotten, in considering the relative results of acupuncture and paracentesis, is the possibility that the cyst may have undergone suppuration, without any marked symptoms, before the operation. If this ever occurs, it would certainly make acupuncture dangerous; but we may probably exclude it when the patient has been in perfect health, and has not experienced the slightest pain nor uneasiness.

Even when a considerable time has elapsed after the apparent cure of a hydatid tumour there still remains a liability to the occurrence of suppuration within it. Dr Wilks has seen more than one instance in which this has happened after a long interval. The retrograde changes which lead to the ultimate disappearance of such tumours are no doubt very slow in their progress. A year or two ago, a patient of Dr Moxon's lay for several weeks in the clinical ward in bed, on account of a pain in the hepatic region which had come on some time after the performance of an operation for a hydatid tumour. The tumour itself could no longer be detected, but the pain was intense and resisted all the measures that we employed for its removal. Doubtless, some of the sensitive structures of the abdomen were subjected to traction by the shrinking of the capsule of the cyst.

Beside the operations that have been hitherto alluded to, several others have been proposed and advocated; among them are the injection of oxgall,

iodine, or oil of male fern into the hydatid cyst; the introduction of a large trocar and the formation of a fistulous opening, or the penetration of the tissues external to the tumour by gradual stages, so as to allow of the formation of adhesions between the capsule of the cyst and the parietal peritoneum. But when these various plans were proposed, it was not known how safely and successfully hydatid tumours may be treated by the simpler methods above described.

When, however, suppuration has once occurred within the capsule of a hydatid tumour, or perhaps in any case if the tumour is very large, it should at once be opened with due antiseptic precautions, the cyst having been first fixed by sutures to the abdominal wall so as to obviate the risk of extravasation. A large trocar should be used, and if the tumour contains secondary cysts, as many of them as possible should be removed. A drainage-tube is then inserted, and the cavity is washed out every day. The obliteration of the cavity in such a case is necessarily a very slow process, and attended with much risk. But with proper management and good nursing, cases of this kind usually end favourably. An enormous multiple hydatid cyst in a patient of the editor was lately opened in this way by Mr Jacobson with complete success.*

Hydatids in other organs.—Although this is not strictly the proper place for it, it will be convenient to say here all that need be said of the occurrence of the echinococcus in other parts of the body than the liver. The fact is that hydatids are comparatively seldom found anywhere else, and that they still more rarely have any clinical interest attached to them. Leuckart appears to think that for every three cases of echinococcus of the liver there may perhaps be one in some other organ, but even this estimate is probably above the mark. Davaine collected very carefully all published cases of hydatids occurring in various parts of the body, and he found that among 200 cases of this kind (those of the liver being excluded) there were about 40 cases of hydatids of the lungs, about 30 of the muscles and subcutaneous connective tissue, 30 of the kidneys, 26 of the pelvis, 20 of the nervous centres, 17 of the bones, and 10 of the heart.

When an echinococcus develops itself in the interior of one of the *lungs*, it is found as a rule in the base of the right lung, a circumstance which can be explained only on the supposition that the six-hooked embryo penetrated into the organ from the liver by its own movements. Thus in either case pulmonary hydatids are almost always migrated parasites of the liver. Clinically, hydatid disease of the lung is scarcely likely to be suspected until one or more of the daughter-cysts have been expectorated. Much more often, however, a primary hepatic cyst opens into the lung as above explained (p. 553).

Hydatids of the *brain* have occasionally been met with in the *post-mortem* room; its symptoms are undistinguishable from those of other cerebral tumours. It may be remarked that the capsule of the parasite is exceedingly thin and delicate when the brain is the organ infested by it.

Echinococci are sometimes found in the *heart;* they may give rise, according to their exact seat, to very varied symptoms, but sometimes to none at all.

The *spleen* is but seldom the seat of a hydatid tumour. In one case of the kind there was a large tumour in the left hypochondrium, but until an

* On the treatment of hydatid tumours in the liver, see an important discussion at the Clinical Society in December, 1887 ('Trans.,' vol. xxi).

autopsy had been made, it remained uncertain in what organ the parasite was seated. Hydatids of the kidney will be mentioned again.

In some of the great cavities, such as the *pleura* and the *pericardium*, the echinococcus may grow to a considerable size, without any tendency to the formation of a capsule round it,—the natural serous membrane seeming to take its place. This is said not to be the case with the *peritoneum*, in which hydatids are described as always having a proper capsule. However this may be, hydatid disease of the peritoneum has considerable clinical interest. One form of it is apt to be mistaken, even by the most skilful surgeon, for cystic disease of the ovaries; and an attempt has several times been made to remove such a tumour by ovariotomy. In another form of this affection a number of distinct globular tumours are found in different parts of the abdominal cavity, some of them having been developed in the omentum, and others in the interspaces between the different viscera. The physical characters of the tumours in such cases ought to render them by no means difficult of diagnosis.

Lastly, there is a very remarkable variety of hydatid tumour, which develops itself in the *pelvis*, between the bladder and the rectum, or (in the female) behind the uterus. In these cases the six-hooked embryo doubtless got into the serous cavity when it had penetrated the walls of the stomach, and fell by its own weight into the most depending part of the peritoneal sac. The result is the formation of a tumour which may assume an oval form exactly like that of a distended bladder, and may occupy precisely the same situation. In a case of this kind which occurred in our wards it was supposed that the bladder was full, but of course the catheter failed to give relief. The patient died, but it was not until the case had been cleared up by an autopsy that its real nature was even suspected. Bright relates a similar instance, and several others have been placed on record by different observers; so that it may be laid down as a rule that whenever a fluid tumour is felt in this position, which cannot be reduced in size by the introduction of a catheter into the bladder, one should think of the possibility that an echinococcus may be present. The hepatic region should be carefully examined in such cases, and indeed whenever there is reason to suspect the existence of hydatids in any other part of the body. For it happens very commonly indeed that the liver is at the same time infested with ecchinococci.

A parasite belonging to the class Arachnida and to the same order of mites to which the *Acarus* and the *Demodex* appertain is occasionally found in the liver, but it is always dead and encysted. It is called *Pentastoma tænioides* and has no clinical significance. We have frequently noted it at Guy's Hospital.

The rare presence of the liver fluke (a Trematode worm) in man has been already mentioned in the chapter on Entozoa (p. 462).

AFFECTIONS OF THE URINARY ORGANS

FUNCTIONAL DISORDERS

Arrangement adopted—Polyuria and oliguria; high and low specific gravity—Diabetes insipidus—"Renal inadequacy"—Reaction: alkaline urine, and phosphatic deposits—Urobilin and Indican—Uric acid and urates—Oxalates—Cystine—Calcic sulphate—Ammoniacal urine—Bacilluria—Hæmoglobinuria.

In dealing with the affections of the urinary organs, we will begin with disorders of function, and afterwards pass on to the inflammatory diseases which are comprised under the title Morbus Brightii, of which a common feature is *albuminuria:* then will follow the various morbid processes that may affect the renal pelvis or the ureter in consequence of the presence of calculi, including suppuration of the kidney and pyelitis. Generally speaking, these affections are characterised by the presence of *blood*, or of *pus* in the urine.

The next chapter will treat of tubercle and new growths of the kidney, and of renal parasites.

A concluding chapter will be devoted to diabetes, a disease in which the urine contains *sugar*.

The present chapter deals with certain morbid conditions of the urine that are independent of the presence of any organic lesion of the kidneys, being mostly characterised by some alterations in its *quantity*, *density*, or *reaction*, by the formation of *precipitates* or deposits, or by the occurrence of putrefactive changes.

There are many morbid conditions of the urine, such as the absence of chlorides in pneumonia (p. 138), and the presence of bile-pigment in jaundice (p. 494), or of leucin and tyrosin in acute yellow atrophy of the liver (p. 534), which require no account in this place, because they were fully described when those diseases were being discussed. Our present task is to give an account of the changes in the urine that do not belong to any other well-marked pathological process, but constitute, from a clinical point of view, so many independent affections.

Among these there is theoretically to be found a line of distinction which it would be very desirable to draw broadly and clearly. In some the urine contains an abnormal material, or a normal material in undue quantity, as the result of a morbid process in the system generally or in organs remote from the kidneys. In others the formation of a deposit is due merely to a relative excess or deficiency of acidity in the urine, or to the occurrence of fermentative changes in it after it has been secreted. Unfortunately, however, one cannot carry out this division completely; for in many cases, as, for example, with regard to oxalate of lime and uric acid, there is the greatest difficulty in determining whether the urine does or does not really contain too much

of the materials which are thrown down from it as deposits. But the order adopted will as far as possible bring out first those affections in which the urine is faulty from the time of its formation, and afterwards those in which its morbid condition is traceable to changes subsequent to its passage into the pelvis of the kidney.

Changes in the quantity and specific gravity of the urine.—Between the amount and the density there is an inverse proportion. In health, when the urine is very abundant, it is always pale and watery; when it is scanty, it is dark and of high specific gravity. Among diseases likewise, diabetes is the only case in which pale and abundant urine is also of great density: on the other hand, a diminished flow of urine is not likely to be accompanied with a low specific gravity of the secretion except when the kidneys have undergone extensive destruction in advanced Bright's disease.

In normal circumstances, the daily amount of urine ranges in different persons and under different circumstances from forty to fifty ounces or more; it is liable to great variation from day to day, and probably there are some perfectly healthy people who habitually void either considerably larger or considerably smaller quantities. The specific gravity of the collected twenty-four hours' urine generally varies between 1018 and 1022.

Physiology teaches that the quantity and the density of the urine depend mainly upon the activity of the blood-current in the renal glomeruli. According to Ludwig the determining factor is the pressure of the blood within the vessels of the tufts; according to Heidenhain it is rather the rapidity of its passage through them. The chief point in favour of the latter theory is the fact that when in experiments on animals the outflow of blood through the renal veins is checked, the urine becomes scanty and of high specific gravity; but Cohnheim shows that this fact is perhaps not so completely inconsistent with Ludwig's view as might at first sight appear. Clinically, the distinction appears to have but little significance. The only local cause of obstruction of the renal veins is thrombosis of these veins or of the inferior cava above their mouths. Now, thrombosis of the renal veins is not very infrequent as a complication of lardaceous and other forms of Bright's disease, and Dr Moxon has recorded in the 'Guy's Hospital Reports' for 1869 two cases in which it was associated with injuries to the lumbar spine. But in the former class of cases the existence of lesions in the renal cortex makes it impossible to determine the effect of the thrombosis upon the characters of the renal secretion; moreover, as the obstruction is probably developed very slowly and gradually, collateral channels have time to enlarge and can carry on the circulation. In both of Dr Moxon's cases the arteries were plugged as well as the veins, so that the urinary secretion was of course entirely suppressed. Bartels has related in 'Ziemssen's Handbuch,' a case in which the inferior vena cava was closed by thrombus from the point where it passes along the groove in the back of the liver downwards; in that instance, however, the urine, which contained blood and albumen, was secreted in fair quantity, and was of sp. gr. 1011 to 1013.

When the systemic venous circulation generally is obstructed, as in cases of heart disease and of pulmonary emphysema, the urine is almost constantly found to be scanty and of high density. This accords equally well with Ludwig's and with Heidenhain's theory, since the pressure in the arteries is in such circumstances lowered as the result of enfeeblement of

the left ventricle. When, under the influence of digitalis, the blood-pressure in the glomeruli can be brought up to a sufficient point, it is surprising how greatly the secretion of the kidneys becomes increased.

Sir William Roberts relates the case of a man, aged fifty, who for several days passed only about thirty ounces of urine daily, of specific gravity 1028 or 1029, containing an amount of urea which was at least a quarter above the average for his body weight, this being only 8 st. 6 lbs. At one time there was a small quantity of sugar in the urine. Prout thought that he recognised a disease, which has since been termed *azoturia*, the fundamental symptom of which was an increase in the excretion of urea. In cases which have been placed under this category the flow of urine has been generally excessive. But it is very doubtful whether they have been rightly interpreted. Urea is itself a powerful diuretic.

A state of urine in which the quantity is augmented, and the density reduced, is of frequent occurrence. We shall find it to be a very important and often an early symptom of certain forms of Bright's disease, being then dependent upon the abnormally high arterial tension that characterises that disease. But it is also seen as an independent condition.

Diabetes Insipidus.*—In this disease the patient passes enormous quantities of urine, exceeding even those that are voided in saccharine diabetes itself, for which it is pretty sure to be mistaken until chemical analysis shows that no sugar is present. The specific gravity constitutes another marked distinction between the two affections, for in diabetes insipidus it is often scarcely above that of water, and seldom reaches higher than from 1003 to 1007. The daily secretion of urine may range from fifteen to thirty and even forty pints. It is clear, and almost, if not quite, colourless; it has a faintly acid reaction, but early undergoes the ammoniacal fermentation. It of course contains a very small proportion of solid matters, but nevertheless the total daily amount of urea excreted appears to be rather excessive than diminished.

One abnormal constituent, *inosite*, or muscle-sugar ($C_6H_{12}O_6$), has been sometimes detected in the urine;† but there is no reason to suppose that its presence is an essential feature of diabetes insipidus. In fact, it is often absent; and on the other hand it sometimes occurs in saccharine diabetes, in Bright's disease, and in the healthy kidneys. As inosite is always to be found in small quantity in the muscles (as also in the lungs, liver, spleen, and other organs), the supposition has been hazarded that its excretion in the urine may be the result of the excessive transudation of water through the tissues; and Strauss is said to have discovered it in the urine of three healthy persons, who, for the purpose of experiment, had drunk a large quantity of water.

* *Synonyms.*—Pseudo-diabetes—Polyuria—Diuresis. The Greek word *διαβήτης* (*i. e.* a siphon, so called from its resemblance to a pair of compasses, from *διαβαίνω*, to straddle) was applied by Aretæus and Galen to the condition in which whatever a man drinks runs through him as through a siphon. Most of such cases were probably true saccharine diabetes, others were chronic Bright's disease. The discovery that in many of these cases sugar is present was made by the celebrated anatomist Thomas Willis (1670), and from that time *diabetes insipidus* has been distinguished from *diabetes mellitus*.

† Scherer's test for this substance in solution, consists in treating with nitric acid, evaporating cautiously to dryness, moistening the residue with ammonia and with solution of chloride of calcium, and then evaporating again; whereupon a rose-red colour makes its appearance. Inosite occurs in many unripe fruits (as French beans, whence it has been called *phaseolo-* or *phaséo-mannite*). It is crystalline, soluble in water and sweet like glycose, but does not reduce copper or rotate the polarized ray.

Although the absence of sugar constitutes the main distinction between non-saccharine and true diabetes, minute quantities of glycose have been present in the urine in some exceptional cases of what in other respects has appeared to be diabetes insipidus.

Symptoms.—A tormenting thirst is one of the main symptoms. At one time it was imagined that this might really constitute the essential feature of the disease, which should therefore be properly regarded as a "polydipsia," rather than a "polyuria." But experience has shown that the patients always pass more urine than healthy persons who drink the same quantities of fluid; moreover when in a case of diabetes insipidus the amount of drink is restricted, the urine does not fall in the same proportion, and the tissues become dry. So severe is the thirst that the patients who have not been allowed to satisfy it have been known to drink up their own urine, as in a case related by Trousseau.

In some instances the general health remains wonderfully good. Sir Wm. Roberts cites the case of a farm labourer, aged fifty-one, who had been affected for twenty-four years, drinking from thirty-two to thirty-six pints of water daily, and voiding urine in proportion, and who yet remained able to do all kinds of hard work, such as threshing and mowing. And another case is recorded of a woman, who bore eleven children while suffering from the disease. It is especially noted that the farm labourer's skin was moist, and that he perspired freely when at work. As a rule, the skin is obviously dry and harsh, and Roberts himself had under his care a boy, who, although rosy and plump, had a dry skin and tongue.

Most patients, however, complain that their rest at night is disturbed by the frequent desire to micturate. Other symptoms are, according to Roberts, a painful dryness and heat of the mouth and fauces, pains in the loins and in the epigastrium, an indifferent or sometimes a voracious appetite, enfeeblement of bodily strength and of mental vigour, irritability of temper, and abolition of the sexual functions. Senator, in 'Ziemssen's Handbuch,' states that the temperature of the body is slightly lowered, possibly from the large quantity of water that is swallowed having a cooling effect. Enforced abstinence from drink aggravates most of the symptoms; the skin then becomes hot, there is an intolerable sense of sinking, or intense pain at the pit of the stomach, and at last the intellect becomes impaired. Sir Thomas Watson relates the case of a boy aged eleven, who was limited during twenty-four hours to drinking a pint and a half of fluid, and who nevertheless passed ten and a half pints of urine. That he absorbed water from the air seemed to be clear from the result of weighing him at short intervals.

Diagnosis.—Diabetes insipidus is of infrequent occurrence as compared with most other diseases. In London hospital practice it is decidedly rare; and with regard to all published statistics there is a doubt whether they are not to a greater or less extent vitiated by the inclusion of cases in which some form of Bright's disease would have been found present if an autopsy had been made. Roberts, indeed, avowedly places in his collection of seventy-seven cases three which ended fatally, although in each of them the kidneys were affected with a marked degree of atrophy in association with hydronephrosis, *i. e.* "consecutive Bright's disease." Now, it is clearly important to distinguish from diabetes insipidus all cases in which the polyuria, however excessive, is a mere symptom of an organic lesion of the kidneys. But the question remains whether, in any of the cases in question

(two of which came under the observation of Dr Eade, of Norwich), the hydronephrosis could possibly itself have been a secondary result of the frequent micturition, just as we shall find hypertrophy of the bladder to be a not infrequent consequence of saccharine diabetes. In one of Roberts's cases the patient, a man aged sixty-two, is said to have suffered for twenty years from excessive thirst and diuresis.

The clinical diagnosis of diabetes insipidus must therefore always be regarded as somewhat uncertain in persons advanced in years, on account of the insidious way in which renal cirrhosis comes on, often without producing any albuminuria. Probably, however, mistakes might be prevented by careful observation of the state of the arterial tension, which in diabetes insipidus appears to be lowered rather than excessive. Even in young subjects one must not overlook the possibility of the presence of hydronephrosis from calculous disease in childhood. Polyuria from excessive drinking, either of water or of alcoholic liquors, must also be excluded.

Ætiology.—Among Roberts's cases there were seven in which diabetes insipidus was said to have begun in infancy (or from the time of birth), fifteen in which it began between the ages of five and ten years, thirteen between ten and twenty years, sixteen between twenty and thirty years, fifteen between thirty and fifty, and four between fifty and seventy. Males bore to females the proportion of two and a half to one.

In a few instances there was a well-marked history of the occurrence of the disease in several members of the same family; the most striking example of this seems to be one recorded by Lacombe, in which a mother, her three sons, her daughter, her brother and his children, were affected in turn. According to Trousseau, diabetes insipidus is not uncommonly seen in persons whose parents had suffered from diabetes mellitus, or from albuminuria. In some cases it has been attributed to trivial circumstances, such as exposure to cold or heat, drinking cold fluids when heated, intemperance, muscular efforts, mental emotions. But the most important of its exciting causes are blows or falls upon the head, and certain organic lesions of the brain. As regards the traumatic cases, Roberts remarks that in some of them the polyuria has set in with its maximum intensity on the very day of the accident, but in others not until after the first loss of consciousness had passed off, or a few days later, or even in one case not until the time of subsidence of severe nervous symptoms at the expiration of six months. In some of the cases associated with cerebral lesions there have been scrofulous or other tumours, occupying various positions,* but in one instance projecting into the fourth ventricle from its floor.

Such observations possess special interest in consequence of the fact that an affection like diabetes insipidus can be produced experimentally in animals by injuries to certain parts of the nervous apparatus. Bernard first showed that this effect followed puncture of the floor of the fourth ventricle at a point a little above the glycosuric centre. There is also experimental evidence as to the production of an excessive flow of urine by irritation of the cervical sympathetic and by lesions of the spinal cord. The probability seems to be that the immediate cause of the affection is a dilatation of the renal arteries, from defect of the controlling action of their vaso-motor nerves. So far as is yet known, there are no nerve-fibres influencing the renal function which are secretory (in Heidenhain's sense, *i. e.* trophic, affecting the secreting epithelium). It is worthy of mention

* See Dr Alexander Hughes Bennett's case, 'Brit. Med. Journ.,' Feb. 24th, 1883.

that, in a case observed by Külz, diabetes insipidus was accompanied by spontaneous persistent ptyalism (the patient spitting from twelve to eighteen ounces of saliva daily); for this is another symptom that has been produced in animals by puncture of the floor of the fourth ventricle.

Prognosis and treatment.—The course of diabetes insipidus varies widely in different cases. Roberts cites one instance in which it set in with absolute suddenness; the patient, a woman, aged thirty-four, went to her work one morning at 6 a.m. in her usual health; two hours later she was seized with intense thirst and diuresis, which continued from that time.*

In those cases which follow injuries to the head, the affection commonly subsides in the course of a few weeks or months, but cases are on record in which it had been of six or seven years' duration. And of the non-traumatic cases beginning from infancy, some are stated to have run on for fifty years or more. When there is a cerebral tumour this is of course almost sure to prove fatal in a comparatively short time. Otherwise, diabetes insipidus does not in itself appear to have much tendency to destroy life; for the patient generally succumbs to an intercurrent malady, such as phthisis or pneumonia.

It is a remarkable fact that the occurrence of some inflammatory or febrile disease has in several cases led to the temporary, or even permanent, subsidence of diabetes insipidus. Thus one patient who had suffered from it for eighteen years recovered completely after an attack of acute rheumatism, and another after an attack of pleurisy, treated by a blister which suppurated for thirty-five days. Roberts suggests that it might be worth while in future cases to try the effect of a large blister applied to the back of the neck, or to the epigastrium. Among medicines the most useful seems to be valerian, which was prescribed by Trousseau in enormous doses, two and a half drachms of the extract daily, or even more. To one patient he gave nearly an ounce, and in the course of four months recovery took place. Roberts relieved a boy under his care with the valerianate of zinc, increasing the dose until it reached twenty grains a day. Dr Dickinson has found benefit result from codeia. Ergot is another remedy that has been used with more or less success. Galvanism (the constant current) has been recommended by some German observers. One pole may be applied to the loin on one side near the spine, and the other to the corresponding hypochondrium for five minutes; and then they may be transferred to the opposite side of the body in the same manner; or, as Külz advises, the positive pole may be placed upon the nape of the neck, and the negative pole, first to the loins for four minutes, and then to the epigastrium for the same period of time.

Renal Inadequacy.—Under this name Sir Andrew Clark has described ('Brit. Med. Journ.,' i, 1883) a class of cases of which the main feature is that the kidneys appear to be unable to excrete more than the normal daily quantity of urine (from forty to fifty ounces); while even this is of low specific gravity (1002 or 1003 to 1008), and is deficient in urea (not containing more than 2 per cent.), though the amount of uric acid may be

* Some years ago I met with a remarkable case in a man living at Dulwich, who had several distinct but very short attacks of what appeared to be diabetes insipidus. I have unfortunately preserved no notes, but my impression is that each attack lasted a day or two, and I remember that he passed enormous quantities of urine, and became for the time exceedingly prostrate and exhausted. I think that this recurred at intervals of some weeks.—C. H. F.

atural. Even if these patients drink freely of water, they do not pass a larger quantity of urine; and a liberal diet with a full allowance of wine is obviously injurious to them. The urine in such cases is devoid of albumen, and contains no casts. Although Sir Andrew Clark admits it to be possible that the kidneys are on the way towards chronic Bright's disease, he says that when he has had an opportunity of making an autopsy the organs have appeared to be healthy.

The patients are generally ailing, without being definitely ill. They are apt to take cold, and do not get rid of the cold easily, and are also liable to be attacked with pneumonia, pleurisy, or pericarditis without apparent reason. They recover slowly from even slight injuries, and they bear surgical operations ill, a fact which appears to have been noticed independently by Sir James Paget. They complain of *malaise*, and weakness, and unfitness for work; they sleep badly, are subject to headache, and suffer from nervousness. Sir Andrew Clark does not say anything about the state of the arterial tension in these cases. He describes patients so affected as ultimately developing a condition very like myxœdema, with pink and white faces, a dry puffy skin, a slow articulation, and a somewhat staggering gait.

The main points in the plan of treatment which he advises for them are a very sparing diet and careful management of the skin. He allows for breakfast, bread and butter and an egg; for the midday dinner, not more than half a pound of meat with vegetables, and afterwards some pudding; about six or seven o'clock, bread and butter again, with an egg.

Reaction of the Urine.—Urine has normally an acid reaction, which is commonly said to be due to the presence of the acid phosphates of soda and potass rather than of free acid. But that this view is, after all, arbitrary, appears to be shown by an experiment devised by Malz and Donati, as cited by Salkowski. It consists in dissolving in a solution of the neutral (rhombic) phosphate of soda ($Na_2 HPO_4$) the amount of hippuric acid necessary to combine with one equivalent of the sodium. If now the resulting fluid, which is strongly acid, be shaken up with ether, hippuric acid is extracted, and the fluid gradually regains the alkaline reaction that belongs (in spite of its name) to the neutral phosphate. But if, on the other hand, the fluid be evaporated, and the residue extracted with ether, no hippuric acid comes away. These apparently contradictory results clearly indicate that the relations of bases to acids in such fluids are unstable, and vary with circumstances.

The degree of acidity in the urine (which must be estimated from the secretion collected through the whole twenty-four hours) is commonly expressed in terms either of the dried carbonate of soda required to neutralise it, or of an equivalent weight of oxalic acid. Roberts found that in a healthy man it amounted on an average to about fourteen grains of the carbonate; but there are wide variations, the range during a period of nineteen days being from six to more than twenty-three grains. Writers who give it in terms of oxalic acid say that it corresponds to about thirty grains.

At different periods of the day, however, the reaction of the urine is by no means uniform. After each of the principal meals it becomes for a time decidedly less acid, and may even be alkaline. This now well-known fact was originally pointed out by the late Dr Bence Jones. The cause of the

change is believed to be, not the formation of gastric juice, but the absorption into the blood of alkali produced by the digestion of the citrates, tartrates, malates, &c., derived from the food. Consequently it is likely to be well marked after meals consisting largely of fruits and of other substances in which salts of the vegetable acids are abundant. In medical practice the effect of food upon the reaction of the urine is seldom noticed except by accident, because what is passed from the bladder is generally a mixture of the secretions formed by the kidneys over a considerable period of time. But one must always be prepared to find a specimen alkaline, as, for example, when it is passed in one's consulting-room in the course of the morning, by a patient who has been to the closet after his breakfast. The urine passed by candidates for insurance, between one and two o'clock, after lunch, is very often neutral or alkaline.

In some abnormal conditions, on the other hand, the urine passed throughout the whole twenty-four hours is found, when collected, to have an alkaline reaction, not due to ammoniacal fermentation of the urine after it is secreted. Quincke has observed this in patients with chronic vomiting, as the result of dilatation of the stomach; and he has doubtless rightly attributed it to the abstraction from the body of the acid of the gastric juice which normally should be reabsorbed into the blood. A like condition of the urine has also been noticed in patients who have the stomach regularly washed out.

Under other circumstances a persistent alkalinity of the urine from fixed alkali is not common. But Bence Jones observed such cases and so have Hutchinson and Roberts. Some of these patients were anæmic after subacute rheumatism or gout, chlorotic, dyspeptic, or phthisical; but others were apparently in tolerable health. Often the urine is alkaline for two or three days together, and then acid for a time, becoming again alkaline later on. But sometimes it remains steadily alkaline for weeks.

With vegetarians, as with herbivorous animals, an alkaline state of the urine is natural.

Deposits.—Urine which is alkaline from fixed bases is commonly turbid when passed, and precipitates certain of its solid constituents, chiefly phosphates of the alkaline earths. The amorphous phosphate of lime ($Ca_3P_2O_8$) collects as a flocculent deposit, always whitish and paler than the supernatant liquid, and thus distinguishable from the fawn-coloured or brick-red amorphous lithates. It also forms an iridescent film upon the surface, the cause of which appears to be the escape of carbonic acid from the urine. Mixed with this is sometimes, though rarely, another phosphate of lime ($CaHPO_4 + 2Aq.$) which is crystalline. It forms rods or needles, smaller at one end, so as to be club- or bottle-shaped, and generally grouped together into stars, rosettes, fans, or sheaf-like bundles. Still more infrequent is the phosphate of magnesia ($Mg_3P_2O_8$) which forms elongated plates with oblique ends. It seems only to have been recognised by two observers, Tollens and Stein. In fact, it is a very soluble salt, so that it is not likely to be precipitated unless present in very large quantity: and, moreover, there must be no ammonia in the urine, since that base, if present, unites with the phosphate of magnesia to form another salt, the well-known "triple phosphate" ($MgNH_4PO_4 + 6Aq.$).

All phosphatic deposits are instantly dissolved by acetic acid, a test distinguishing them from other urinary precipitates.

Carbonate of lime seems to be frequently mixed in small quantity

with the amorphous phosphate of lime; sometimes it appears in small spheroids.

Not infrequently urine when warmed in a test-tube becomes cloudy and opaque, so that it may look as if it contained albumen, owing to a separation of phosphate of lime that had hitherto been retained in solution. A drop of acid, however, decides the point by dissolving the cloud. This precipitate has generally been attributed to the driving off of carbonic acid from the urine. But Salkowski has pointed out that this cannot be the case, since when the turbidity is but slight the urine often becomes quite clear again on cooling. Dr Walter Smith ('Dublin Jour. of Med. Sci.,' 1883) has since taken up this subject, and has arrived at the conclusion that the precipitation of phosphate of lime by heat depends upon "a nice adjustment of the proportions and basicity of the phosphatic salts in the urine." He imagines that when the fluid at the ordinary temperature contains the dicalcic phosphate ($Ca_2H_2P_2O_8$), held in solution by the presence of other salts, the effect of heat may be to resolve it into tricalcic phosphate (Ca_3PO_4) and monocalcic phosphate (CaH_2PO_4), the former of which is insoluble; cold, on the other hand, may lead to the inverse change and to a disappearance of the precipitate. Urine which thus deposits its phosphates on being heated does not always do so on neutralisation by acids; indeed, it is sometimes alkaline or neutral to begin with.

If the test-tube is heated, until all the precipitate has fallen, and this is allowed to subside, the clear supernatant urine will again deposit a cloud of earthy phosphates on being heated, and this may be repeated several times.

The presence of a phosphatic deposit in urine alkaline from fixed alkali may be regarded as a matter of no consequence, so far as concerns the formation of concretions within the urinary passages, since the amorphous phosphate of lime which forms the bulk of it has scarcely any tendency to cohere into solid masses. Only the very rare calculi which consist entirely of this substance can have had their origin in such a state of the urine.

Nor is there any ground for supposing that visible precipitation of phosphates, whether spontaneous or as the result of heat, is an indication that these salts are being excreted in excess. To determine that, it would be necessary to make an exact quantitative analysis of the twenty-four hours' urine. Such analyses have been often made, but with the most meagre results, so far as their clinical value is concerned. There is therefore no antecedent probability in favour of the view, which at one time was entertained by good observers, that a "phosphatic" state of urine is dependent upon an undue disintegration or waste of the nervous tissues. And certainly direct evidence in support of such a view is altogether wanting.

In the *treatment* of cases in which the urine is habitually alkaline from fixed alkali the main thing is to improve the general health by change of air to the seaside or to a mountain health resort, exercise short of fatigue, and other measures tending to the same end. The direct administration of acids is found to have very little effect.

Urinary Pigments.—With regard to the substances that give to urine its various shades of colour in health and in disease there is still much uncertainty and confusion.

Urobilin.—According to Salkowski and other recent writers, the principal urinary pigment is a substance to which Jaffé first gave the name of *urobilin* (*hydrobilirubin* of Maly): it gives peculiar absorption lines in the spectrum,

and a green fluorescence is produced by its ammoniacal solution on the addition of chloride of zinc. Vierordt, however, has pointed out that this cannot be the only colouring matter, inasmuch as the spectrum of urine is not absolutely identical with that of a solution of urobilin. And Huppert, in his eighth edition of the well-known work of Neubauer and Vogel, declares that the real pigments of normal urine are still unknown. Urobilin, he states, is not present as such, but in the form of a chromogen, which only yields it on the addition of mineral acids. But in certain pathological conditions urobilin exists in the urine in a formed state; and the quantity that can be extracted is far greater than natural, though it still amounts to only from $\frac{1}{3.2}$ to $\frac{1}{16}$ in a thousand parts of the fluid. Urobilin ($C_{32}H_{44}N_4O_7$) can be derived from bile-pigment, or from hæmatin or hæmoglobin, by a process of deoxidation; and MacMunn has found it can be oxidised to choletelin. Salkowski says that its origin within the human body is from the bile in the intestines, under the reducing influence of hydrogen set free as the result of putrefaction and other chemical processes that take place there. In fact urobilin gives the fæces their colour, all the bilirubin which passes into the bowel being converted first into choletelin and then into urobilin.

It is especially in febrile urine, and in that passed by patients with obstruction of the venous circulation, that urobilin is found in excess. But it must be remembered that such urine is generally scanty, so that the increase may not be so great as it appears. Salkowski says that constipation seems not to augment the urobilin in the urine. In jaundice there is often a great excess, which, however, can be recognised only after the bile-pigment has been precipitated and removed. The urine may likewise be found loaded with urobilin before an attack of jaundice and after it has passed off. Surely its presence in jaundice (for which we have the authority of Hoppe Seyler) shows that absorption of deoxidised bile-pigment from the intestine is not the only source of urobilin in the healthy urine.

Indican, &c.—The fact that *indigo-blue* is sometimes present in the urine was noticed many years ago by Prout, and afterwards by other observers, some of whom showed that this colouring matter in many cases made its appearance only when the urine had been exposed to the air. Schunck, of Manchester, first recognised in urine the constant presence of *indican*, a colourless material, which he had also discovered in plants, and which readily passes into indigo-blue by oxidation. More recently, however, it has been found that the indican of urine is not identical with vegetable indican; according to Baumann, it is an indoxyl-sulphate of potass ($C_8H_6NKSO_4$). The only observations by which any clear light seems as yet to have been thrown upon the origin of this substance in the human body are those made by Jaffé in 1872, and by Baumann and Brieger in 1879, who observed that indican could be made to appear in the urine of animals in large quantity by feeding them with the allied substance indol (C_8H_7N), or by injecting that substance under the skin. Now, indol is formed within the intestine in dogs, and to some extent in man, as the result of a change in albumen induced by the pancreatic ferment. The absorption of it from the intestine may therefore be fairly supposed to give rise to the presence of indican in the urine; and Jaffé has in fact detected indican in very large quantity in cases of obstruction of the small intestine or of strangulated hernia, and also in dogs after ligature of a loop of small intestine. But it is quite another question whether this is the sole, or even the usual, source of a morbid excess of indican in the

urine. Senator ('Centralblatt,' 1877) and Heninger ('Deutsches Archiv,' 1879) have investigated the conditions under which such an excess is met with. The former finds it in states of inanition and wasting, such as arise from cancer of the stomach, gastric ulcer, multiple lymphomata, phthisis with diarrhœa, or granular disease of the kidneys. The latter insists that the excess is especially marked in cases in which wasting is dependent upon affections of the intestinal canal. He observed it not only when there was constipation, but also when diarrhœa was present, and even in cases of *cholera nostras*. On the other hand, in cases of catarrhal jaundice and in cases of cirrhosis of the liver, the amount of indican in the urine was always small. The general result of these observations appears to be that the recognition of indican in the urine is at present useless from a practical point of view. It is often found in large quantity in urine which is pale and contains little formed pigment.

The test for indican, as given by Jaffé, is to add to the urine an equal volume of hydrochloric acid, and then to pour in drop by drop a solution of chloride of lime, shaking the fluid well, and adding no more of the chloride after a greenish colour begins to appear. If any considerable quantity of indican is present, a blue colour will soon show itself; and if the quantity is very large, indigo-blue will be deposited in flocculi. A dilute solution of bromine may be used instead of the chloride of lime. The blue pigment may be afterwards extracted by agitating with chloroform or ether; and in this way the amount of it may be roughly estimated.

According to Brieger, a still more frequent constituent of human urine is an allied substance, skatoxyl-sulphate of potass, which is derived from *skatol* (C_9H_9N), this being (like indol) a product of the decomposition of albuminous substances within the intestine. Both indol and skatol have a strong fæcal odour. Jaffé's test with hydrochloric acid and chloride of lime gives a reddish-violet instead of a blue colour, when the skatoxyl-sulphate is present.

Another urinary pigment which appears to be different from all of those hitherto mentioned, has been called *uro-erythrin;* it gives a pink or red colour to deposits of lithates. It is amorphous, not very soluble, and turned green by Liq. Potassæ. Uro-erythrin is sometimes spoken of as identical with what Dr Golding Bird called "purpurin." Probably, however, he included under that term several substances which are now described as distinct, since his test for it was to add hydrochloric acid to the hot urine, when he obtained a colour "varying from a delicate lilac to the deepest crimson."

Adventitious pigments.—In conclusion, it must never be forgotten that colours that may be mistaken for the effects of urinary pigments may be due to the administration of drugs. For example, rhubarb colours the urine a deep gamboge yellow, which is changed to red by the addition of ammonia. After operation under the carbolic acid spray, patients often pass urine of a deep, sometimes almost black colour. Senna communicates a brownish, and logwood a reddish tinge. Santonin gives a conspicuous orange-yellow colour to the urine if alkaline, a rich golden-yellow if acid.

A curious point observed by the late Dr Moxon is, that in persons who are taking iodide of potassium the addition of nitric acid to the urine produces an orange-coloured zone, the appearance of which is characteristic.

Saline Deposits.—We have already spoken of certain sediments in the urine: of tyrosine in cases of acute atrophy of the liver, and of phosphate

of lime in conditions in which the urine is alkaline; and the precipitation of the phosphate of ammonia and magnesia will be described further on, as an effect of ammoniacal fermentation.

Most of the urinary sediments now to be described are of clinical importance from one or both of two different points of view: either as indications of disturbance of the chemical processes in some other part of the body, or else as involving the risk of the formation of gravel or calculus within the urinary organs. They are (1) lithic acid and the lithates, (2) oxalate of lime, (3) cystine, (4) sulphate of lime.

Lithic (or *uric*) *acid and the mixed urates.*—In urine having the normal acid reaction lithic acid exists in the form of acid salts of soda and other bases, which at the temperature of the body are fairly soluble. But as the fluid cools, these are often precipitated. Very slight changes may disturb the balance between them and other saline ingredients, so as to separate the lithic acid, which is then thrown down, inasmuch as it requires a very large quantity of water (14,000 parts in the cold) to hold it in solution. Scherer many years ago asserted that during the first few hours after being voided, urine, as a rule, undergoes what he termed an "acid fermentation." This, however, is not now believed to be the case. By Voit and Hoffman ('Bayerische acad. Sitzungsbericht,' ii, p. 79) it is maintained that the acid phosphate of soda (which is commonly held to be the cause of the acid reaction of urine) gradually takes away from the uric acid more and more of the bases with which it is combined. It is said that an appreciable diminution in the acidity of the urine may be produced in this way, inasmuch as the uric acid, being deposited in a solid form, is no longer capable of affecting the reaction.

Uric or lithic acid ($C_5H_4N_4O_3$).—As a deposit from urine, this substance appears in the form of crystalline grains which have a reddish colour, so that they often look almost exactly like cayenne pepper. They commonly lie loose at the bottom of the fluid, but sometimes they adhere to the sides of a glass vessel, or may float in a film upon the surface. Their colour is not proper to the acid itself, for this, when derived from other sources, is colourless; it really belongs to urinary pigment, for which uric acid seems to have a strong attraction, and which is consequently carried down with it. Dr Lionel Beale says that he has three or four times seen colourless crystals of uric acid deposited from urine which happened to contain hardly a trace of colouring matter.

The form of uric acid crystals is primarily that of a rhombic prism or lozenge, but they present a great many varieties of shape. Sometimes they are short and thick and barrel-shaped, or almost cubical; sometimes they form rods which seem to have rectangular extremities; sometimes they appear as flat plates which may be fiddle- or halberd-shaped. Very often they form large stellate aggregations, and sometimes fan-shaped masses, which may occasionally be connected together in pairs so as to have somewhat the character of "dumb-bells." Dr Ord has pointed out ('Med.-Chir. Trans.,' lviii) that the deviations in form which those crystals present from the regular four-sided or six-sided plates that are seen when the pure acid is crystallised from water, depend upon the presence of mucus and of colouring matter, which substances favour Rainey's "molecular coalescence" rather than crystallisation, so that by a kind of compromise the resulting crystals, instead of having sharp angles and straight sides, are more or less rounded

off, and also tend to cohere together in masses having a common centre. He has also found that the presence of albumen in the urine still further modifies the form of the crystals, rendering them small and thick, with their angles more or less nearly equal, so as to give them the barrel- or cask-shape. The association with sugar, on the other hand, tends to produce flat and elongated crystals, which may have the typical hexagonal shape that is otherwise so rarely seen in specimens derived from urine. The microscopical character, or generally even the naked-eye appearance, of a deposit of uric acid is quite sufficient to distinguish it from any other substance that occurs in the urine. But unless the quantity be very small, the well-known murexide test can be easily applied.

Mixed amorphous lithates or urates.—The commonest of all urinary deposits consist of a loose pulverulent substance, which varies very much in tint, but is always of a deeper colour than the urine from which it is derived. It is often spoken of as brick-coloured, or "lateritious" (*later* = a brick). It generally settles quickly, leaving the urine above almost clear, but sometimes it remains a long while diffused through the fluid. Not infrequently, if the urine has been put aside while still warm, different strata of the deposit in the same glass have different colours: fawn-coloured, orange, brick-red, pink, or purplish. A part adheres to the side of the vessel as a sort of film or bloom, which is not very easily cleaned away. With the microscope this precipitate is seen to consist of minute granules, which are coarser or finer, and more or less opaque, according to the closeness of its aggregation. All doubt as to its nature may be removed by applying heat. As soon as the fluid is warmed, it becomes bright and clear; and even when albumen is present, there is seldom any difficulty in obtaining a satisfactory result, for the urates dissolve at a far lower temperature than that at which albumen coagulates, though of course more heat is required to clear up a dense precipitate from which the supernatant fluid has been poured off than if it were merely diffused through a bulk of urine.

Until lately the lateritious deposit was commonly spoken of in this country as consisting of the urate of ammonia, while German writers described it as a urate of soda. In reality it consists of a mixture, in different proportions in different cases, of the urates of soda, potass and lime, to which that of ammonia is sometimes added. Moreover, the quantity of uric acid in it is largely in excess of that which would correspond even with the acid salts of these bases (for uric acid is bibasic), being in fact about twice as much, and making up 80 or 90 per cent. of the whole precipitate. Roberts says that this loosely combined acid can be separated from the acid lithates by warm water (which must of course be used sparingly), or even by cold water, with which the deposit is to be repeatedly washed upon a filter.

Crystalline lithates or urates.—It is a curious circumstance that urate of soda is never deposited from urine in those needle-shaped crystals with which we are familiar in gouty concretions, and which may also be readily obtained artificially from solutions of the salt. In some cases, however, it forms opaque globular masses from which project spiny crystals, straight or curved. These "hedgehog" or "thornapple" *(Stechapfel)* bodies occur in putrid urine. But they are also sometimes seen in patients (especially children) who are suffering from pyrexial disorders. Their occurrence probably depends upon the urine being very scanty and concentrated, and being detained in the bladder. In one case a child three years old was

suffering from fever, and had passed no water for three days: while Roberts was examining the abdomen, the child cried, and urine began to flow: the first that came was turbid and of a gamboge-yellow colour, containingthe spiny masses; then, after about an ounce had passed, there followed several ounces of clear fluid.

Clinical significance.—In medical practice, deposits of uric acid and of urates have to be looked at from two points of view.

First, there is the question whether they are the cause, or likely to be the cause, of calculi, or of lumbar pain, hæmaturia, and other symptoms of pyelitis. Now, as regards free uric acid, much depends upon whether or not it is precipitated soon after the urine is passed from the bladder. As Roberts remarks, if the crystals are seen before the urine cools, there is always a risk that they may also be found within the urinary passages. Even if the deposit takes place within three or four hours, it is certainly not natural, though it hardly requires special treatment. But if it does not occur until after twelve hours or longer, it has no pathological significance whatever.

Amorphous urates cannot in themselves produce irritation of the kidneys or bladder, since they appear never to be precipitated so long as the urine is of the temperature of the body. But the hedgehog crystals of lithate of soda are perhaps less innocent.

Secondly, there is the question how far deposits of lithic acid or of lithates indicate a disturbance of the chemical processes by which nitrogenised substances in the blood or in various organs are prepared for excretion by the kidneys. Now, it cannot be too strongly insisted on that the formation of such precipitates is not in itself a proof that the quantity of uric acid is excessive. In the case of the amorphous urates a great deal depends upon the *temperature* to which the urine falls when it cools. It does not appear that there is any other cause than the lower temperature for the fact that deposits of these substances are so much more frequently observed in winter than in summer. Again, whatever diminishes the *amount of water* excreted by the kidneys increases the likelihood that urates will be precipitated. This seems to be the reason why amorphous urates are so often seen in healthy persons after violent exercise and after profuse sweating, and also in patients who are suffering from any disease, such as rheumatic fever, attended with perspiration in its whole course, or who are passing through the crisis of an attack of pneumonia when there is excessive perspiration. Thirdly, the degree of *acidity* of the urine is also of importance. Most observers say that urates are never deposited except from urine which is acid, but according to Salkowski the reaction is occasionally found to be neutral. In the case of free uric acid, the degree of acidity of the urine is the most essential factor of all in determining its precipitation.

To determine the exact significance of deposits of uric acid or of urates would be a comparatively light task if it were easy to make quantitative analyses by which the amount of the acid in a given specimen of urine could be exactly ascertained; but unfortunately this is very far from being the case. The usual plan is to precipitate with hydrochloric acid, to collect the uric acid upon a filter, and to weigh it. This, however, is found not to give correct results, since a variable amount of the uric acid remains in solution; it may even happen that no precipitate occurs notwithstanding that uric acid is present. Salkowski, therefore, has proposed a method which depends upon the formation of a compound salt of sodium

and silver; this, he says, is much more accurate and takes less time, but there are difficulties in carrying it out correctly. Dr Pavy has recently advocated ('Med.-Chir. Trans.,' lxii) the use of the ammoniated cupric solution, which he employs for the estimation of sugar. He finds that 0·01866 gramme of uric acid is required to decolourise 20 cc. of this test. The reducing action of healthy urine, however, is not due solely to the uric acid in it; a small quantity of sugar is also usually present, which takes part in the effect. Dr Pavy therefore repeats the experiment with a second specimen of urine from which all the uric acid has been precipitated by acetate of lead. The difference between the two results gives the amount of uric acid which the urine contains. Dr Pavy gives figures which appear to prove the accuracy of this method. The total quantity of uric acid excreted by healthy persons in the twenty-four hours is not large; as a rule, it is from three to eight grains. The proportion between the uric acid and the urea is usually as one to fifty or as one to sixty. Dr Haycraft has introduced another method which will be found described in the 'Journal of Anatomy and Physiology,' vol. xx, p. 695.

Liebig suggested the idea that uric acid is formed by oxidation out of the same materials as urea, and represents a stage in the formation of the latter substance in which the oxidising process is as yet incomplete. Unfortunately this hypothesis seems to be incapable of proof. The most obvious way of testing it seems to be that of adding uric acid to the food, and determining whether or not the amount of urea excreted afterwards undergoes an increase. Wöhler and Frerichs originally proposed this experiment, and came to the conclusion that the uric acid was mainly converted into urea; other observers have since repeated it with a like result. Salkowski found in dogs that allantoin appeared also to be formed out of the acid, but as this must itself have arisen by a process of oxidation, he regards it as rather tending to confirm than to upset the view that uric acid constitutes a step in such a process. The same may be said likewise of the facts which seem to show that oxaluric acid and oxalic acid may be formed in the body out of uric acid. And, on the other hand, it is a significant fact that in birds uric acid is derived from the same substances (aspartic acid, glycin, leucin, and asparagin) which in mammalia pass into urea. Urea itself, when given to birds, is said to undergo conversion into uric acid; this, at any rate, indicates how close is the relationship between the two bodies. But according to the best physiologists the formation of uric acid is separate from that of urea throughout, and is not a stage in the process.

Clinically, several sets of facts have been adduced in support of the view that uric acid may be excreted in excess as the result of a deficient supply of oxygen, and take the place of urea; but this explanation seems to be open to question in every case. For example, as to the "uric-acid infarcts," which are seen in the kidneys of newly-born infants, Cohnheim remarks that he has observed them especially in strong healthy children who had breathed well, whereas they were often absent in cases in which there was pulmonary atelectasis, bronchitis, or broncho-pneumonia. So, again, many observers have shown that there is in leuchæmia a marked increase of uric acid in the urine, both absolutely and relatively to the urea. This has been attributed to the deficiency of red discs as oxygen carriers. Pettenkofer and Voit, however, found ('Ztschft. f. Biol.,' v) that in a case in which the excess of uric acid amounted to 64 per cent., the absorption of oxygen, and the evolution of carbonic acid from the lungs were normal. Lastly Bartels some

years ago ('Deutsches Arch.,' 1865) endeavoured to show that in various affections attended with insufficient aeration of the blood, the amount of uric acid in the urine is increased in proportion to that of the urea, the proportion rising from the normal rate of one to fifty or sixty up to that of one to thirty-five, or even of one to twenty-five. But experiments made on animals by Senator and others have for the most part failed to confirm these observations; and of late it has been shown by Fränkel, and also by Fleischer ('Virch. Arch.,' 1882) that a constant effect of dyspnœa is actually to augment the secretion of urea. Possibly the over-activity of the respiratory muscles plays a part in bringing about this result, and at any rate we may agree with Salkowski that the conditions in dyspnœa are far too complicated to allow of our attributing solely to deficiency of supply of oxygen the changes in the urine that may be associated with it.

But it is especially in relation to disorders of the chylopoietic viscera that the meaning of deposits of uric acid or of urates has to be considered by practical physicians. Roberts made during seven days a series of observations on a person who dined at 2 p.m. and afterwards took no solid food till the next morning. He found that during the period when the urine was alkaline after the meal—which was from about 4 p.m. till 7 p.m. —the quantity of uric acid excreted in each hour was three times greater than it was from 9 p.m. till 11 p.m., or later on during sleep. The proportion which the acid bore to the rest of the urinary solids, and even to the water, was likewise greater, though, being alkaline, the urine of course threw down no deposit of urates. The food taken while these observations were being made was very simple. Consequently, it seems fair to conclude that the effect of rich food and of frequent meals in increasing the excretion of uric acid would be even more decided. And, as a matter of fact, many persons who habitually live plainly, and who ordinarily pass clear urine, find that the occurrence of a lateritious deposit is an inevitable consequence of any considerable indiscretion in diet; while other persons, whose rule it is to indulge in the pleasures of the table, secrete during a large part of every day urine that becomes turbid with urates as it cools. It is true that such deposits do not in themselves prove an excessive excretion of lithic acid, but, if we also take Roberts's observations into consideration, we may surely infer that this is in reality the case. At any rate, no theoretical doubts can do away with the practical significance of this symptom, especially if it is associated (as we so constantly find it to be) with a foul tongue, yellow conjunctivæ, an irritable temper, and all the other signs which have been enumerated at p. 489 as symptoms of lithæmia. Of course, even if we admit that the amount of lithic acid contained in the urine in such cases is absolutely increased, it does not at all follow that it is increased relatively to the urea. That substance itself may be increased likewise, though, as it is readily soluble, there is nothing to show it. But what seems to prove conclusively that deposits of lithates are not merely an indication of excessive nitrogenous elimination as the result of the ingestion of undue quantities of nitrogenised food is, that among the articles of diet which are most apt to be followed by the appearance of such deposits in the urine are some (such as sweet things, port wine and champagne) which are themselves non-nitrogenous. It is difficult to resist the conclusion that such substances are capable of disturbing the balance of the chemical changes which nitrogenous foods (and also probably the products of the waste of nitro-

genous tissues) undergo in the liver or elsewhere in the body, and of causing an excess of uric acid to be formed. Parkes found, it is true, that no excessive excretion of uric acid occurs as the result of the experimental addition of either sugar or starch or alcohol to the food. But, in all probability, the limits of health were not overpassed in the observations upon which these statements were founded, as they are in the cases of lithiasis just referred to.

The presence of colouring matter and chromogens in the urine in excessive quantity is generally held to afford corroborative evidence of the same kind beyond that which is afforded by the mere presence of the uric acid or of the urates. The deeper the tint of a lateritious deposit occurring in a non-febrile patient, the more surely would it be generally regarded as a proof of lithiasis. Probably the same conclusion may in most cases be safely drawn from the formation of pigmentary matters in large quantity on the addition of mineral acids to urine. The late Dr Golding Bird, for example, was convinced that the presence of what he termed "purpurine"—detected by the violet colour which followed the addition of hydrochloric acid to urine and heating—was a proof of "derangement of the hepatic function." He thought it was sometimes a valuable clinical aid to diagnosis in cases in which there was an abdominal tumour of doubtful nature, or in which it was uncertain whether ascites was due to hepatic disease or to chronic peritonitis. The same significance applies to the zone of pigment of various hues that is so commonly observed when urine is poured upon nitric acid. This seems to depend upon the presence, in different amounts in different cases, of all the substances mentioned at pp. 568, 569: the chromogen of urobilin, indican, and skatoxyl-sulphuric acid. And accordingly the tint is sometimes crimson, sometimes purple, sometimes bluish black. Perhaps the purple and indigo-blue colours may be evidences of constipation.

Oxalate of lime ($CaC_2O_4 + 4Aq.$).—This substance produces in the urine a cloud like that caused by mucus, and so little noticeable in an ordinary vessel that a patient's attention is seldom, if ever, attracted by it. Roberts, however, says that when the deposit is allowed to form in a conical glass vessel it presents appearances which are quite characteristic. The sides of the glass become marked with fine lines, running transversely or obliquely, and making it look as if it were scratched; these are due to the crystallisation of the salt along minute irregularities left on the surface of the glass when wiped. In the urine itself there is near the bottom of the vessel a soft pale grey mucous-looking sediment, and above this a snow-white denser layer with an undulating but sharply-limited surface. If a drop of the deposit be taken up with a pipette, and placed under the microscope, the oxalate is generally seen to form small colourless octahedral crystals, beautifully transparent and lustrous.* So transparent are these octahedra that all their facets and angles are commonly visible at the same time. They have a flattened shape, the principal axis being much shorter than the other two. In size they vary considerably; the late Dr Golding Bird found that the length of the sides of different specimens ranged from $\frac{1}{5600}$th to $\frac{1}{750}$th of an inch. They usually lie upon one pole, and then have the appearance which is aptly compared to that of a square envelope. But in different positions they may seem to have a rhombic outline, or

* In a case of jaundice they are said to have been found by Fürbinger stained yellow by bile.

even one which is rectangular or hexagonal. Other modifications in their shape are due to flattening of their lateral edges. This gives them a dodecahedral form, so that they consist of a rectangular prism with a pyramid on each summit. It is also said that half crystals are sometimes seen—pyramids on a square base. Much more rarely, oxalate of lime assumes a non-crystalline character; it is then said to appear in the form of "dumb-bells." Recent observers, however, have shown that the real shape of these bodies is that of a flattened, rounded disc, with a central depression in each surface; it is when these discs lie on the side that they look like dumb-bells. They, too, vary in size. According to Dr Golding Bird the long diameter ranges from $\frac{1}{1420}$th to $\frac{1}{500}$th inch; their short diameter from $\frac{1}{2500}$th to $\frac{1}{750}$th of an inch. There was at one time some doubt as to the chemical nature of the dumb-bells. Dr Bird was disposed to think that they differed in composition from the octahedra, and suggested that they might consist of the oxalurate of lime. But this question has been finally set at rest by the observations of Dr Ord, who has shown that when oxalic acid and lime come into contact in the substance of a mass of gelatine, both forms are obtained. The assumption of a discoid, rather than of a crystalline character, is doubtless due to the influence of colloid substances. Dr Beale has shown that dumb-bells are sometimes found in the interior of renal tube-casts.

Chemically, oxalate of lime may be distinguished by its being insoluble in potash and in acetic acid, but soluble in nitric and in strong hydrochloric acid. The shape of the octahedra, however, is in itself a sufficient proof of their nature; it is only the dumb-bells which might be mistaken for like bodies of a different composition.

The origin of oxalate of lime is still unknown. There is no doubt that it may in part be derived directly from the food. Many vegetables and fruits contain this salt; not only rhubarb (which is so largely eaten in the spring in England) and sorrel (which on the Continent forms a common article of diet), but also, to a less extent, spinach, cauliflower, asparagus, tomatoes, apples, and grapes. Buchheim and Piotrowski are said to have found that from 10 to 14 per cent. of the quantity of oxalate ingested could be recovered in the urine. But Auerbach detected oxalate of lime in the urine of dogs when fed entirely on animal food; so that probably oxalic acid may be also formed within the human body as the result of chemical changes. But as yet there is no certainty as to its source. English physicians of a former generation were inclined to think that it arose out of sugar; its presence in the urine in a conspicuous form has sometimes been observed in connection with diabetes; but this can hardly be said to have much bearing on its origin. A more probable view is that it is derived from uric acid, by a process of oxidation with which chemists in the laboratory are familiar. Schunck's discovery, that oxaluric acid in minute quantity is a normal urinary constituent, is generally held to confirm this view, inasmuch as that substance forms a step in the process in question. But it seems not yet to have been positively shown that the ingestion of uric acid is followed by an increased excretion of oxalic acid.

Prout and Golding Bird regarded the recognition of the oxalate of lime in the urine as affording a key to the right explanation and to the successful treatment of a group of symptoms which they enumerated, and of which the chief were a constant pain or sense of weight across the loins, irritability of

the bladder, incapacity for exertion, impairment of sexual power, dryness of the palms of the hands and soles of the feet, a painful susceptibility to external impressions, nervousness, hypochondriacal depression, and emaciation. But in the urine of many patients who have such symptoms no oxalate can be found, while in that of many other patients who are quite free from symptoms it is present in abundance. Again, Fürbinger has shown that no conclusion as to the quantity of the salt contained in the urine can be drawn from the fact that crystals are discovered in the urine. Healthy persons, according to Schultzen ('Reichert's Archiv,' 1868), pass about a grain and a half in the whole of the twenty-four hours; in a morbid condition, in cases of jaundice, he found as much as seven and a half grains. But, from what is known with regard to uric acid, it might well appear that this increase is too small to allow of its leading to disturbance of the general health.

However this may be, the chief clinical importance of oxalate of lime in the urine depends upon the risk of its deposition while the secretion is still within the body. It may furnish a clue to the probable presence of a calculus formed of the same substance, or to that of minute agglomerations producing lumbar pain, hæmaturia, or pyelitis. All of these effects will be discussed further on. At present it only remains to state the conditions under which the salt is apt to be deposited.

It has long been known that, in some cases at least, the octahedral crystals are formed slowly in the urine *after* it has been voided. They have been found to be both more numerous and larger in urine that has stood for a time than they were when it was first passed. Dr Rees even maintained in his 'Croonian Lectures' for 1856 that the oxalic acid itself arose out of uric acid in the urine itself, especially when heat was applied to it for testing purposes. This, however, seems to be very doubtful; and it may be noted that Neubauer ('Ztschrift. f. anal. Chem.,' 1868) found that even on adding oxalurate of ammonia and chloride of calcium to urine no oxalate of lime was formed, the only change being the conversion of the oxalurate into carbonate of ammonia when the fluid putrefied and became alkaline. Moreover, Voit and Hoffman seemed to have satisfactorily accounted for the gradual separation of the oxalate of lime from urine which had for a time held it in solution. Neubauer, as far back as 1856, showed that this salt can be kept dissolved by a solution of the acid phosphate of soda. Now, it appears that when urine is allowed to stand, the urate of soda in it becomes acted upon by the acid phosphate of soda, so that an acid urate of soda, and ultimately free uric acid, are formed, while the phosphatic salt loses its acidity. Consequently, the conditions under which alone the oxalate of lime can be held in solution are no longer present, and it crystallises out. Obviously, therefore, the fact that octahedra are discovered in a patient's urine is in itself no reason for supposing that there is danger of the deposition of oxalate of lime in the renal pelvis or in the bladder. And it would seem to follow that the danger must be less in proportion as the urine is more highly acid. Almost all observers, however, are agreed that oxalate crystals are most often found in very acid urine; Salkowski alone (influenced possibly by theoretical considerations) speaks of them as being present chiefly in urine that is only just acid, neutral or faintly alkaline. They are sometimes seen in association with deposits of urates or of uric acid; and radiating crystals of phosphate of lime are not uncommon in urine containing oxalates, even, as Dr Beale says, when it is acid.

Oxalate of lime is insoluble in hot and cold water, in potash, and in acetic and dilute hydrochloric acids. Only strong hydrochloric acid, with the aid of heat, decomposes this very stable salt.

From what has been already stated, it is evident that *treatment* is by no means necessary for all cases in which oxalate of lime is discovered in the urine. Dyspepsia and the effects of dyspepsia must be dealt with in the same way as if no such deposit were present. As a rule the nitro-hydrochloric acid does more good than alkalies. Cold sponging, the use of a flesh-brush, exercise on horseback, change of air to the seaside or to an elevated health resort, are each of service. The food should be light, digestible, and varied. Leube, however, cites Cantani as having found that the urine was rendered free from the oxalate by an exclusive meat diet. Patients who have had lumbar pain or hæmaturia should be cautioned against eating rhubarb. To prevent the deposition of the salt within the urinary passages the best plan seems to be that of maintaining the naturally acid state of the urine; but Roberts says that in some cases the urine has temporarily ceased to contain the crystals when it was rendered freely alkaline.

Cystine ($C_3H_5NSO_2$).*—In 1810 Wollaston discovered, in analysing a urinary calculus, that it was composed of a peculiar substance, to which he gave the name of "cystic oxide." Subsequently the same substance, which is now almost always known as cystine, was found to occur as a light flocculent deposit from the urine. It looks very like a fawn-coloured sediment of amorphous urates, but with the microscope it is seen to be crystalline, consisting of hexagonal tablets, which Roberts describes as having an iridescent, mother-of-pearl lustre, and as being often chased on the surface by lines of secondary crystallisation; there may also be thick rosettes of great brilliancy.

Acetic acid throws down from urine exhibiting this deposit an additional quantity of cystine, which had remained dissolved; and sometimes, perhaps, might reveal its presence in urine which contained it in too small an amount to yield a spontaneous sediment. But the occurrence of cystine in any form is very rare. Urine which deposits cystine is usually faintly acid, and is described as having "a honey-yellow colour, an oily appearance, and a peculiar sweetbriar odour." The deposit is instantly dissolved by caustic ammonia; when this evaporates, the hexagonal crystals reappear, but mixed with them there may also be highly refracting prisms lying singly or forming stars, which are never seen in the urinary sediment.

The formula for cystine corresponds with sulph-amido-pyruvic acid, pyroracemic or pyruvic acid being derived from lactic acid. From a chemical point of view, the most remarkable fact about it is the large amount of sulphur that it contains, nearly 26 per cent. of its weight. One effect of this is that, when urine holding cystine in solution decomposes, it evolves sulphuretted hydrogen, so that a glass vessel (if it happens to be standing in one) becomes blackened from the lead of flint glass. The composition of cystine forms an obvious point of resemblance between it and taurine, one of the elements of the bile. Hitherto, however, no one has succeeded in making out anything abnormal about the hepatic secretion in those persons who pass cystine in the urine. Nor has it been shown that this substance takes the place of other normal constituents of the urine containing sulphur. The amount of sulphates appears not to be much less,

* Not seven atoms of hydrogen, as usually given (Dewar and Gamgee, 'Journ. Anat. and Phys.,' vol. v).

if at all, in urine containing cystine than in that which is healthy; but whether there is any change in the quantity of the organic sulphuretted compounds that exist in healthy urine seems at present to be unknown. The absolute weight of the cystine excreted daily is probably never considerable; for it is a light, though comparatively bulky, deposit.

It is a curious circumstance that cystine has several times been observed in the urine of persons related to one another as brothers or sisters. It is more often seen in males than females, and in children or young adults than in those who are older, but Roberts had a patient whose age was fifty-seven. No condition of general ill-health is associated with the formation of this substance, and the only risk that appears to attach to it is that of the development of concretions or calculi. Some patients go on voiding it for many years continuously; in some cases it disappears from the urine for a time and then returns; ultimately the excretion of it may cease altogether.

The treatment that has been advised consists in the administration of the nitro-hydrochloric acid, or of tincture of perchloride of iron, but it is doubtful whether benefit is to be derived from either of these remedies.

Sulphate of lime ($CaSO_4 + 2aq.$).—Considering that both sulphuric acid and lime are normal constituents of the urine, it is remarkable that the not very soluble compound which they form is so rarely deposited. As a matter of fact it has only been recognised in two instances, first by Valentiner ('Ctrlblatt.,' 1863) and afterwards by Fürbringer ('Deutsch. Arch.,' xx). It formed a bulky white sediment consisting of long needles and prisms with oblique ends, arranged in sheaves and rosettes. Valentiner's patient was an anæmic boy, Fürbringer's a wasted man affected with paraplegia. The conditions required for the production of such a precipitate appear to involve something beyond the mere presence of a moderate excess of sulphuric acid and of lime in scanty urine, for in these respects there was no change in Fürbringer's case, when after about three weeks it gradually ceased to appear; and as the urine was highly acid he is inclined to think that there was a deficiency of the alkaline bases.

The Ammoniacal Fermentation of Urine.—As is well known, urine left exposed to the air soon undergoes a change which renders it turbid and offensive to the smell, and which may be regarded as putrefactive. It is of some practical importance to be aware that this change takes place earlier in hot than in cold weather, that it is accelerated by the presence of pus or blood or mucus, and still more by admixture with even the smallest quantity of urine already putrid, and that it occurs much less rapidly in urine which is concentrated and highly acid than in that which is pale and watery and of a faintly acid, or neutral, or alkaline reaction.

But far more important is the fact that this same ammoniacal decomposition may take place within the urinary passages, while the urine is still unpassed and may become a source of severe inflammation.

Decomposed urine may be recognised by its penetrating fœtid odour, an odour which is unfairly termed urinous (since it is quite unlike that of healthy urine), and the immediate cause of which has not been exactly ascertained. It also gives off carbonate of ammonia, the pungency of which may irritate not only the nose but the conjunctivæ, if the vessel containing the urine be held close to the face. This carbonate of ammonia arises from the decomposition of the urea, thus: $CH_4N_2O + 2H_2O = (NH_4)_2CO_3$. It gives

to the urine an alkaline reaction, consequently the change which produces it is called the alkaline or ammoniacal fermentation. That the alkaline reaction is due to this cause, and not (as in other cases) to the presence of fixed alkali, can of course be easily determined. A piece of reddened litmus paper, or of turmeric paper, may be suspended in the mouth of a vessel over the urine but without touching it, when it will be found slowly to change its colour. Or the paper, after being dipped in the urine, may be left exposed to the air to dry, in which case the change in its colour that at first occurred will presently disappear. It must be remembered, however, that urine which was originally alkaline from fixed alkali may afterwards become putrid: in that case it will change the colour of paper suspended over it, but nevertheless paper dipped in it will continue to show an alkaline reaction after being dried.

Another character of urine that has undergone this change is its turbidity, a condition which filtering will not remove. The microscope shows that its cause is the presence of innumerable minute plants, occurring singly, or two or more in chains, or in a group with mucus as zoogloea. It is by these microphytes—*Micrococcus ureæ*—that the ammoniacal fermentation is brought about, probably immediately by a ferment which they secrete, just as alcoholic fermentation is brought about by the yeast plant *Torula cerevisiæ*.*

Urine placed in perfectly clean vessels, and guarded from the approach of organisms from without, can be kept free from this change for an indefinite length of time: and the same is the case in the body. Some observers have maintained that mere stagnation of urine in the bladder, or in the pelvis of a kidney, suffices to render it putrid, especially if it contain mucus or cast-off epithelium; but that this is not the case has been proved by experiments on animals, for when the neck of the bladder or ureter has been ligatured the urine has remained acid.

In too many cases the surgeon introduces the microphytes into the bladder by using catheters which had not been cleaned with the scrupulous care necessary to render them "antiseptic." This danger was pointed out by Traube, in 1864. The case which drew his attention to it was that of a man who had apparently had a distended bladder for two years, but whose urine was clear and acid when it was first drawn off by a catheter, whereas the next day it was turbid, and within six days it became alkaline, ammoniacal, and slightly fœtid.

It is, however, perfectly true that stagnation of urine in the bladder favours its decomposition. Cohnheim says that fluid containing bacteria may actually be injected into the bladder of a healthy dog without ill-effects, because they are all expelled the next time that the animal micturates. And in men, when this change has once occurred in the urine, nothing tends so much to keep it up as an inability on the part of the bladder to empty itself completely; so that some of the putrid fluid is always left behind to induce the same process in that which is afterwards secreted. It is a notorious fact, too, that formerly, when catheterism was constantly practised without any precautions against the introduction of septic matters, the urine seldom became ammoniacal unless either the bladder was paralysed or the urethra in some way obstructed.

* It is not certain whether the *Micrococcus ureæ* of Pasteur and Cohn and the bacterium of Leube are identical with the micrococcus cultivated by Dr Wm. Robert Smith, and described in the 'Quart. Journ. of Micr. Sci.' for 1887.

In some cases the putrefactive change takes place in the urine when no instrument has been passed. An abscess may have opened into some part of the urinary passages, and brought bacteria with the pus. Or bacteria may have found their way along the urethra from outside, possibly through a layer of mucus which may have been left upon the surface of the mucous membrane in the act of micturition, when the urine contains much mucus. Or possibly they may have come from the blood through the renal glomeruli.

One effect of the occurrence of ammoniacal fermentation, whether in the body or out of it, is entirely to alter the conditions under which the solid ingredients of the urine are held in solution. The phosphates are at once precipitated. The phosphate of lime ($Ca_3P_2O_8$), with a little of the corresponding magnesian salt, comes down in the form of amorphous granules, not as dumb-bells or crystals. There are also brilliant crystals of another salt, the phosphate of magnesia and ammonia ($MgNH_4Po_4 + 6aq.$), formerly spoken of as "triple phosphate." The form of these crystals is that of a triangular prism with bevelled ends; but they are liable to a great many modifications by planing off of their edges and angles, and sometimes their sides become hollowed out. Some of the prisms may be so short that their bevelled ends meet one another on one edge; they then look not unlike the octahedra of oxalate of lime. The addition of acetic acid at once distinguishes them; it dissolves all phosphatic deposits. Another element of the precipitate in putrid urine is occasionally formed by the lithates of soda and of ammonia: the former appears as round or irregular masses, from which project spiny crystals, straight or curved, like the "morning star" of mediæval warfare; the latter are opaque globular bodies, or very small, slender dumb-bells, which may lie across one another, or be united into a rosette.

The precipitate consisting of these various substances has no tendency to carry down urinary pigments with it, like the amorphous urates which occur in acid urine; it therefore has usually a white colour, though Roberts says he has known the urate of ammonia in putrid urine to possess a beautiful violet hue. Besides the deposit, there is often on the surface of the urine an iridescent film containing the same elements.

An important character of the mixed phosphates and urates thrown down as the result of the ammoniacal fermentation is their liability to undergo concretion into a mortar-like mass; this forms a large part of many calculi, and it may also collect upon the end of a catheter left in the bladder, and upon the surface of the mucous membrane itself.

It is often of great consequence to determine whether the occurrence of the alkaline fermentation is limited to the urine which has already entered the bladder, or whether the bacteria which produce it have also made their way up into the ureters and into the renal pelves. Sometimes this may be made out by very carefully washing out the vesical cavity, and by drawing off a few minutes later the first few drops of urine that collect. If this is found to be acid the change takes place only in the bladder. How quickly the reaction may be reversed is well shown when there is extroversion of the bladder, so that the orifices of the ureters are exposed to view. In a case of this kind Dr Rees many years ago found ('Croonian Lectures on Calculous Disease,' 1856) that the urine as it flowed out from the orifices possessed its natural amount of acidity; but when reddened litmus paper was applied a quarter of an inch lower down, so as to test the urine after it had passed over that short distance of mucous membrane, its blue colour

was restored. This result, however, he attributed, not to ammoniacal fermentation (for which there was not sufficient time), but to the admixture of alkaline exudation, from the reddened and inflamed surface.

In most cases, before putrefaction of the urine within the bladder begins, cystisis is already present; and even when that is not so, its occurrence is not long delayed. Urine which has become ammoniacal acts as a powerful irritant, though it is not certain whether the inflammation is set up directly by the bacteria, or by the carbonate of ammonia that results from their presence. The exudation of a large quantity of pus follows. This, however, does not retain its usual characters, but is converted by the ammonia into a viscid, semi-transparent, mucoid material, which often blocks the urethra, and causes the patient severe pain. In a vessel, it collects at the bottom, and coheres so firmly that when the urine is poured out it does not separate, but glides away as a gelatinous mass, which hangs from the vessel like a liquid rope. The cystitis is often intense, leading to "diphtheritic" infiltration and sloughing of large tracts of mucous membrane. The inflammatory process often extends along the ureters, and reaches the kidneys, as will be pointed out further on.

In this way ammoniacal fermentation of the urine becomes indirectly the cause of death. It is therefore a matter of the highest importance to prevent its occurrence, and to check it whenever it shows itself. The strictest antiseptic precautions should be observed whenever a catheter is introduced, and this should never be done without necessity, when there is likely to be subsequent difficulty in emptying the bladder. If the change has already taken place before the patient comes under observation, the bladder should be washed out regularly with warm water containing borax and boracic acid. Roberts says that the urine may sometimes be restored to its natural condition by making the patient drink large quantities of fluid at regulated intervals, so as to keep the bladder washed out by the renal secretion. We have often kept the urine sweet and acid, or restored it after it had become ammoniacal, by giving salicylate of soda or benzoate of soda in ten or fifteen grain doses three times a day.

Ammoniacal urine without sepsis.—It would seem that an ammoniacal state of the urine, at the time when it is voided from the bladder, is not in itself a proof of the occurrence of putrefaction in it. At least, Sir William Roberts says that he has observed this in two cases of advanced Bright's disease, without there being any evidence of delay in evacuation, and without any part of the urinary passages being afterwards found inflamed at the autopsy. He does not mention whether bacteria were present. Graves gives two cases, in each of which the urine, although free from any smell of putrescence, contained a large quantity of carbonate of ammonia, even when it was passed soon after being secreted. In one (a case of dropsy) the fluid was tested for urea, which was found absent; the other was a case of fever.

Non-septic bacilluria.—Again, the presence of bacteria in the urine is not in itself proof that the ammoniacal fermentation has taken place, or is about to take place in it. Roberts, in the 'Transactions of the International Medical Congress' held in London in 1881 (p. 157), has related several cases, in which the urine when voided was opalescent, and full of micrococci and moving rods. In two of the cases it had a disagreeable odour. But it was acid in reaction, and showed no greater (and perhaps less) tendency to decomposition than healthy urine. Moreover, in the course of about twenty-

four hours, the bacteria sank slowly to the bottom, leaving the liquid itself clear, and it remained in that condition and retained its acidity for several days afterwards. The organisms seemed to be incapable of multiplying in the urine, even when it was kept at blood-heat. Roberts was therefore inclined to think that their original seat of development was in some part of the mucous membrane of the urinary tract. Some of the patients had had catheters introduced at a former period of their lives; in others this had not been the case. Two only were women, and one of them had no symptoms beyond a slightly increased frequency of micturition, especially at night. The others were men, and they suffered from scalding pain in passing water, with frequent desire to micturate, symptoms which in one instance had lasted thirteen, and in another seven or eight years. In one case there were severe intermittent attacks, as of acute cystitis. The administration of salicylate of soda, in doses of twenty to thirty grains twice or thrice daily, usually proved effectual; the urine became free from bacteria within a few days, and all the symptoms disappeared.

A similar case occurred in the clinical ward of Guy's Hospital a few years ago. A boy who was under the editor's care for another disease, without any symptoms of renal or vesical disorder, passed urine which contained rod-shaped bacteria, when examined immediately after it was voided. The urine was not ammoniacal, and in all other respects was normal.

Sarcinæ.—Several observers have recognised the presence of sarcinæ in the urine, sometimes in sufficient numbers to form a greyish-white amorphous deposit. They resemble those derived from the stomach, except that they are smaller. They may occur in either acid or alkaline urine. The patient is generally troubled with vesical or renal symptoms, but in some instances these have been probably attributable, not so much to the growth of the sarcinæ as to some concurrent disease, such as stricture or enlargement of the prostate. In a case recorded by Munk the sarcinæ were abundant in the summer, but almost wholly disappeared in the winter, notwithstanding that the patient, being paraplegic, was bedridden. Dr Bateman, of Norwich (cited by Dr Beale), met with a case in which the urine on four separate occasions contained sarcinæ for a few days at a time, apparently in connection with dyspepsia.

HÆMOGLOBINURIA.—In some cases in which the urine is reddened by the colouring matter of the blood, no red discs can be found in it. Within the last twenty years this condition has attracted much attention, and has been found to occur in various circumstances. It must be strictly distinguished from hæmaturia, where blood itself is present; this will be considered in the chapters upon calculi, tubercle and cancer of the kidneys, for these diseases are its most frequent cause.

Characters.—Urine containing hæmoglobin varies in its tint in different cases. It is sometimes of a pinkish hue, much more often dark red, chocolate brown, and occasionally almost black. It is often compared to strong tea or to porter. It may be clear and transparent when passed, but as it cools it is apt to throw down a thick sediment of lithates having a chocolate colour. Or the hæmoglobin itself may form a more or less abundant precipitate. With the microscope there is often only a granular *débris* to be seen, but in some cases a few shrivelled or altered blood-discs are visible. And sometimes the hæmoglobin itself appears in the form of rounded reddish-yellow

drops, looking not unlike red discs, but variable in size and sometimes arranged in rows like the beads in a necklace. It may also be moulded into casts of the renal tubules, which have an opaque granular appearance and a reddish-brown colour, but with few, if any, exceptions, there is no true hyaline "cast" of fibrin enclosing the pigment. Sometimes, but very rarely, it assumes the form of crystals. Oxalate of lime crystals have been met with, but their presence was probably altogether accidental.

When urine containing hæmoglobin is heated, it yields a coagulum which has a brownish-red colour, and is described as differing from the ordinary coagulum of albuminuria in floating upon the surface of the fluid, instead of subsiding quickly to the bottom. This coagulum is said to be formed solely by the proteid of hæmoglobin itself, no serum-albumen being present. Cohnheim speaks of a case in which Roux found that there was enough iron in the urine to correspond, according to that view, with all the albumen. If there is any doubt as to the nature of the colouring matter, the various chemical tests of hæmoglobin may be applied. With the spectroscope a perfectly characteristic appearance is obtained. This consists in the presence, not only of the well-known absorption bands of oxyhæmoglobin in the yellow and in the green between the solar lines D and E, but also of a third broad band in the red between the lines C and D but nearer to C. This third band belongs to a modification of hæmoglobin, called methæmoglobin, intermediate in oxydation between oxyhæmoglobin and reduced hæmoglobin. It is probable that methæmoglobin occurs in the blood before its excretion by the kidneys, inasmuch as Marchand has shown that in poisoning by chlorate of potass (which we shall find to be one of the causes of hæmoglobinuria) the blood itself yields a spectrum showing the band between C and D.

Morbid anatomy.—In fatal cases of hæmoglobinuria the kidneys are found of a deep chocolate colour, with radiating striæ of a still darker tint. With the microscope the renal tubes are found to be completely plugged with masses of hæmoglobin. These have also been shown by Dr Bridges Adams to be present in the Malpighian capsules—a point of some importance as showing that the hæmoglobin is excreted through the tufts rather than the epithelium of the tubes. In one instance Hofmeier further noticed that the spleen had a peculiar reddish-brown appearance on section, and that the medulla of the femur in its upper half was brown. Ecchymoses have also been present in the mucous membrane of the stomach and intestines.

Ætiology.—Hæmoglobinuria is probably always the result of a disintegration of some of the red discs within the circulating fluid, or at least of the escape of their hæmoglobin from their "stroma." It is not due to any primary alteration in the structure nor even to perversion of the functions of the kidneys. The causes of the change in the blood are various, so far as we know.

Transfusion of blood from one kind of animal to another—as when, for example, lamb's blood is thrown into the veins of a dog—will produce hæmoglobinuria in the dog; the explanation being apparently that the foreign red discs are broken up by the blood-serum of the animal into which they are introduced. Heat, again, may destroy the vitality of the blood-discs. Cohnheim says that hæmoglobinuria is a common symptom after extensive burns, if they are not too rapidly fatal, though this symptom has attracted less notice from surgeons than from pathologists who have made experiments upon animals.

The affection is said to have been observed in Germany as a result of heat-stroke. It has been recorded by Immermann as a complication of enteric fever during a relapse ('Deutsches Arch.,' xii), by Heubner in a case of scarlet fever on the twentieth day (ibid., xxiii), and by Stolnikow in one of ague ('Petersburg. med. Woch.,' 1880).

Toxic form.—In other cases hæmoglobinuria is produced by the action of some poison absorbed into the blood; among the substances which have been known to cause it, either in animals or in man, may be mentioned hydrochloric acid, sulphuric acid, chlorate of potass, nitro-benzol, naphthol, and carbolic acid. Eitner has recorded a fatal case ('Berl. klin. Woch.,' 1880) in which it was set up by the inunction of pyrogallic acid into the skin of a patient affected with severe psoriasis.

In several instances it has been due to the entrance of arseniuretted hydrogen gas into the air-passages.

Eitner has recorded, in the same communication, four cases due to this poison, in which the sufferers were a professor of physics and three of his students. The professor had two attacks of hæmoglobinuria, separated by an interval of some days, and resembling the paroxysmal form of the affection in all respects except perhaps in having a rather longer duration. It was not until other persons were found to have suffered from like symptoms that their real nature was suspected. He and his pupils had, in fact, been repeating Tyndall's experiment of inhaling hydrogen gas, for the purpose of showing that the pitch of the voice becomes altered by it; and the zinc used in generating the hydrogen was impregnated with arsenic.

From a clinical point of view, chlorate of potass far surpasses in importance all the other toxic causes of the affection. Hofmeier collected ('Deutsche med. Woch.,' 1880) no fewer than twenty-seven cases, all but four of which proved fatal. Some of the patients took the salt by mistake for sulphate of soda or for some other saline aperient. But for most of them the chlorate of potash was prescribed, and the mischief arose either from the dose being unduly large or too frequently repeated, or else from their swallowing large quantities of a solution intended only as a gargle. In young children it would appear that from one to two drachms in the course of twenty-four hours is a dangerous quantity; in adults perhaps from two or three drachms upwards. In many instances the disease for which the chlorate of potass has been ordered has been diphtheria, and one cannot help fearing that in many other cases the salt may have produced like effects without their real cause having been suspected, the state of the urine having been attributed to diphtheritic nephritis, and the severe constitutional symptoms regarded as indicative of the "collapse" which sometimes proves so rapidly fatal in diphtheria.

The symptoms of toxic hæmoglobinuria differ in intensity, but seem always to belong to a common type. In the most marked and severe cases the patient is perhaps seized with a rigor; vomiting and diarrhœa then set in; he becomes collapsed and cyanosed, falls into a state of stupor, and dies.

Infantile hæmoglobinuria.—Spontaneous hæmoglobinuria occurs in circumstances which vary somewhat in different cases. Winckel has related ('Deutsche med. Woch.,' 1879) a most remarkable outbreak, which occurred in 1879, in the lying-in hospital at Dresden, where, between March 20th and April 29th, twenty-four newly-born infants were attacked by it, of whom twenty-three died. The symptoms were in every instance similar:

the child, generally about the fourth day after its birth, became suddenly cyanosed and collapsed, with cold extremities; and it usually succumbed within thirty-six hours. There was seldom any diarrhœa or vomiting, but the skin had in many cases an icteric tinge. The respiration and the pulse were very rapid; the temperature in the rectum was 90° or 100°. At many points the superficial veins became visible as dark lines; when they were incised no blood escaped, but on pressure a brownish-black fluid of syrupy consistence could be squeezed out. The urine was of a brown colour, and exhibited the usual characters of hæmoglobinuria. Death was preceded by convulsions. On *post-mortem* examination the kidneys were dark brown and their papillæ showed plugs of hæmoglobin. The chief other morbid appearance was an enlargement of the mesenteric lymph-glands and of the spleen, the latter being tough and of a brownish-red colour. Every effort was made to discover the cause of this affection, but without success. Winckel described it as *Cyanosis infantilis icterica perniciosa cum hæmoglobinuria.* There can be little doubt that its essential character was an intense and rapid disintegration of blood-discs within the circulating fluid. But why this should have occurred in a number of young infants in succession—themselves apparently healthy at birth, the offspring of healthy mothers, and in a hospital where no like disease had been observed before—remains a mystery.

Paroxysmal hæmoglobinuria.—In marked contrast with this fatal infantile form of hæmoglobinuria is one to which adults are chiefly liable, and which repeats itself over and over again without danger to life or severe disturbance of health. It was first described by Dr George Harley, in the 'Med.-Chir. Trans.' for 1865 as "intermittent hæmaturia;" but a probable case may be found recorded by Dressler in 'Virchow's Archiv' for 1854. A case was described by Sir William Gull in the 'Guy's Hospital Reports' for 1866, as "intermittent hæmatinuria," by which name it has been since commonly known; but the best name for it appears to be "paroxysmal hæmoglobinuria," used by Dr Pavy in 1866 and 1868. Dr Hassal described it in 1865 ('Lancet,' i, 568) as "intermittent or winter hæmaturia," and Lichtheim published three cases in 1878 (No. 134 in 'Volkmann's Samm-lung') under the title "periodical hæmoglobinuria."

Ætiology.—In some few instances no cause can be discovered. Two such cases occurred in our wards during the height of summer, in June, 1876, and in August, 1880. In neither case were any subjective sensations, except pain in the loins, associated with the discharge of urine, which was almost black with much hæmoglobin. Fleischer has recorded ('Berlin klin. Woch.,' 1881) an instance in which a soldier was attacked every time he marched for an hour or two, although no other kinds of bodily exercise had a similar effect.

But in the great majority of cases the affection is due to one particular exciting cause, namely, *cold.* Sometimes the degree of cold that causes an attack (especially the first attack) is notably excessive, or, at any rate, far beyond that to which the individual is accustomed. A London physician, a friend of the author, observed hæmoglobinuria for the first time when he was one day fishing in Scotland in a biting wind, and his next seizure occurred some months later while he was skating without a great coat. But he subsequently became liable to the affection when the provocation appeared inadequate to produce such a result, as, for example, after standing

four or five minutes at a railway station on a foggy morning, or riding two or three miles in a hansom cab. In some instances it is stated that all but the early attacks have been altogether independent of cold. Thus a patient of Sir William Roberts said that he was just as bad in the summer as in the winter. As a rule, however, the affection ceases entirely during the warm part of the year, returning in the cold season, perhaps for many years in succession. That this is entirely a question of temperature has been shown by Rosenbach, who, by means of a cold foot-bath, brought on an attack during the summer in a person liable to the disease. Roberts relates two cases, in each of which there were as many as three daily attacks for days together. But the rule is that they recur at much longer intervals and quite irregularly. The part of the day at which they are most apt to take place is the *morning*. This circumstance is doubtless related to the fact that the temperature of the body is naturally lower then than it is in the afternoon and in the evening. It is especially when the patient is exposed to cold directly after breakfast, before the meal has been digested, that hæmoglobinuria is apt to show itself; and a cupful of warm beef-tea on first waking may prove an effectual preventive. Mental or bodily exhaustion, as from sexual intercourse, or from study late at night, favours the occurrence of an attack. A like influence appears to be exerted by the free use of wine at dinner, probably from the recoil after its stimulant action as well as from its relaxing the cutaneous vessels. Anxiety and nervousness also play a part in the ætiology of the affection. It is seen particularly in persons whose hands and fingers turn cold on excitement, and in those who are liable to *digiti mortui*. The previous occurrence of *ague* has been noted in some instances, and it seems not impossible that this may sometimes be a more or less direct cause of hæmoglobinuria, just as it is of splenic leuchæmia. The only doubt is whether the peculiar appearance of the urine may not have been overlooked during the early attacks, and whether they may not therefore have been wrongly ascribed to the influence of malaria, especially in countries where ague is prevalent. This doubt would of course be set at rest if there were a clear history of tertian or quartan periodicity. Syphilis was present in a case observed by Ehrlich; and a patient who was under the author's care in the clinical ward of Guy's Hospital in 1882, and who did not improve at all under the usual remedies, was subsequently cured by Dr Moxon with mercurial treatment. In that instance the spleen was much enlarged.

The disease is very much more common in men than in women. It is most apt to occur in young adults, and continues up to the age of forty or fifty, but it is said to have been observed in a child only two years old. Dr Druitt was fifty-one or fifty-two when he was first attacked.

Seven cases of this remarkable disorder have been under the editor's care. They all occurred in men; the youngest fourteen years old, and the eldest above sixty, the remainder between nineteen and thirty-three.

Two were pretty clearly of malarial origin, associated in one patient with dysentery during the Zulu campaign, and benefited or cured with quinine; two were complicated by gangrene of the ears or fingers and toes; and one case which occurred in a schoolboy three times at intervals, the first after bathing, was marked by high fever.

The symptoms of paroxysmal hæmoglobinuria vary in different cases. One of the most constant is a feeling of languor and weariness, with a dis-

position to yawn again and again. With this there may be a feeling of chilliness, so that the patient is inclined to huddle up to the fire. The fingers and the toes—some or all of them—may turn white and dead. A most graphic account of the disease was given by the late Dr Druitt in the 'Med. Times and Gazette' for 1873; it is now no secret that he was himself the patient. He describes his palms and his soles as becoming "cold, wet, blue and cramped, like those of a cholera patient." At other times he felt numbness of the right foot and of the left hand, without coldness; or his nose, or some part of his cheek, would become first pale, then red, then purplish, and at last quite black. In several instances the ears not only turned livid during the seizures, but failed to regain their natural appearance afterwards, so that a reddish-brown eschar formed along the edge of the helix, leading to a permanent loss of its substance.

While these symptoms are developing themselves, and until they have entirely passed off and been succeeded by a feeling of warmth and comfort over the whole body, there may be nothing to show that the secretion of urine is other than normal. There is often no desire to micturate. In a case which the author had many opportunities of watching, it has sometimes been only after the lapse of several hours that any water has been passed, when the patient has almost forgotten that he had been chilly; so that he has been quite surprised to find it like chocolate or porter. The urine, beside methæmoglobin, often contains a few red discs, more abundant leucocytes, and sometimes crystals of calcic oxalate. But in other cases the bladder is irritable, and Dr Druitt says that on some occasions he suffered great pain in the bladder, and was obliged every half hour to void urine, which then was of a bright scarlet colour. Sometimes, but not always, there is pain in the back, or radiatiag across the abdomen or down the thighs. Retraction of the testes has also been recorded.

The bodily temperature during a seizure of hæmoglobinuria is, as a rule, normal. Dr Druitt says that during the course of his illness (which had then lasted six years) he had severe attacks of remittent pyrexia, in which the thermometer would rise to about 103° in the evening, and fall to 100° in the morning; during these periods jaundice was always present, but the urine never contained any blood-colouring matter. His pulse generally fell to 55 or 50 when the hæmoglobinuria was about to occur. In one of the editor's cases, the patient, a robust youth of nineteen, had pyrexia reaching 103° on one, and exceeding 104° on another occasion, with a pulse of 90 to 100 during the attacks.

One of the most remarkable features of the disease is the rapidity with which, after a seizure, the urine regains its normal characters. The patient may once or twice, or oftener, have voided a fluid like porter; and that which he passes an hour or two later is perfectly clear and pale. Even in Roberts's two cases, in which there were two or three attacks in the course of a single day, colourless urine was secreted in the intervals. It is also curious how completely the appetite is unaffected; a patient who had a well-marked attack in the morning may at one o'clock eat a hearty dinner as usual, sometimes even before he has discharged from his bladder the dark urine formed during the attack.

Pathology.—It has now been ascertained that the starting-point of paroxysmal hæmoglobinuria (as we found in the tonic form of the affection) is disintegration of red discs within the circulating fluid. Microscopical examination of the blood does not, indeed, show any marked change in it, as

the writer and several other observers have found. Küssner, however, took blood with a cupping glass ('Deutsche med. Woch.,' 1879) from a patient on six occasions during a seizure, and each time found that the serum was of a ruby-red colour, whereas at other periods it had the normal yellowish appearance. A still more striking experiment has since been performed by Ehrlich (*ibid.*, 1881). Having under observation a woman who was liable to the disease, he bound an elastic ligature round one of her fingers, and placed it for a quarter of an hour in ice-cold water, and afterwards for the same length of time in tepid water. Such a procedure on a healthy person produces no change in the blood, but in the patient in question it caused the red discs to break down in considerable numbers. When a drop of blood from the finger was placed in a capillary tube and allowed to coagulate there, the serum was distinctly seen to be reddened; and with the microscope the blood was found to contain "phantom discs"—stromata or *œcoids* of discs that had more or less completely lost their hæmoglobin—though there were also many normal corpuscles, as well as pœcilocytes and microcytes. The disease therefore seems to originate in an undue sensitiveness to cold on the part of certain of the red blood-discs.

In some patients, however, there may be observed a circumstance which at first sight seems inconsistent with this view of the disease. It is that attacks of chilliness which fall slightly short of the degree of severity necessary to bring about an excretion of hæmoglobin render the urine albuminous. This is a fact which we have verified on many occasions, the patients being perfectly free at all other times from any indication of Bright's disease. Probably the true explanation is that when the disintegration of red discs occurs only to a moderate extent all the hæmoglobin which is set free splits up into globulin and hæmatin. We may then suppose the albumen (or globulin) to be incapable of remaining as a constituent of the serum, and to be excreted as such by the kidneys. Indeed, it is certain that even in ordinary cases of hæmoglobinuria a part of the hæmoglobin is decomposed, so as to form a pigment resembling bilirubin, if not altogether identical with it. The proof of this is the sallow bilious appearance which is invariably presented by patients who frequently suffer from the disease, and which is sometimes apparent during or after a single severe attack.

Prognosis.—Paroxysmal hæmoglobinuria has never been known to destroy life, nor does it appear to entail very serious consequences, though one can hardly doubt that if the attacks were allowed to recur frequently, and for a great length of time, the patient's health would ultimately break down. The author once saw chronic Bright's disease in a man who had previously suffered from hæmoglobinuria, and had lost the margins of his ears from it. The attacks may continue for many years in succession, or they may cease after a few weeks or months. Dr Druitt had suffered for six years when he published the account of his case; in another recorded instance the disease ran on for eleven years.

Treatment.—During an attack the essential requisite is warmth. If necessary the patient may be put to bed. Probably the best thing to drink is a basin of hot soup. When there is severe pain in the back, it may be relieved by the application of mustard, or by dry cupping, or (as Dr Druitt found) by the internal administration of extract of hyoscyamus.

But the important object is to prevent the recurrence of the disease. The utmost care should be taken to avoid exposure to cold in the early part of the day, and in such circumstances as may be shown by experience

to be injurious in the particular case under observation. The meals should be so arranged that there may be no deficiency of already digested food in the body at the time when a cold journey is to be made, or when any unaccustomed task is to be gone through that may cause nervousness and anxiety. The clothing must be warm; flannel under-clothing, fur-lined boots and gloves, wash-leather waistcoats, and cork soles are all useful.

The question of removal to a hot climate during the winter should in severe cases be seriously considered; Dr Druitt himself went to Madras, where he escaped the disease almost entirely. During the summer a bracing air is probably advisable.

Throughout the twenty-four hours the habits should be so arranged that the body shall become as little exhausted as possible. The patient should not spend his evenings in heated rooms, nor should he devote them to arduous mental work, but should go to bed early. He should have nutritious food in the latter part of the day, but little or no alcohol; for its effect is an immediate stimulation that rapidly passes off.

The one medicine that seems to have a marked effect in warding off the attacks is *quinine*. It must be given in full doses. It sometimes proves perfectly successful, so that the patient becomes again able to live his usual life without fear of the disease. But in severe cases, like that of Dr Druitt, no permanent benefit can be obtained by it, even when it is used in such quantities as to cause singing in the ears and other disagreeable effects. Salicine, the tincture of iron, and arsenic, may each be of service in their turn. Chloride of ammonium is said to have done good in one instance. If syphilis is present, iodide of potassium or the bichloride of mercury will sometimes work wonders.

Raynaud's disease.—There can be little doubt that this singular disorder, in its most characteristic idiopathic form, is closely related to the local asphyxia with symmetrical gangrene of the extremities, which was described by the late Dr Raynaud in 1862, and which, in its most severe forms, is known by his name.

The age of the patients, the coldness of the extremities, and the mortification of the ears, or fingers, or toes, are very suggestive of this relation; and in typical cases of Raynaud's disease hæmaturia or hæmatinuria has been frequently observed. Curiously enough, in the twenty-five cases on which his monograph is based (five of his own and twenty collected from previous writers), no mention is made of the state of the urine. There are, however, points of difference: of Raynaud's twenty-five cases, twenty occurred in women and only five in men. Some of the most severe cases of hæmoglobinuria show no tendency to gangrene, and the patient's circulation is perfect in the intervals between the attacks. Some of Raynaud's cases are not accompanied by any change in the urine, or the change is to true hæmaturia. At present it is perhaps best to keep together the cases of hæmoglobinuria (and even of hæmaturia) which are decidedly paroxysmal and unaccompanied by gangrene, and to reserve the title of Raynaud's disease for the continuous and severe cases described by him. See on this subject Dr Southey's papers in St 'Bartholomew's Hospital Reports,' vol. xvi, and in the Clinical Society's 'Transactions' for 1883; also Dr Thos. Barlow's communication in the same volume, with the sequel to his cases in 1885 (vol. xviii, p. 307), and Dr T. C. Fox's two cases (ibid., p. 300).

BRIGHT'S DISEASE

History—The two more frequent forms—Additional varieties—Common characters of the disease—Albuminuria: its detection and estimation, pathology, and significance—Casts: their structure, varieties, and meaning—Dropsy: its pathology, its primary and secondary form—Retinitis—Serous and other inflammations—Changes in the heart and arteries: the renal pulse, hæmorrhage—Uræmia: eclampsia, coma, amaurosis, &c.; vomiting; theory of uræmia.
Parenchymatous Nephritis—causes—anatomy—symptoms—event.
Lardaceous Nephritis—chemistry—anatomy—symptoms—diagnosis—causes—prevention.
Cirrhosis of the Kidney—pathology—causes—anatomy—symptoms and clinical aspects—Consecutive renal cirrhosis—its relation to local disease of the bladder, urethra, and uterus—Microcystic degeneration—Hypertrophic cystic kidneys.
General prognosis and treatment of Bright's disease.

In 1827 Dr Richard Bright published, in his 'Reports of Medical Cases,' the fact that in many cases of dropsy there are well-marked lesions of the kidneys, and that the urine is albuminous. The novelty of the discovery lay in his connecting the clinical symptoms of dropsy and albuminuria with the anatomical fact of renal disease. That dropsy is often attended with the presence of serum in the urine had been pointed out a short time before by Dr Wells (the author of the 'Essay on Dew'), by Dr Blackall, of Exeter, and Dr Osborne, of Dublin; in the previous century it had been noticed by Cotugno and by Cruikshank. But notwithstanding that Wells and Blackall each made autopsies in which they found the kidneys "remarkably hard," they both regarded the presence of disease in these organs as an accident, Blackall's idea being that the reason why serum was excreted in the urine must be that it was in a vitiated state from some cause or other, possibly from having already formed part of the dropsical fluid in one of the serous cavities or elsewhere, and having afterwards been reabsorbed into the blood. To Bright belongs the full credit of first showing the real and constant relation of disease of the kidneys both to dropsy and to albuminuria, and his name is therefore most justly associated with it throughout the world.

After the publication of his first cases and drawings, two wards at Guy's Hospital were devoted to further investigations into the new disease, and Dr Bright, with the help of Dr Barlow and Dr Owen Rees, made the further observations which were printed and illustrated in the 1st volume of the 'Guy's Hospital Reports,' and in the 5th of the 2nd series.

Its divisions.—A difficulty, however, which has stood in the way of all subsequent writers is that the lesions of the kidneys described by Bright vary greatly in different cases. He himself admitted three forms of the affection, leaving it an open question whether or not they should be regarded as separate diseases. In 1851, Professor Frerichs, of Berlin, propounded the doctrine that the diverse appearances presented by the kidneys belong to the succes-

sive stages of a single morbid process. This view, however, was refuted two years later by Dr Wilks, who showed in a paper in the 'Guy's Hospital Reports' (2nd series, vol. viii) that, under the name of Bright's disease, there are included at least two independent affections differing in their causes, in their mode of onset, their symptoms, and their course—"the large white kidney with considerable dropsy," and "the hard contracted kidney, often destitute of symptoms"—and added a summary of twenty-three cases of the former, and thirty-three of the latter affection.

To make clear the argument upon which he mainly relied, we must remember that, although Bright laid so much stress upon dropsy as an effect of the renal disease which he described, he knew from the first that in many cases but slight dropsy was present. Now, Wilks pointed out that since this symptom is associated in a special manner with the "large white kidney," one ought, if that were an early stage of the "small red kidney," to obtain a definite history of there having been at a former time an attack of dropsy in each case in which the small red kidney was found after death. Frerichs, however, had not brought forward a single instance in which there had been this sequence of events, nor were any furnished by Wilks's own large experience. The statement that the large white kidney does not pass on into the small red kidney may perhaps be put too absolutely; but in the main it is undoubtedly correct. Moreover, in the *post-mortem* room it is not uncommon to find in the bodies of those who have died of various diseases kidneys showing all the stages of the morbid process by which they gradually became shrunken; and this certainly does not involve the occurrence of any enlargement, nor is it attended with any change of colour. As for the other points of distinction pointed out by Wilks, it may be briefly stated that, whereas the large white kidney often results from scarlet fever, or from exposure to cold, the small red kidney is not traceable to either of these causes, but in many cases to gout or to lead-poisoning; that whereas the former occurs in children and young adults, the latter is seldom seen in persons under five-and-thirty, and is not frequent before the age of fifty; that the former is often rapid in its onset and acute in its course, while the latter always begins insidiously, and is very slow in development; and lastly, that although either may be attended with changes in the heart and arteries, such changes are far less marked in the former than in the latter kind of Bright's disease, which often appears clinically under the mask of cardiac symptoms, or of apoplexy due to rupture of an artery in the brain.

It would, of course, add to the force of these considerations if it could be shown that two distinct pathological processes are concerned in the production of the two forms of Bright's disease, or even that the same process attacked in the one form one anatomical element of the kidneys, in the other form another. But the pathological process is, in both cases, inflammation; and, in both, it always falls short of suppuration. Many years ago, Virchow, in his 'Cellular Pathology,' suggested the distinction that in the large white kidney the tubular epithelium, in the small red kidney the interstitial tissue, was mainly affected. Recent observations, however, have shown that this distinction is very far from being absolute; and therefore the two names proposed by Virchow, "parenchymatous nephritis" and "interstitial nephritis," can no longer be regarded as perfectly applicable. In both forms the tubes are affected; in both there is intertubular exudation; in both the cortex is the seat of the lesion: the distinctions are rather of the origin, course, and result than of the nature of the process.

Still the tubular parenchyma of the cortex is swollen in the large smooth kidney, whereas in the small rough kidney it is contracted and atrophied as in other chronic interstitial inflammations. Both clinically and pathologically we must admit the fundamental unity of Bright's disease, together with the broad and important difference between the two forms just stated. As physicians, we are quite justified in regarding them as distinct affections when we find that they differ in their causes, in their symptoms, and in their clinical course. Some German writers describe separately "acute Bright's disease," "chronic Bright's disease," and "contracted or granular atrophy of the kidneys." Such a division, however, keeps apart cases that should be brought together, and it brings together cases that should be kept apart. An acute renal affection may be the result of scarlet fever, or of cold, or of pregnancy; but if it does not prove fatal or get well, it soon becomes chronic, and may ultimately pass into a stage of contraction and of atrophy. All such cases should be held to belong to a single form of Bright's disease; and since an affection of the tubal epithelium is a more or less constant feature, we may call them *tubular*, or *parenchymatous nephritis*. On the other hand, Ernst Wagner's "contracted or granular atrophy" includes two sets of cases that are essentially distinct; one is the advanced stage of the affection just mentioned, the other is the original chronic affection which produces the "small red kidney." It is to the latter process alone that the term *interstitial nephritis* should be applied, or its synonym, *cirrhosis of the kidney*.

These two chief forms of Bright's disease, however, are by no means the only ones. That in some cases the primary lesion is a *lardaceous degeneration* in the arterioles and in the glomeruli of the kidney has long been known, and of late it has become apparent that such cases are far more numerous than was formerly suspected. It is true that the lardaceous affection always tends to become complicated sooner or later with parenchymatous or with interstitial lesions, or with both. But inasmuch as its causes differ from those of Bright's disease in general, and as there are also differences in its symptoms and course, all authorities are agreed to keep it apart.

There are two other subordinate forms; one is the affection commonly known as *cystic disease of the kidneys*, the other, which has only recently been recognised, is in its origin secondary to lesions of the renal pelvis or of the lower urinary passages, so that it may be termed "*consecutive Bright's disease*." Both are varieties of chronic interstitial degeneration.

It might be thought that the recognition of so many independent forms of Bright's disease would render it advisable to drop the common designation. But, first, many important characters belong to them all alike. Secondly, in clinical practice one is not infrequently in doubt as to which form is present. Thirdly, we often meet with transitional forms:—the acute tubal nephritis is passing on to, but has not reached, the chronic stage of the large white kidney, as red hepatisation passes into grey; the granular atrophic organ is overtaken by an acute tubal inflammation, as a bronchial catarrh supervenes on chronic cirrhosis of the lung; lardaceous disease usually complicates tubal nephritis, or is complicated by it, but sometimes it is seen in a granular contracted kidney.

Certainly a satisfactory definition of Bright's disease is very difficult if not impossible. It should not include symptomatic albuminuria, such as accompanies certain forms of heart disease, cholera, erysipelas, or diphtheria. It should not include cases in which a transitory albuminuria occurs without

there being reason to suppose any organic change in the kidneys. And, lastly, it should not include any suppurative lesions of the kidneys. Taking these reservations into account, we may perhaps say that by Bright's disease is meant diffused non-suppurative primary nephritis, attended with albuminuria; and its divisions we will treat as follows:

A. *Acute and chronic parenchymatous nephritis.*—This, as a rule, is attended with general dropsy. The onset is sudden, when scarlet fever or cold is the cause, but in other cases it may be gradual. In the acute stage the kidneys are swollen and red; when the disease has become chronic they are whitish yellow, and sometimes very large. Finally, they may shrink and become granular. The urine is at first dense, high coloured, scanty, containing much albumen, tube-casts, and often blood. Later on, it may be pale, with a variable amount of albumen. Retinitis and uræmia are of frequent occurrence. In advanced cases the heart becomes hypertrophied, and the systemic arteries are thickened.

B. *Lardaceous disease of the kidneys.*—This is caused by protracted suppuration or by syphilis. The kidneys give the characteristic reaction with iodine. Gradually they become very large, pale-yellow, and waxy looking, as the result of the supervention of parenchymatous nephritis. Finally, they may shrink and become granular. The urine is excessive in quantity, pale, and contains much albumen. But when there is much nephritis it may be scanty and high coloured. General dropsy is frequent. Cardio-vascular changes, retinitis, and uræmia are seldom observed.

C. *Cirrhosis of the kidneys, or red granular atrophy.*—This is a slow and insidious affection, of which the chief known causes are gout and lead-poisoning. It is very rare in early life, but begins to occur towards forty years of age, and beyond this it is frequent up to seventy. It gradually destroys the renal cortex until this may not be more than a line in thickness; the surface of the organ remains of a red colour, but it becomes very uneven and granular. The urine is abundant, clear, pale, of low specific gravity; it contains only a small quantity of albumen, or there may be none at all for days together. Marked general dropsy occurs only when the affection becomes complicated with parenchymatous nephritis. Cardio-vascular changes are constantly developed, and reach an extreme degree. In many cases the patient dies with the symptoms of heart disease, including obstructive dropsy, which affects the dependent parts of the body. Cerebral hæmorrhage is another frequent cause of death.

(α) *Consecutive Bright's disease.*—This is seen as the result of such affections as stricture of the urethra, stone in the bladder, prolapsus uteri, compression of the ureters by an abdominal tumour, calculous pyelitis, and scrofulous disease of the kidneys. The kidneys become tough, hard, and whitish; they may be either of normal size or shrunken, either smooth on the surface or puckered by cicatrices, or granular. The general symptoms and the characters of the urine are those of renal cirrhosis.

(β) *Cystic disease of the kidneys.*—The main peculiarity of this affection is the presence of innumerable cysts of various sizes. Commonly, the kidneys are small and contracted, but sometimes they are sufficiently large to be felt as abdominal tumours during life. In its clinical features it resembles renal cirrhosis.

Before entering upon a detailed description of these different forms of Bright's disease we must consider certain symptoms and effects which belong

in common to several, if not to all of them. These are albumen and tube-casts in the urine, dropsy, retinitis, secondary inflammations, cardio-vascular changes, hæmorrhages, and uræmia.

1. *Albuminuria.*—The coagulable substance, albumen, which is found in the urine in Bright's disease is, strictly speaking, a mixture of two albuminous compounds, "serum-albumin" and serum-globulin or "paraglobulin," both of which are naturally present in the liquor sanguinis. They can best be separated by Hammarsten's process of saturating with crystallised sulphate of magnesia; this precipitates the paraglobulin, but leaves the serum-albumin in solution. Estelle found ('Revue Mensuelle,' 1880) that in certain cases of albuminuria in which he investigated the point, sometimes the whole of the so-called albumen, and, as a rule, all but one third of it, was really paraglobulin. The distinction seems, however, to be devoid of practical importance; both substances react in precisely the same manner to the usual tests, and have the same clinical significance.* Peptones are occasionally present but appear to be of no diagnostic importance.

Tests.—The oldest method of detecting albuminuria is by *heat.* The best way of applying it is to fill two thirds of a test-tube with urine and then to hold it near the bottom, while one gently warms the upper part of the liquid over a spirit lamp. When the urine is acid, any albumen that may be present becomes precipitated. Sir William Roberts says that at what temperature this occurs depends upon the amount of albumen; if it is very small, no change is perceived until the boiling-point is reached; if it is large, an opaque coagulum forms at a much lower temperature.

For urine which is turbid with urates this test is peculiarly applicable. The existence of turbidity from this cause shows that there is sufficient acidity. The first effect of the heat is to redissolve the urates, and to make the fluid transparent; presently the albumen begins to appear and renders it again cloudy. In a long column of fluid all three conditions may be seen at the same time; at the bottom, a part which is cold and turbid; above it, one which is warm and clear; still higher, one which is hot and opaque. But in alkaline urine heat may produce no change, although albumen is present. There is thus a risk of overlooking albuminuria by trusting to this test. It is true that one may avoid the risk by adding a very little acetic acid—one drop, just enough to acidify the urine—before beginning to warm it. But this introduces a new source of error; for if the amount of acetic acid be at all excessive, it may itself prevent the heat from throwing down any small quantity of albumen that the urine may contain, unless indeed the urine is also rich in salts, the presence of which is found materially to increase the coagulability of albumen in such circumstances. The safe way, therefore, is, besides the acetic acid, to mix with the urine about one sixth of its bulk of a concentrated solution of common salt, or of magnesian or sodic sulphate. On heating the liquid after treating it in this manner, the albumen is seen to come down. Salkowski says that the test so applied is not only absolutely conclusive but unsurpassed by any other in delicacy.

If the urine is neutral or faintly acid, the application of heat frequently produces an opacity which looks exactly like that due to albumen, but

* See Dr Saundby's papers ('Birmingham Medical Review,' October, 1879, and 'Brit. Med. Journ.,' June 5th, 1880).

which really consists of a precipitate of phosphate of lime. By adding a little acid, one can of course redissolve the phosphatic precipitate, and so prevent it from being mistaken for albumen. And it may be laid down as a rule of the first importance that, unless the urine has been ascertained to be strongly acid, the presence of albumen in it must never be affirmed as the result of the application of heat alone, without the subsequent addition of an acid. But there is always the possibility that urine which throws down the phosphate of lime when warmed may also contain albumen. Now, if acetic acid be the acid employed to redissolve the phosphate it is obvious from what has already been stated that it must be uncertain whether the albumen will show itself or not. If nitric acid be used, coagulation is perhaps sure to occur; but, on the other hand, most observers are agreed that it is unadvisable, with the object of bringing down albumen, to add nitric acid to urine that has just been boiled, because this acid is apt to induce in the hot liquid other changes, the nature of which is not well understood.

A second extremely delicate test for albumen in urine is the *nitric acid* test. When urine is opaque with urates they should be first redissolved by warming the test-tube, until its contents are raised to about blood-heat. The best way to use nitric acid is to put a moderate quantity first into the tube, to hold it in a very slanting position, and then to let the urine slide gently down its side until it floats on its surface. If no albumen is present the two fluids are separated by a more or less deeply coloured layer, from oxidation of chromogen (p. 568). If there is albumen it forms an opalescent zone, of greater or less thickness, at the line where the acid and the urine meet. When its amount is exceedingly small, the zone may appear only after the interval of a minute or two. It is made more conspicuous by holding up the test-tube against a dark background, as, for instance, the sleeve of one's coat. Employed in this way, nitric acid is on the whole the most delicate and the most certain of all tests for albumen.*

In urine of high specific gravity lithates are sometimes precipitated by the acid; but they first appear, not at the line of junction of the two fluids, but near the surface of the urine, the turbidity gradually spreading downwards. If there is any doubt, it may be removed by gently warming the test-tube, when urates will at once disappear. In concentrated urine nitrate of urea will crystallise out, but this takes time and is quite unlike the white cloud of albumen. In the urine of patients who are taking copaiba or cubebs a resinous substance is excreted which is precipitated by nitric acid, but not in so well-defined a zone. The application of heat diminishes the opacity from this cause, and the addition of alcohol entirely removes it.†

Many other tests for albumen are known besides the two already given. Acetic or citric acid with *potassium-ferrocyanide*, is a convenient one and free from fallacies, but less delicate than either heat or nitric acid. Dr Pavy has invented pellets of citric acid and the ferrocyanide which form the best portable test.

Dr George Johnson has strongly advocated the use of *picric acid*. One

* Sir Wm. Roberts, however, has shown that the readiness with which albumen is precipitated by nitric acid is to some extent affected by the presence of other dissolved matters. The proof of this is that if two samples of the same albuminous urine be diluted, the one with successive quantities of pure water, the other with the same quantities of healthy urine, the former continues to yield an opaque zone with nitric acid, after the latter has ceased to give any sign of the presence of albumen.

† I remember a man under treatment for gonorrhœa, who came out with the copaiba rash, and was supposed to have scarlet fever with his urine full of albumen!—C. H. F.

advantage which it possesses over nitric acid is that it can be carried about without the risk of its giving off fumes by which other apparatus may be destroyed. On the other hand, picric acid, like heat, fails to precipitate albumen in alkaline urine. Like nitric acid it redissolves the cloud when only a trace of albumen is present, and it may precipitate lithates or lithic acid. It precipitates peptones (which are redissolved by heat) and it causes a cloud in the urine of persons taking quinine in full doses, also cleared by heat.

Dr Oliver, of Harrogate, has brought out a series of paper slips, saturated with picric acid, potassio-mercuric iodide, tungstate of soda, and other solutions, which are certainly as portable as could be wished.

Peptones, including propeptone or hemi-albuminose, a compound intermediate between peptone and albumen, are not precipitated by heat; and nitric acid has no effect on peptone, but it at first precipitates propeptone, which, however, is afterwards redissolved by an excess of the acid, with the production of a yellow colour. Very little seems to be known with regard to the conditions under which peptones or syntonin are found in the urine; but their presence is, at any rate, not an indication of Bright's disease, so that it is important not to confound them with albumen.

Tincture of galls has long been known as a precipitant of proteids in urine. So is a solution of mercuric iodide in iodide of potassium, but this is too sensitive, for it precipitates not only peptones and other proteids but also mucus.*

Quantitative estimate.—To determine with absolute accuracy the amount of albumen in the urine takes up a great deal of time; it has to be precipitated from a known bulk of the fluid, washed, dried, and weighed. In clinical practice, however, there is no sufficient object to be gained by this troublesome procedure. Sir William Roberts in 1876 proposed a method which is far easier, and which appears to yield sufficiently satisfactory results. It consists in diluting the urine with water until it almost ceases to give a reaction with nitric acid, the point fixed being that at which the opalescent zone at the junction of the two liquids begins to be visible between thirty and forty-five seconds after the addition of the acid to the urine. To calculate the number of grains of albumen per fluid-ounce of urine all that is necessary is to multiply the figure 0·0034 by the number of dilutions with an equal bulk of water that the urine has undergone.

An easier plan, but one that yields only comparative results, is to take a column of urine of definite depth in a test-tube, and after precipitating all the albumen in it with heat or with nitric acid to let it stand until the coagulum has sunk to the bottom, forming a layer the depth of which can be expressed as a fraction of that of the urine, a half, or a quarter, or one sixth, as the case may be. Vogel, however, found that the space occupied by the same quantity of albumen might vary widely according as it happened to be thrown down in larger or smaller masses; and it is also influenced by the specific gravity of the urine, the range of error from these causes being as much as from 30 to 50 per cent.

The actual weight of albumen contained in the most bulky coagulum is but small. Accurate analysis seldom gives more than 5 per cent., even when the urine becomes solid when boiled.

* With respect to the relative merits of tests for albumen in the urine, including his own, of acidulated brine, see Sir William Roberts's valuable criticism in the 'Discussion on Albuminuria,' at Glasgow, in 1884, p. 16.

Theory of albuminuria.—In endeavouring to understand why albuminuria should occur, whether in Bright's disease or under other circumstances, it is necessary to begin by considering how it is that the renal secretion normally contains no albumen. What prevents its escaping from the blood with the water, urea, and salts, which are drained off through the glomeruli? Only one answer to this question seems possible, namely, that it is kept back by the epithelial layer which covers the capillary tufts; and, as Cohnheim remarks, it is interesting to notice that the vessels of the choroid plexuses, which also yield a non-albuminous fluid, are the only ones that have a similar investment. At one time, indeed, a theory was current, according to which albumen was supposed to be present in the transudation from the glomeruli, but to be taken up again and restored to the blood by the epithelial cells of the convoluted tubes; but that notion has been refuted by the observations of Posner ('Virch. Arch.,' 1880).

That, when albumen appears in the urine it escapes through the glomeruli, is rendered probable by Nussbaum's experiments on frogs ('Arch. f. Phys.,' 1878), in which animals these structures have an arterial supply distinct from that of the renal tubes; he ligatured the glomerular arteries and found that after this operation egg-albumen, injected into the stomach or into the blood, no longer passed into the urine, as it does when the circulation of the kidneys is undisturbed. Again, Ribbert ('Centralblatt,' 1879), having set up an artificial albuminuria in rabbits with egg-albumen, excised the kidneys, and placed them directly in alcohol, so as to coagulate *in situ* the albumen in their interior; he then found that the spaces within the Malpighian capsules always contained coagulum as well as the tubes. Such experiments cannot, of course, exclude the possibility that an albuminous fluid may also sometimes transude through the tubal capillaries. But it must be remembered that in that case it would naturally find its way, not into the urine, but rather into the lymph-spaces between the tubes. Consequently, even when albuminuria is dependent upon obstruction to the blood-flow through the systemic veins, and associated with an "obstructive" form of dropsy, it does not seem likely to be due merely to an escape of serum through the tubal capillaries as the result of increased pressure.

What appears to be the most probable cause of albuminuria in general is the occurrence of some nutritive change in the epithelium covering the glomeruli, rendering it no longer capable of resisting the passage of albumen. This view is maintained by Leube and by Ernst Wagner, as well as by Cohnheim. It is held that whenever a full stream of arterial blood is not kept up through the capillary tufts, their epithelium is liable to be damaged, so that it can no longer fulfil its normal function. The instance which Cohnheim adduces as most obviously supporting such an opinion is that of the albuminuria which follows the suppression of urine during an attack of cholera (vol. i, p. 284). This, he maintains, is precisely analogous to the albuminuria which can be experimentally produced by temporary obstruction of the circulation through the renal artery, and which lasts for hours or even days after the obstruction is removed. Another cause of deficiency of blood supply to the glomeruli may be an impeded outflow through the veins of the kidneys. As a matter of fact, Ribbert has shown that after arrest of the circulation through the renal artery the cells of the glomerular epithelium become obviously swollen and altered in appearance; but in many cases it is a mere assumption that any change occurs in them, nothing having as yet been detected with the microscope.

According to this conception of the pathology of albuminuria, it has no essential relation to the state of the blood pressure on the vessels of the kidneys. The prevalent doctrine, that nothing favours the escape of albumen so much as an increase of blood pressure, was the conclusion at which Stockvis arrived as the result of his elaborate investigations, and it was adopted by Bartels. According to Cohnheim, however, it is based upon no good evidence whatever, whether experimental or pathological. The occurrence of albuminuria as the result of venous obstruction certainly lends it no support, because the pressure in the glomeruli is probably thus diminished rather than excessive, in consequence of the enfeeblement of the heart's action, which arises at an early period in such cases; and Runeberg actually maintains that albuminuria is always dependent upon a *deficiency* of blood pressure.

There does not appear to be any more foundation for another theory of albuminuria, according to which it depends upon a change in the albuminous elements of the liquor sanguinis, enabling them to pass with undue facility through the walls of the glomeruli. Stockvis ingeniously disproved this notion by the direct experiment of injecting albuminous urine from patients with Bright's disease into the veins of animals, when he found that the albumen did not, as a matter fact, escape from their kidneys. The same observer failed altogether to obtain experimental corroboration of the idea, formerly common, that hydræmia may be a direct cause of albuminuria.

"*Physiological*" *albuminuria.*—If now we pass on to discuss the conditions under which albuminuria occurs, we find, in the first place, that it is seen in many persons who are, so far as can be ascertained, in good health, and whose kidneys appear to be perfectly sound. It is only within the last few years that this fact has been clearly ascertained. Leube tested ('Virch. Arch.,' 1878) the urine of 119 soldiers, and found albumen in the urine passed in the morning by five of them, and in that passed at midday after a march by no fewer than nineteen; the urine passed in the evening was never albuminous. Fürbringer (*ibid.*, lxxi; and 'Ztschft. f. klin. Med.') tested the urine of sixty-one children, and detected albumen in seven cases, always in the latter part of the forenoon. In other instances the presence of albumen in the urine of healthy persons has been traced to some definite cause. Thus Dr George Johnson has recorded ('Clin. Soc. Trans.,' vol. vii) several cases in which it was temporarily produced by cold bathing; the same observer ('Brit. Med. Jour.,' 1879, ii) alludes to other cases in which it followed active walking exercise, an instance of which also occurred to a medical friend of the author's in his own person. In another case which came under the editor's notice, temporary albuminuria followed a day's hunting Fürbringer relates a case in which it was more than once brought on by distress of mind. Dr Moxon related in the 'Guy's Hospital Reports' for 1878, several cases in which albumen was from time to time discoverable in the urine of boys and young men who were generally anæmic, listless and languid; with all in whom he was able to trace the further progress of the affection, it sooner or later passed off, usually in the course of a few months. Sir William Gull had previously observed the same condition, also in young persons, and associated with similar symptons. In some of Dr Moxon's cases, "albuminuria of adolescents" (as he terms it) was associated with oxaluria. Ernst Wagner speaks of having seen similar cases in anæmic and weakly girls. Dr Dukes, of Rugby, shortly afterwards stated that he had seen ten cases in boys of thirteen to seventeen, in whom albuminuria had occurred as the

result of cold, exertion, or excitement, but subsided when they were kept in bed and on a milk diet ('Brit. Med. Journ.,' Nov. 30th, 1878). Dr Pavy has described the same clinical condition as "cyclic albuminuria," by which is denoted the recurrence of traces of albumen in the urine at more or less regular intervals, which correspond chiefly to the periods of taking food. As the same author long ago proved (1863), the amount of albumen passed in cases of Bright's disease varies according to the amount and nature of the diet. Mr Lucas has met with several cases among surgical out-patients, associated with flat-foot, in youths whom he believed to be habitually guilty of masturbation.

One of the most recent investigations on this subject was made by Dr A. W. Stirling on 369 boys between twelve and seventeen years of age, on a training ship ('Lancet,' 1887, vol. ii, p. 106). Of the whole number he found more or less albumen in 77, without any other sign of renal disease. It was far from constant, and most often present soon after rising. Beside the exposure to cold on leaving bed, two other causes were suggested,—one that assigned by Mr Lucas, and the other, exertion in blowing wind instruments.

The view which is taken by recent German writers with regard to what they call "physiological albuminuria" is that it depends upon a congenital deficiency in the power of the glomerular epithelium to resist the passage of albumen through it. It may perhaps be urged in support of such a theory that there are two pairs of brothers among the seven cases of which Dr Moxon gives details; and Leube also mentions having met with the affection in two brothers. But these cases are quite exceptional.

The condition is not a constant but an intermittent or occasional one, and the causes are not very far to seek. Cold to the surface, causing congestion of the kidneys, is the most important. Staying long in a cold bath is the most frequent cause of this occasional albuminuria. It is also a cause of intermittent hæmoglobinuria; and in the case of the schoolboy cited above (p. 587), as in others, the same cause produced on most occasions hæmoglobinuria, on others albuminuria. Moreover, there is no absolute line between physiology and pathology, and if we believe (as we shall see there is good reason to believe) that exposure to cold over a large surface of the skin leads to acute congestion of the kidneys with passage of albumen, which goes on to acute nephritis, it is surely reasonable to suppose a less degree of the same effect in the cases under consideration. The effect of prolonged efforts of expiration would produce passive renal congestion like that which leads to albuminuria in cardiac disease.

The passage of albumen associated with oxaluria and "nervous debility" has probably a somewhat different pathology. Is it quite certain that the albumen was renal in origin, or that a precipitate of mucin by picric acid has not sometimes been taken for albumen?

But the most important question is whether in such cases the occurrence of albuminuria indicates any tendency to the development of organic renal disease. Ought a young man in whom this affection is discovered to be regarded as eligible for life insurance at the ordinary rate? Leube and Fürbringer would doubtless have answered this question in the affirmative, and so would Moxon, if it were clearly ascertained that the urine contained albumen only occasionally, and that in the forenoon. Both Fürbringer and Moxon detected a few hyaline casts in more than one instance, but this is certainly a rare exception; as a rule, the amount of albumen is small, it

is intermittent, not constant, and there are no other signs of disease in the urine.

It seems clear that the cases above recorded by so many independent observers are far too numerous to be set down as examples of latent Bright's disease. Had they all been of that nature we may be sure that some of them would have revealed their real character while they were still under observation; moreover, latent Bright's disease is of the cirrhotic kind, which is almost unknown in early life. But is there equal reason to be sure that they are not examples of incipient Bright's disease, or of the want of power to resist external influences which is the preceding condition of all diseased action? Dr Johnson has expressed ('Brit. Med. Jour.,' 1879) a decided opinion that temporary albuminuria, even when traceable to food, or over-exertion, or exposure to cold, will, if neglected, sooner or later lead to persistent albuminuria and to fatal disease of the kidneys. Of the fact that it is possible for albuminuria to be the only indication of ill-health, and yet for the kidneys to be undergoing grave structural changes, there is no doubt whatever. Dr Johnson mentions the case of a medical man, actively engaged in a large practice until shortly before his death from uræmia at forty-five years of age, whose urine had been albuminous from the time when he had been a student, and probably earlier still, for he had scarlatinal dropsy when fifteen years old. Clearly, therefore, an insurance office which ignored the presence of albuminuria in applicants who appeared otherwise well would sometimes lose thereby,

The only insurance company that is known to have inquired into the subsequent state of health of persons whose lives had been declined on account of albuminuria is the United States Company in New York. Among those who made applications to that office in the three years, 1878–80, there were sixty-nine (or from 10 to 12 per cent. in each year) whose urine was found to be albuminous. Before the end of 1880 four of these persons died, and it is stated by Mr Munn that the "general appearance of the majority of the others who had been under observation for more than a year was gradually deteriorating." It is to be noted, however, that few of them were under the age of thirty, and that the albumen was often present in considerable quantity. Consequently, although the results of this investigation show that an insurance company runs a great risk if it neglects to have the urine of applicants tested, they can hardly be said to throw much light upon the question of the occurrence of a physiological albuminuria in young subjects.

It is a significant fact that Mr Eales, of Birmingham, found retinal changes in five out of fourteen cases of supposed temporary albuminuria in persons between the ages of eleven and twenty-eight ('Birmingham Medical Review,' 1880).

Albuminuria in non-renal diseases.—Albumen occurs in the urine withou there being any marked or permanent lesions of the kidneys, under various morbid conditions, which may be briefly enumerated under the following heads:

i. *Mechanical causes.*—(1) Congestion: usually from obstruction of the general venous circulation as the result of valvular disease of the heart, or occasionally of dilatation of the right ventricle from emphysema or like affections of the lungs.

Obstruction of the renal veins, independently of any disease affecting the whole circulation, occurred in a case recorded by Bartels of obliteration of the inferior vena cava above the mouths of those veins. Albuminuria from

pressure on the inferior cava and renal veins by ascites or tumour or a pregnant uterus is not uncommon.

(2) Obstruction in the renal arteries, usually by an embolus detached from the heart, and producing congestion with escape of albumen or of blood into the urine, by the same mechanism as explains cerebral hæmorrhage from a similar cause.

(3) Anæmia, leading to deficient blood supply to the kidneys through the renal arteries, as in cholera. (See p. 598, and p. 284 of the first volume.)

The same explanation probably belongs to albuminuria occurring in affections attended with abdominal pain and collapse. This cause of albuminuria has been especially pointed out by Fischl ('Deutsches Arch.,' 1881).

(4) Albuminuria appearing after obstruction of the ureters has been removed. This cause has been established by experiments on animals, and a case in point is given by Bartels in which the obstruction was produced by a calculus.

ii. *Fevers.*—Pyrexial albuminuria occurs most frequently in acute pneumonia and diphtheria, but also in typhus, enterica, cerebro-spinal meningitis, erysipelas, ague, pyæmia. In scarlet fever, also, albuminuria may occur during the pyrexial stage, and ought probably to be distinguished from that which appears later and is dependent upon nephritis. Cloudy swelling of the renal epithelium is constantly found in the bodies of those who have died of febrile maladies, but it is doubtful whether this causes the albuminuria, which is far less frequent.

iii. In certain affections of the nervous centres, especially cerebral hæmorrhages, concussion of the brain, epilepsy, tetanus, delirium tremens, albuminuria may occur; but it is doubtful whether it is caused by the nervous disorder. Cerebral hæmorrhage is notoriously frequent in cases of cirrhosis of the kidney, and drunkards are liable to Bright's disease. The albuminuria which follows a fit of epilepsy should always awake suspicion of uræmic eclampsia.

iv. *Poisoning* by different substances which act as direct renal irritants, *e.g.* cantharides and turpentine. Phosphorus probably acts in a different way.

If we ask what explanation can be given of albuminuria under these several conditions, the answer is more or less unsatisfactory, except in the case of cholera. Taking, for example, febrile albuminuria, we are quite unable to say whether the invisible physical change in the epithelium of the glomeruli, to which (as we believe) must be attributed the escape of albumen, is due to the heat of the blood itself, or to the action of the heat on the renal nerves, or to the chemical changes in the blood, or to the disturbance of the circulation through the kidneys as the result of diminished arterial pressure. When albuminuria follows an epileptic fit, or accompanies apoplexy or tetanus, it is probably a secondary result of venous obstruction from impeded respiration. But the relation of albuminuria to venous obstruction is itself susceptible of various interpretations. It may depend upon a deficient supply of arterial blood to the glomeruli, interfering with the due nutrition of their epithelium; or distension of veins at the line of junction of the renal cortex with the medulla may compress the straight tubes, and so interfere with the flow of urine, and exert pressure backwards in the Malpighian capsules upon the outer surface of the glomeruli. It has been thought that when albuminuria follows plugging of the ureter, the distended renal tubes press upon the veins; and thus that this cause of albumen in the urine may also be included under the head of "venous obstruction."

Albuminuria in renal disease.—Turning now to the albuminuria which

accompanies disease of the kidneys, we find that in one important particular it differs from that which occurs under all other circumstances, namely, in the much larger amount of the albumem, both absolutely and in proportion to the urine. In Bright's disease, for example, the urine may contain 5 per cent. of albumen, a quantity more than half as great as that in normal blood-serum, although the proportion is generally much smaller.*

It is undoubtedly to changes in the glomeruli that the albuminuria of Bright's disease is principally due, though, perhaps, the renal tubes may also have a share; whether this is more likely to be the case when their basement membrane has been exposed as the result of exfoliation of the epithelium must at present be regarded as doubtful.

2. *Tube-casts.*—The discovery of these bodies—"urine-cylinders," as the Germans call them—in the urine is generally associated with the name of Henle, who described them in 1844; but they had not escaped the notice of some previous observers. The merit of distinguishing and figuring their varieties and of applying their discovery by the microscope to the diagnosis of renal disease, is undoubtedly due to Dr George Johnson (1852). There are several different kinds of casts.

(1) *Hyaline or fibrinous casts.* These are delicate, transparent, and colourless, with defined outlines, but so little refractile that they are not always recognised under the microscope in the fluid in which they float, unless stained by carmine or iodine or aniline dyes. They vary greatly in breadth, from 0·01 to 0·05 mm.; their length may be only a few times greater than their breadth, or may reach 1 mm.: they are the longest of any kind. They may be either straight or curved.

(2) *Blood-casts, i.e.* fibrinous cylinders filled with red blood-discs. Their presence shows that the exudation is hæmorrhagic and probably acute.

(3) *Corpuscular casts,* containing small round nucleated cells which may be pretty certainly identified as leucocytes or "exudation corpuscles," though often confounded with epithelial cells. Like the hyaline casts they are signs of nephritis.

(4) *Epithelial-casts,* containing glandular cells from the convoluted tubules, more or less altered and granular, but by their size and shape distinct from white blood-corpuscles. They point to desquamation as the result of nephritis.

(5) *Oil-casts,* or fatty-casts, containing highly refracting oil-drops. These prove that the nephritic process has become chronic.

(6) *Granular-casts.* These are the most common and the least distinctive, for the granules may result from the disintegration of blood-discs (when they have a yellow tint), or of epithelium or leucocytes, and, in fact, are often mingled with these elements in the same cylinders. Or they may be fat-granules, shown as bright dark points. Or they may be only lithates accidentally deposited after the urine is passed.

(7) Lastly, one or two minute crystals of calcic oxalate or uric acid may be seen in a tube-cast.

In acute Bright's disease, for weeks together, the casts may contain red blood-discs, to the exclusion of all other elements. In other instances, tube-

* We must remember that the urine as we obtain it is, after all, a mixture of the fluids poured out by an almost infinite number of glomeruli and renal tubes, which may yield secretions of very different quality. So that when renal emboli, or localised new growths, are surrounded by zones of hyperæmic and inflamed kidney-tissue, any albumen that may be contained in the secretion from these parts is necessarily distributed over the very much larger quantity of normal urine poured out from the rest of the cortex.

casts look as if they consisted almost entirely of epithelial cells, packed so closely together that little or none of the hyaline sheath can be seen. The fat-granules or fat-drops are probably always derived from disintegrating epithelium; casts in which they are abundant are opaque and conspicuous, almost black by transmitted light. Wagner describes "granular casts" as sometimes "opaque like ground-glass," "appearing as if eroded or breaking down at their edges," and "sometimes presenting numerous indentations, or looking as though they were made up of a number of square pieces fused together." Lastly, "waxy" or "lardaceous" casts, which show the reaction with iodine, are described as occurring, though very rarely; they are highly refractile, and show more resistance to reagents than the common hyaline casts. Bartels is disposed to admit the possibility of their acquiring the lardaceous character as the result of being long retained in the renal tubes; and some writers affirm that they are not peculiar to cases in which the kidneys are themselves lardaceous.

Under the name of "cylindroids,"—for which "false casts" would perhaps be the best English equivalent,—some German observers have described certain flat, riband-like bodies, which are found in the urine of patients with scarlet fever, and also in cases of cholera and of recurrent fever. They are pale, homogeneous, colourless, and reach a much greater length than ordinary hyaline casts. Wagner says that their nature is still unknown. Some pathologists think that they are mucous, and that the urine does not contain them till after it has escaped from the pyramids; others believe that they have found them in the renal tubes. It does not appear clear that there are any transitional forms between "false" and "true" casts, though they may be found together in the same specimen of urine.

After death, casts may be seen in the kidneys in every part of their substance, from the convoluted tubes near the glomeruli down to the wide collecting tubes in the pyramids. They are most abundant in the looped tubes; as Wagner thinks, because they are slow in passing through these narrow canals. Certain writers have doubted whether casts from the convoluted tubes are capable of traversing the looped tubes so as to be discharged with the urine; but they are so elastic and flexible that this does not seem impossible for at least the smaller casts, and probably some of the larger casts are formed in the convoluted segment just before the collecting tube. Possibly materials that originally solidified in the highest tubes close to the glomeruli may afterwards be, so to speak, re-cast, taking the form of tubes lower down. It is stated, both by Wagner and by Bartels, that very wide, waxy, or granular casts are found chiefly in cases in which the urine is very scanty, and especially in the chronic forms of Bright's disease, within a few days of the fatal termination; and Bartels observes that such casts, moulded in the collecting tubes of the pyramids, can only be retained there long enough to undergo secondary changes when the secreting activity of the kidney is at a very low point.

The chemical nature of the hyaline material which appears to be the basis of all recently formed tube-casts has been especially studied by Rovida, who arrived at the conclusion that it is not identical with either fibrin or albumen, so that it can only be described as an *albuminoid* substance.

There have been various opinions as to the mode of origin of casts. Some observers have supposed them to be produced by a process of secretion from the epithelial cells of the tubes. Bartels upholds this view on the ground that spheroidal masses of plasma can often be seen protruding

from the cells into the lumen of a tube. Wagner, however, says that such appearances may be observed even in healthy kidneys. Another theory has been that they arise by the fusion together of altered epithelial cells. According to Weigert ('Volkmann's Sammlung,' 162–3) this is obviously the case in animals when nephritis is set up by the injection of chromate of potass under the skin; and in Bright's disease such an origin seems probable in the case of certain casts which have indented margins or look as if they were made up of an agglomeration of angular pieces. Some writers think, too, that casts which turn reddish-brown with iodine are formed out of epithelial cells that have first become lardaceous. But for the ordinary hyaline casts by far the most probable view is that they result from the coagulation of fibrinogen exuded from the glomeruli, just as in any other case of plastic or "croupous" inflammation. The fact that their reactions are not identical with those of fibrin may perhaps be explained by the supposition that they undergo some further chemical change under the influence of the acid urine which bathes their surface. The very short time which sometimes passes between the commencement of a morbid change in the kidney and the appearance of casts in the urine affords strong argument in favour of the view that they arise by coagulation of exuded plasma. Bartels, for instance, states that in a patient who underwent the operation of transfusion with lamb's blood, and whose urine up to that time was normal, urine passed two hours afterwards contained not only albumen, but also hyaline casts. In another case, that of a man who fell from a height upon his sacrum, urine voided five hours later showed hyaline as well as blood-casts. There is indeed a very close relation between albuminuria and the presence of tube-casts. In some cases, however, they appear in the urine a few hours, or even a day or two, before albumen is discoverable. In the urine of jaundiced patients casts of a greenish-yellow colour are often found, and Dr Finlayson says that, as a rule, in such cases, no albumen is present. Roberts alludes to cases of venous obstruction from heart disease or emphysema as being also accompanied with renal tube-casts, although there is no discoverable albuminuria. Of course nothing is proved by the fact that in cases of acute Bright's disease casts sometimes continue to be passed after albuminuria has ceased, because they may have been retained in the renal cortex for a considerable time after their formation. As a rule, the abundance of casts in a case of Bright's disease is proportionate to the amount of albumen in the urine; but to this there are exceptions, and in the same patient the number of casts may vary from day to day.

It is generally said that tube-casts possess great clinical importance from the fact that they prove the kidneys to be diseased. This is undoubtedly so far true that in cases in which the urine contains pus or blood, which may have been derived from the renal pelvis or the lower urinary passages, the discovery of casts is good evidence that the renal cortex is affected. If none can be found, it proves little or nothing, for their recognition is very difficult when leucocytes or red blood-discs are present in numbers. On the other hand, if albumen only is present, the presence of casts is not absolute proof of the existence of Bright's disease rather than of those slight or temporary changes in the glomeruli which occur in association with pyrexia, or as the result of venous congestion.

2. *Dropsy.*—In Bright's disease we meet with two kinds of dropsy. One is identical in its characters with that seen in heart disease, and depends upon

obstruction of the systemic veins. When it appears in the course of Morbus Brightii, it is only an indirect effect of the primary malady, its immediate cause being failure of the heart to maintain the needful activity of the circulation. It is always more marked in the dependent parts of the body than elsewhere, especially in the lower limbs, and it is associated with dyspnœa, with orthopnœa, and often with lividity. It occurs only in the most chronic forms of Bright's disease, usually when the kidneys are contracted, red, and granular, *i. e.* cirrhotic.

Widely different from this is the kind of dropsy which, although perhaps not absolutely more frequent, has been always justly associated with Bright's disease as its characteristic symptom. This kind of dropsy often begins in the face, about the eyelids, even before it affects the ankles. Its distribution is not independent of the influence of gravitation; for one may often notice that whereas the face is œdematous when the patient rises in the morning, this subsides towards the latter part of the day, and the ankles are swollen when he goes to bed. But it is not limited to the dependent regions of the body like the other form of dropsy, and it is not accompanied by dyspnœa or lividity. Its favourite seats are the eyelids and conjunctiva, the penis and scrotum (or the labia in women) and the loins, when it forms what Bright called "the renal cushion." We may explain the two former seats as due to the fact that the skin of eyelids and genital organs has no subcutaneous fat. Often, however, the whole of the body and limbs swell at the same time, and acquire a peculiar white waxy appearance, which is very characteristic. The occurrence of such general dropsy is frequently the earliest symptom of Bright's disease, and first draws the patient's attention to the fact that he is unwell. Generally, however, the urine is found, if tested, to be already albuminous; and after scarlet fever, when the supervention of dropsy can be anticipated as likely to happen, albuminuria may be known to be present for several days before œdema can be detected. On the other hand, it sometimes happens that the dropsy precedes the albuminuria by a day or two.

Dropsy without albuminuria.—Cases are now and then met with in which there is dropsy of precisely the same character as that which is so constantly associated with Bright's disease, but in which no albumen can at any time be found in the urine. Such cases are sometimes dignified by the name of "*essential dropsy*," but one may fairly doubt whether the kidneys are healthy, although no clinical evidence to the contrary can be obtained. What, however, is more frequent is for a patient to come under observation with general dropsy that has already lasted several days, or even weeks, and for his urine to yield no coagulum, either then or at any subsequent period, while the dropsy more or less rapidly subsides. In these cases, which are not very rare, nothing is more likely than that albuminuria was really present at first, for it is well known that in the more transitory forms of Bright's disease the urine often becomes normal before the dropsy disappears.*

Theory of renal dropsy.—The most obvious suggestion is, that the characteristic anasarca of Bright's disease depends upon an altered state of the blood, the result of the perverted action of the kidneys. Bostock and Rees, who made analyses of the blood for Bright himself, Christison and many later

* On this point see some cases in children recorded by Dr Duckworth in the 'St Barth. Hosp. Rep.,' and by Dr Johnson in his recent lectures (1887). I have seen this acute anasarca without albuminuria once in a child five years old, and twice in adults, one a young man in hospital, the other a young married woman; two cases were watched throughout.

observers, have found that the density of the serum is greatly reduced, being not more than 1020, or even 1013, instead of the normal density of 1030. It is natural to refer this physical change in the blood to the deficient excretion of water; but some writers have laid special stress upon the loss of albumen, through the glomeruli of the kidneys, and the resulting "hypalbuminotic" state of the blood, as the main cause of the low specific gravity of the blood-plasma. But Cohnheim points out that the amount of albumen which is secreted by the kidneys is after all inconsiderable. In most cases the percentage of albumen in the urine does not exceed 2 per cent.; in exceptional instances it may reach 4 or 5 per cent., but then the quantity of urine passed in the twenty-four hours is always much diminished, so that after all the total daily loss of albumen cannot be calculated at more than from eight to ten or twelve grammes (two to three drachms). It is obvious that, unless the assimilation of food is greatly interfered with, such an amount of albumen can be very easily replaced. Moreover, quite as large quantities of albumen are lost, without any dropsy resulting, by patients with large granulating wounds, and by those who have chyluria; and far larger quantities by women during lactation.

Consequently, it has been urged by Bartels and others that the really important factor in the production of renal dropsy is the deficient excretion of water by the kidneys. Rehder is cited by Bartels as having made a very elaborate series of investigations, in several cases of Bright's disease, as to the relation between the amount of water drunk (that contained in the solid food being, however, left out of consideration) and that discharged in the urine from day to day: in one case particularly he found that during periods when the dropsy was on the increase the water excreted was not more than from 29 to 49 per cent. of that which was ingested, whereas during periods when the dropsy was decreasing, the ratio was from 72·5 to 100·5 per cent. But, as Cohnheim remarks, such observations, after all, warrant no conclusion as to the nature of the connection between scantiness of the urine and dropsy. One has just as much right to suppose that the variations in the dropsy caused those in the activity of the kidneys, as to take the converse view. The effect upon the blood of a deficient excretion of water by the kidneys (supposing it not to be corrected either by diminished ingestion of water, or by an increased loss of water through some other channel) must obviously be to increase the whole bulk of the circulating fluid, while diminishing the percentage of solids in it. Cohnheim expresses this by saying that the resulting state of the blood must be, not mere "hydræmia," but "hydræmic plethora." Now, he and Lichtheim ('Virchow's Arch.,' lxix) made a series of experiments upon dogs, in which they found that the injection of enormous quantities of a half per cent. solution of salt into the blood produced not the slightest anasarca, even when the renal arteries were ligatured, so as to cut off the escape of the fluids through the kidneys. So far, therefore, as experiment can settle the question, it appears that a "hydræmic plethora" is incapable of causing the dropsy of Bright's disease.

But, in fact, there is no evidence whatever that such a condition of the blood occurs in this disease, or *can* as the result of impairment of the renal functions. Unfortunately, nothing is positively known as to the amount of water which escapes from the lungs or from the skin, though it must be admitted that the dry harsh state of the cutaneous surface in many cases of Bright's disease, and the difficulty with which visible sweating can

be induced, render it unlikely that the skin takes up any part of the renal function. But in one way or other the inactivity of the kidneys is compensated for, and the volume of the blood remains unaltered or nearly so. Further, there is abundant clinical proof that even complete arrest of the secretion of urine causes no dropsy. Not to mention the anuria of hysterical women, there are the cases of "obstructive suppression" resulting from plugging of the ureter of a single kidney, the other kidney having been previously destroyed by disease (*infra*, p. 669). In animals, again, ligature of the ureters is equally incapable of producing dropsy. Lastly, in many cases of scarlet fever dropsy sets in before there is evidence of impairment of the renal functions, and certainly long before there has been time for the development of any great change in the density or in the volume of the blood as the result of such impairment.

Such considerations render it clear that some further explanation is needed of the occurrence of general dropsy in Bright's disease; and this is sought for by Cohnheim in a change which he supposes to take place in the walls of the capillaries, rendering them more readily permeable by fluids than they are in normal circumstances. In most cases the deficiency of albumen in the blood might be imagined to produce such a change. But this view is inconsistent with the fact that in some instances the dropsy sets in before there can have been time for the blood to become "hypalbuminotic" or subalbuminous. Cohnheim therefore falls back upon the ingenious suggestion that the vessels of the skin and of the subcutaneous tissue become altered by the same cause which sets up the renal affection. He points out that whereas dropsy accompanies the nephritis that follows scarlet fever or exposure to cold, no such result is observed when a like nephritis arises in the course of diphtheria or of relapsing fever, in which diseases the skin remains intact. Obviously, the explanation is valid only so far as anasarca is concerned; and Cohnheim accordingly insists that dropsy of the serous cavities and of mucous membranes does not occur in most cases of Bright's disease—at least in an early stage, when failure in the heart's action cannot be supposed to play any part in their production. But the author's experience certainly accords with that of Wagner, that in autopsies upon some most acute cases, as, for instance, after scarlet fever, one generally finds some fluid effused into deeper parts of the body, though not, perhaps, in very large quantity.

It is evident, as Cohnheim himself points out, that the hypothesis of a change in the capillary walls, as the immediate and fundamental cause of renal dropsy, brings the affection somewhat closer than before to the inflammatory forms of œdema. But in one respect there is an important difference, namely, as regards the composition of the effused liquid. This, in Bright's disease, has always an extremely low specific gravity, and contains but a very small quantity of albumen; in fact, it exactly resembles in these points the liquid that is poured out in the "mechanical dropsy" of heart-disease, or of pulmonary emphysema. C. Schmidt found in one case that the dropsical fluid from the subcutaneous tissue contained 0·36 per cent. of albumen, that from the meninges 0·6—0·8 per cent., that from the peritoneum 1·13 per cent., that from the pleura 2·85 per cent. Bartels examined fluids taken directly after death from different parts of the body of a person who died of advanced dropsy; and found that the specific gravity of the blood-serum being 1015·60, that of the pericardial fluid was 1009·7, that of the peritoneal fluid 1009·6, and that of the anasarcous

fluid 1007·65; in each of the dropsical fluids the main part of the solid constituents was made up of inorganic salts. Urea, in the proportion of about 0·3 per cent., was detected in anasarcous fluid, and also in ascitic fluid by Edlessen; in pericardial fluid he once found as much as 1 per cent. of urea.

Difficult as it is to answer all objections, it seems probable that the characteristic anasarca of acute Bright's disease, as well as the pleuritic and ascitic effusion, is really—as it was supposed to be before Bright began his researches—an inflammatory exudation.

4. *Albuminuric retinitis.*—One of the most characteristic indications of Bright's disease is, in some cases, the presence of changes in the retinæ. These are said to have been first noticed *post mortem* by Türck in 1850; but the discovery of their importance in relation to kidney disease is assigned to Heymann, in 1856. They occur only in cases which are already chronic; in advanced stages of tubal nephritis after scarlet fever or during pregnancy; when the kidneys are cirrhotic; very seldom in cases of lardaceous disease, and probably only when it has long been associated with atrophic changes. Sometimes, however, the recognition of changes in the retinæ by means of the ophthalmoscope is the first thing which suggests that the patient is out of health. Their frequency is believed by Dr Gowers to agree with the statement of Mr Eales, who found them in 28 out of 100 cases of chronic Bright's disease, or in about 2 of every 7 cases ('Birm. Med. Rev.,' 1880). They vary in character in different instances, but they are commonly included under the name of "albuminuric retinitis," although this is not quite appropriate to all the morbid conditions.

The most common form of this lesion is, in fact, one which seems to be merely degenerative. It consists in the formation of *whitish spots*, sometimes close to the optic disc, sometimes elsewhere; near the macula lutea they often appear as fan-like streaks. They may be round dots, so minute as to be only visible by the direct method of examination; or they may be large, irregular patches, which equal the disc in size, and which may coalesce into large areas round it. A less intense diffuse opacity often extends over more or less of the retina. Associated with the white spots, or occurring independently of them, *hæmorrhages* are very frequently observed. These lie, for the most part, in the nerve-fibre layer of the retina; and they therefore are often "flame-shaped" (to use Dr Gowers' expression), following the radiating course of the fibres. They may also run by the side of and parallel to the vessels. When they are large they may be irregular in shape, or may penetrate into the deeper layers of the retinæ. In some cases, again, *optic neuritis* may be the most conspicuous retinal change. If no white spots are discoverable, the appearance will then be identical with that which is so commonly produced by intracranial disease, and it may ultimately run on to atrophy in exactly the same manner. Lastly, there may be a general *œdema* of the retina, with complete obscuration of the disc. The arteries are narrow, and to a great extent concealed; the veins distended and tortuous. There are always many hæmorrhages, forming large streaks in the course of the nerve-fibres. White spots are commonly numerous, large, rounded, and soft-edged. Dr Gowers, from whom the above description is taken, says that this form of albuminuric retinitis is confined to cases of severe and rapidly fatal Bright's disease, so that there is rarely time for it to subside or to pass into an atrophic stage.

Anatomically the white spots depend upon a degenerative change in the

nerve-fibres, which become greatly thickened, varicose, and filled with fat-globules; "compound granule-masses, too, appear in large numbers." The vertical fibres of Müller become swollen and fatty. The fan-like distribution of the spots near the macula lutea is said to depend upon the peculiar arrangement of these fibres in that position; they radiate from the *fovea centralis*, and their direction is somewhat oblique. The diffuse opacity is due to œdema, which gives rise, in hardened preparations, to the appearance of cavities, separating the retinal elements from one another. Dr Gowers' drawings of microscopical sections show how greatly some of the layers (including even the layer of rods and cones) may be thickened where there is a white spot.

With regard to the *pathology* of these retinal changes but little is yet understood. Traube was disposed to insist on the fact that they scarcely ever occur except in cases in which cardiac hypertrophy is already present, as showing that the heart affection was mainly concerned in producing them; and Cohnheim still upholds the same opinion. But probably the material fact is rather that albuminuric retinitis requires a considerable time for its development, so that before it appears the heart is almost certain to become enlarged. Dr Gowers draws attention to the small size of the retinal arteries, which in some cases of chronic Bright's disease are seen with the ophthalmoscope to be not more than one half or even one third of the size of the veins; but he regards this as the result of a vital contraction of their coats, and not of any organic changes in them. Mr Brailey, however, has shown that these vessels become affected with an *endarteritis obliterans*, exactly like that which we shall presently find to occur in the arterioles of the kidneys and of other parts of the body. Dr Gowers describes and figures irregular dilatations of the capillaries, with increase of the nuclei in their walls; he thinks it probable that such dilatations often lead to hæmorrhages. As for the optic neuritis, which we have seen to be sometimes the main ophthalmoscopic change, he has observed this especially when there have been conspicuous symptoms of cerebral disturbance, such as intense headache, delirium, and convulsions. He is therefore disposed to regard them as the cause of its predominance over the other retinal lesions in the cases in question.

Albuminuric retinitis, in its less intense forms, may be altogether unaccompanied with subjective symptoms. Or there may be more or less marked amblyopia, which may cause the patient to seek professional advice. Voelckers speaks of cases in which transitory darkening of the field of vision occurs from time to time during excitement or on exertion. Even in the most severe forms of albuminuric retinitis it rarely happens that central vision is lost, or that complete blindness follows. It is to be observed, too, that the occurrence of defective sight in a case of Bright's disease may depend upon very different causes. Wagner remarks that, altogether apart from the presence of retinal changes, it is not uncommon for hypermetropic patients whose general health is much affected to complain of impairment of vision which is really due to failure of accommodation. Such amaurosis is often an effect of uræmia.

It must not be supposed that any retinal changes are in themselves conclusive of the existence of Bright's disease. Dr Gowers relates a case in which a chlorotic girl became affected with what seemed to be an idiopathic neuro-retinitis, which ultimately left appearances undistinguishable from those of albuminuric retinitis. And sometimes there are difficul-

ties in the diagnosis of the retinal effects of renal from those of cerebral disease. When the white spots are very small, and limited to the region of the macula lutea, they require careful looking for, and dilatation of the pupil by atropine is often necessary to enable one to make sure of not missing them.

As a rule, when albuminuric retinitis has once developed itself, it persists until the patient's death. The exact appearances, however, vary from time to time; hæmorrhages may disappear and fresh ones may form; even the white spots may subside, though Dr Gowers says that this is very rarely the case with those that surround the macula lutea. It is in the Bright's disease associated with pregnancy that there is most ground for hoping for a permanent recovery from the retinal affection. The free use of purgatives is believed to favour its subsidence and to diminish the tendency to recurrence.

5. *Secondary inflammations.*—Among the most serious effects of Bright's disease as being frequently the direct cause of death, must be mentioned the occurrence of inflammation in one or more of the serous cavities, or in the lungs. Of the different serous membranes, the pleura is most apt to be attacked, the pericardium next, and the peritoneum only shortly before death or when paracentesis has been performed. Meningitis is very rare, and perhaps, when it seems to be of renal origin, some other well-accredited cause would be found, if carefully looked for.

The general tendency of the serous inflammations due to Bright's disease is to become suppurative; this is particularly well marked in the case of the peritoneum.* The pathology of these secondary inflammations is not very clear; they are commonly attributed to the undepurated state of the blood. Whatever their explanation, we may associate with them the occurrence of various inflammations of the skin and subcutaneous tissues in Bright's disease. These are often produced by irritation; hence the caution necessary in using acupuncture, and still more a permanent trocar, to draw off the serum. *Erythema leve* frequently occurs after pricking the legs, or severe erysipelas and sloughs may ensue.

But such dermatitis, both superficial and deep, may arise spontaneously: erythema of the face, the legs or the genitals, eczema, or diffuse scaly dermatitis; and occasionally, universal desquamative inflammation resembling pityriasis rubra.

6. *Cardio-vascular changes: hypertrophy of the heart, and thickening of the arteries.*—Bright himself observed that hypertrophy of the heart is frequently associated with renal disease; and since the publication of his researches, successive clinical teachers at Guy's Hospital—Barlow, Rees, and their successors—have never failed to insist upon the peculiar characters of the renal pulse as "hard," "wiry," "resisting," or "incompressible," or (to use a more modern expression) upon the *increase of arterial tension.* More recently the relation between Bright's disease and cardio-vascular changes has been zealously studied by numerous pathologists both here and on the Continent; and although there are still very wide differences of opinion on

* I do not remember to have ever seen a purulent exudation in the pericardium, even in the meshes of lymph.—C. H. F. On the other hand, Dr Sutton, a pathologist of no less experience, mentions purulent pericarditis as common, and almost characteristic of Bright's disease.

many questions, a great deal may be stated upon which all, or almost all, observers would agree.

In the first place, *hypertrophy of the heart* occurs in chronic "parenchymatous nephritis" as well as in "renal cirrhosis."* It also sometimes develops itself when the kidneys have become atrophied as the result of hydronephrosis, or of some other affection of the renal pelvis, as in cases recorded by Cohnheim. In association with the lardaceous change, however, it is not seen unless the renal cortex has become also affected with advanced tubal nephritis. It is comparatively slight when chronic Bright's disease is complicated by phthisis, cancer, or some other wasting disease, and in those who are very old.

Evidently, therefore, no explanation of the occurrence of cardiac hypertrophy can be valid unless it is applicable to both the principal forms of chronic Bright's disease. The *extent* to which the heart becomes enlarged differs in different cases—partly according to their duration; and it is far greater in renal cirrhosis than in any other form. Thus, whereas in the earlier stages of tubal nephritis its weight may attain fifteen or sixteen ounces, and in the latest granular and atrophic stage of that affection seventeen or possibly twenty-one ounces, there are some instances of (primary) renal cirrhosis in which it reaches twenty-three, twenty-four, twenty-five, or even twenty-eight ounces. Compare the tables of the weight of the heart in 188 cases of chronic parenchymatous nephritis, in 329 cases of cirrhosis ('Guy's Hosp. Rep.,' xliii, pp. 104, 111, 109), and in 146 cases of lardaceous disease.

The chamber chiefly affected is the left ventricle, the walls of which (and also the papillary columns of the mitral valve) become extraordinarily thick and fleshy, their substance being made up of muscular fibres of perfectly normal appearance. Sometimes the cavity is of normal size, sometimes more or less dilated. In a good many cases the right ventricle also is somewhat enlarged. This generally indicates that the left ventricle has not been able to maintain the circulation efficiently, or that pulmonary obstruction has arisen from bronchitis, œdema of lung, or some other cause. But to some extent it is inevitable that the right ventricle should share in the process of enlargement, especially when the left ventricle becomes very greatly increased in size.†

With regard to the exact character of the changes that take place in the *arteries* there are extraordinary discrepancies in the statements of different observers. In fact, among the pathologists who have especially studied this question within the last few years hardly any two entirely agree in their descriptions. That in middle-aged or old persons affected with Bright's disease the lesions commonly termed atheromatous are often found in the aorta, in the cerebral arteries, and in other arteries of large or medium size, was well known to Bright himself, and also to Wilks, Dickinson, and others who at different times wrote on the subject. But no great importance

* This fact was ascertained by Dr Galabin from cases in Guy's Hospital between 1868 and 1872; and Dr Goodhart's further experience of ten years (1873–82) confirms the conclusion ('Guy's Hosp. Rep.,' vol. xliii, p. 104). The left ventricle was hypertrophied in 109 cases, and normal in only 25.

† Traube asserted ('Ges. Abh.,' iii, p. 239) that he had been able clinically to determine the presence of hypertrophy of the heart within four weeks from the commencement of an acute renal affection. Similar cases were recorded by the late Dr Peacock and since by Dr Stone in his Croonian Lectures (1879), and Dr Goodhart ('Path. Trans.,' xxx). A case of recession of a hypertrophied ventricle has been recorded by Sir Wm. Roberts ('Glasgow Med. Journ.,' 1884).

seemed to attach to this circumstance on account of the frequency with which atheroma is seen in those who are advancing in years. It was Dr George Johnson who in the 'Med.-Chir. Trans.' for 1868, pointed out that the arterioles, not only in the kidneys, but also in the subcutaneous and submucous tissues, in the muscles, and in the pia mater of the brain, become remarkably thickened, and he attributed this change mainly to a hypertrophy of the muscular fibres in their walls. Four years later Sir William Gull and Dr Sutton read before the Royal Medical and Chirurgical Society a paper in which they declared the thickening to be the result of a hyaline fibroid formation, partly outside the muscular layer, in the *tunica adventitia*, partly in the *intima*, the muscular layer itself being often rather atrophied than hypertrophied, and the nuclei of its fibres degenerated. In the course of the discussion which followed, the "hyaline" appearance described by these observers was shown to be due to the action of the acidulated glycerine in which the preparations were placed for examination. Leyden among foreign writers alone lays stress upon this character; his figures ('Ztschrift. f. klin. Med.,' 1880) represent circumscribed glassy patches lying in the coats of the thickened vessels, and are very unlike those given originally by Gull and Sutton. On the other hand, the different German pathologists who have taken up the question likewise fail to confirm Dr Johnson's statements as to the existence of muscular hypertrophy. Ewald, indeed, admits ('Virch. Arch.,' 1877) that there is an increase in the thickness of the muscular coat, but this, he says, is due to an enlargement of the fibres, and not to a multiplication of them. But according to Sotnitschewsky ('Virch. Arch.,' 1880) when this coat is found thickened, it is as the result of increased fibrous tissue. Lastly, all recent writers agree in considering that an almost invariable condition affecting the intima is that which was originally named by Friedländer *arteritis obliterans*.

It seems impossible to reconcile these varying descriptions except by supposing that the affection of the arterioles in Bright's disease differs in different cases, perhaps even in the same case at different periods in its course. Another explanation may be that this change is most constant in the chronic atrophic form of Morbus Brightii, which is undoubtedly less common in Germany, and perhaps on the Continent generally, than in England. But after all, as we shall presently see, it is only with reference to the exclusive theory proposed by Dr Johnson that the exact character of the arterial change is of primary importance.

The view which is to be taken of the relation between Bright's disease and these cardio-vascular changes, centres upon the explanation of the fact that one of the most marked clinical features of the disease is a state of high pressure or tension in the arteries. This forms an important element in the hard or incompressible pulse. Since the invention of the sphygmograph it can be estimated much more accurately than formerly.

The first point to be noted in all such tracings is that the pressure applied to the artery while they are being taken is far greater than that which brings out the characters of the pulse most distinctly in health; as registered by the late Dr Mahomed's spiral eccentric it was from four to six ounces, instead of being from one and a half to three ounces. The next points are the breadth or "bluntness" of the tidal wave and the distance of the dicrotic notch from the upstroke; these indicate prolongation of the ventricular systole. The last point is the distance of the bottom of the dicrotic notch above the base line (cf. vol. i, pp. 906, 972).

If, now, we consider the pulse as it is appreciated by the finger, we find, according to Dr Mahomed, that these characters may be distinguished. First, it is *persistent*; even in the intervals between the cardiac beats, the artery feels full; it may even be visibly full and tortuous. One might imagine that its coats were thickened, but on emptying it by pressure above, one finds that it cannot be rolled beneath the finger, as a thickened vessel can be. Next, it is *long*, not falling away as soon as it has reached the finger, but pushing or laboured in character. Lastly, it is *hard* or *incompressible*, requiring much force to overcome it. Both the last characters are really indications of the state of the left ventricle generally associated with high arterial tension—the slow prolonged systole, and the hypertrophy of the ventricular walls.

Examination of the heart often yields valuable corroborative evidence. There may be an enlarged area of percussion-dulness, displacement of the apex outwards, and a heaving, laboured impulse. It is, however, important to be aware of the fact that these signs are not seldom absent, even when there is no obvious emphysema or other disease of the left lung to account for it. Thus, in cases of renal cirrhosis, when perhaps the patient has been admitted with cerebral hæmorrhage, we have again and again failed to detect any indication of cardiac hypertrophy, although at the autopsy a day or two later the heart has been found enormously enlarged. It is particularly in those cases in which the hypertrophy is unattended with any dilatation that the difficulty arises. On auscultation the first sound may be faint, or dull and prolonged, or reduplicated, or even replaced by a murmur. Mahomed also maintained ('Guy's Hosp. Rep.,' 1879) that it is sometimes preceded by a short sound resembling the presystolic murmur of mitral stenosis.* A more characteristic auscultatory sign, and one which is the direct result of the increased arterial tension, is the loud, ringing, or metallic quality of the (aortic) second sound, as it is heard at the base of the heart, or over the carotid artery. Occasionally, also, a diastolic shock can be felt by the hand placed over the cardiac region.

With regard to the *cause* of the high arterial tension in renal disease there is much difference of opinion. From the days of Bright down to the comparatively recent time when the question first began to be actively discussed, the view generally accepted was that the altered state of the blood created an obstacle to its passage through the capillaries, and that the heart had consequently to put forth more force to maintain the circulation. In 1868 Dr George Johnson, relying upon his observations as to the existence of hypertrophy in the muscular walls of the arterioles, propounded the theory that these vessels exert a "stopcock" function, resisting the passage into the capillaries of blood which, as the result of defective elimination by the kidneys, is noxious to the tissues. In fact, he imagined an active antagonism between the heart and the arterioles, as the result of which they each become hypertrophied. Mahomed reverted to the view that the obstruction is in the capillaries, and this view is also ably supported by Dr Saundby. But, believing that the high tension in the arteries precedes the development of renal disease, their notion is that the supposed impurity of the blood is due, not to imperfect excretory activity on the part of the kidneys, but rather to over-eating and over-drinking, by which it becomes charged with injurious matters. The objection to all such theories, however, is that there is no proof whatever that any changes in the circulating fluid are capable of

* Surely this is the same sign as what is called reduplication.

retarding its flow through the capillaries. Physiologists admit no causes of such retardation except alterations in the capillary walls themselves; and these only affect the circulation locally, as in the case of inflammation. And although the injection of urea into the blood is capable of increasing the arterial pressure in animals, yet this is only when the quantity injected is so large as to render the experiment inapplicable to human pathology.

Such considerations have lead some German pathologists to look elsewhere for an explanation of the cardio-vascular changes in Bright's disease, and of the high arterial tension which is so closely associated with them. Traube, in 1856, suggested that destruction of the renal parenchyma would have two results, each of which might tend to augment the pressure in the arteries; one being the accumulation of water in the blood from impairment of the secretory activity of the kidneys, the other the diminution in the amount of blood flowing from the arterial into the venous system as a consequence of obliteration of vessels in those organs. But the researches of Cohnheim and Lichtheim (see p. 607) have shown that the first of these two conditions cannot act in the manner supposed, and the second certainly seems altogether inadequate to produce any marked effect.

Cohnheim, however, has recently put this part of the question in an entirely new light. He gives reasons for thinking that the activity of the circulation through the kidneys at any moment—in other words, the state of the smaller renal arteries as regards contraction or dilation—depends not (as in the case of the tissues generally) upon the need of those organs for blood, but solely upon the amount of material for the urinary secretion that the circulatory fluid happens then to contain. This suggestion has bearings upon the development of hypertrophy in one kidney when the other has been entirely destroyed. But another consequence deducible from it is that when parts of both kidneys have undergone atrophy, the blood-flow to the parts that remain must, *cæteris paribus*, be as great as it would have been to the whole of the organs if they had been intact. In order, however, for such a quantity of blood to pass through the restricted capillary area now open to it, an excessive pressure is obviously necessary. This can be brought to bear only by increased energy in the pulsations of the left ventricle, combined with the maintenance of a corresponding resistance in all other districts of the arterial system. And so one can account at once for the high arterial pressure and for the consequent cardio-vascular changes.*

There is not any novelty in the idea which forms the basis of this theory, namely, that the hypertrophy of the heart in Bright's disease is a *compensatory* change, enabling the organism to withstand the consequences of the disease. But what seems not to have been clearly perceived is that a hypertrophied heart cannot effect this result unless it is supported by a resistance in the systemic arterioles proportioned to that which exists in the kidneys. Surely the fact that the secretion of urine is maintained or increased, shows that considerable compensatory changes must have taken place in the systemic arteries generally, as well as in the heart. One advantage of such an explanation of the high arterial tension of Bright's disease is that it enables us to see that the exact means by which it is kept up may vary in different cases and at different periods of the same case. At an early stage of the parenchymatous affection it can only be by contraction of the muscular walls of the arterioles, such as Dr Gowers believes that he has seen in the retina;

* In explaining this theory, the language used is somewhat different from that which Cohnheim employed, but the author's view was essentially the same as his.

and even in the chronic form of Bright's disease this must still play an important part, at least in cases in which a state of low pressure and dicrotism can be induced by the inhalation of nitrite of amyl, as has been shown by Dr Broadbent. But the presence of *endarteritis obliterans* is no doubt also concerned in producing and maintaining the peripheral resistance which leads to the high arterial tension.

It is obvious that this view fits in with the fact that cardio-vascular changes, like those that occur in Bright's disease, may likewise accompany atrophy of the kidneys from hydronephrosis. Nor is there much difficulty in bringing it into accord with Mahomed's observation, that there are some young persons with presumably normal kidneys in whom the arterial tension is constantly high, notwithstanding that they are in perfect health. Assuming that the urine in such cases is natural in quality and in quantity, one can but suppose that the kidneys are for some reason incapable of secreting such urine, except under excessive pressure. The condition would thus be comparable with the "renal inadequacy" described by Sir Andrew Clark. A very important question is whether it is to be regarded as a warning of the probable supervention at a later stage of Bright's disease, or of endarteritis and vascular changes. One of the points insisted on by Sir William Gull and Dr Sutton was, that the arterio-capillary fibrosis which they describe sometimes occurs independently of any affection of the kidneys.

Hæmorrhages.—It is doubtless as a more or less direct result of the high arterial tension of Bright's disease that the rupture of vessels in different situations is to be explained. Thus arise retinal hæmorrhages (p. 609), cerebral hæmorrhage, epistaxis, and hæmorrhage from the stomach and intestines. As regards epistaxis, Mahomed notes ('Guy's Hosp. Rep.,' 1881) the fact that even when the patient is much blanched by loss of blood the pressure in the arteries may still remain excessive.

7. *Uræmia.*—That the chief symptoms of Bright's disease are in many cases cerebral has been well known from an early period in its history, but there have been wide differences of opinion as to the mode of origin of such symptoms. These differences of opinion, however, have not prevented their being universally termed "uræmic," although the name uræmia (invented by Piorry) implies in strictness the acceptance of the theory that they depend upon an accumulation of urea in the blood, a theory which cannot now be accepted in that precise form.

a. The cerebral symptoms of acute uræmia vary in different cases. The most striking form consists in the occurrence of seizures precisely like those of epilepsy. Such "*epileptiform*" paroxysms are seen sometimes in patients who are already confined to bed with dropsy or suffering from other effects of Bright's disease; sometimes in those who are still engaged in their daily occupations or who may even be apparently well. The onset may be either sudden or preceded for a few hours or days by headache, drowsiness, vertigo, a strange fixed expression of the face, dragging pains in the extremities, or a transient rigidity of the face, or of the lower jaw, or of a limb. Nausea, again, and even vomiting, may be among the prodromal symptoms; or severe dyspnœa, of some hours' duration. Wagner mentions that the pulse sometimes falls to 60 or even to 40 in the minute.

A description of the paroxysms themselves will be found in the chapter on epilepsy (vol. i, p. 795); for they are identical in every detail, even to the biting of the tongue, the foaming at the mouth, the involuntary dis-

charge of urine and fæces, and the subsequent sleep or stupor, sometimes replaced by an attack of maniacal excitement. Wagner says that the pupils are generally dilated, seldom small; but at Guy's Hospital, from the time of Addison, it has been usual to describe them as being more often contracted or of the natural size; they usually retain their sensitiveness to light. The temperature may rise several degrees, reaching 102° or 104°, or even a higher point still; in one case 107°. During the coma which follows the convulsions it slowly falls to normal, or below; for some days afterwards it may remain as low as 94° or 95°. The pulse is commonly accelerated while the spasms continue; afterwards it returns to its natural rate, or may become slower still, remaining perhaps (as in a case of Wagner's) between 44 and 64 for the next fortnight. After the attack has ceased the patient is sometimes dull and depressed for some days. Hemiplegia has very rarely been observed; it might be expected to occur as it sometimes does after epileptic seizures.

In many cases, before the insensibility has passed off, after one uræmic paroxysm another sets in; and thus twenty or thirty may occur in succession simulating the *status epilepticus* (vol. i, p. 797). The disease then is very likely to prove fatal. But even after a series of fits it is not uncommon for recovery to take place; the convulsions cease, and the patient regains consciousness, to the surprise of his friends. A single paroxysm seldom ends fatally; but in 1862 a woman, aged thirty, died in Guy's Hospital within seven minutes from the commencement of uræmic symptoms. She had just eaten her breakfast, when slight spasmodic movements of the arms suddenly set in. She became pale, and her lips and fingers livid; there was foaming at the mouth. The pupils were dilated. The heart at first continued to beat regularly, but its action very quickly ceased.

Sometimes, instead of a uræmic fit having the typical epileptiform character its symptoms are of a different kind. In the 'Guy's Hospital Reports' for 1839, Addison described one variety as consisting in a "sudden attack of coma with stertor, or, in other words, *apoplexy*." Probably it is now a result of his teaching, that we at Guy's Hospital have for many years past been very cautious in diagnosing cerebral hæmorrhage in cases in which there was reason to suspect the existence of renal disease, notwithstanding the well-known frequency with which it occurs under such circumstances. Nevertheless, in the chapter on cerebral hæmorrhage, we found that our *post-mortem* room experience does not support the view that it is liable to be simulated by uræmia, at least when there have been no convulsions (cf. vol. i, p. 595). Roberts cites three uræmic apoplectiform cases, but each of them is open to criticism. One patient had had "a few drops" of laudanum given to him for diarrhœa just before the cerebral symptoms set in, so that it may be doubtful whether they were not due to the excessive action which even small doses of that drug are known to exert when the kidneys are diseased; in the second case epileptiform convulsions were present; in the third case there seems to have been no autopsy, so that the possibility of cerebral hæmorrhage is not excluded. Addison was of opinion that the two affections might be distinguished by the characters of the stertor that accompanied them; in uræmia, he said, the sound was more hissing, "as if produced by the air striking against the hard palate, or even the lips, rather than against the velum and the throat, as in ordinary apoplectic stertor." He also believed that the respiration was from the first much more hurried than in true apoplexy.

In other instances uræmia is said to manifest itself by *delirium*, lasting

for days together, or by rigidity of one or more of the limbs, or according to Charcot, by tremors like those of paralysis agitans. In a case of Roberts's in which the paroxysms coincided with the catamenial periods, consciousness was not lost; "during the convulsions the patient knew the persons about her, and called loudly to be held fast." Bright, in the 'Guy's Hospital Reports' for 1840, related a case in which for two days before death there occurred a very distressing and almost incessant twitching of the muscles, which increased until the arms and the legs were forcibly drawn up and the face was distorted by the spasms, yet the faculties of the mind were perfect to the last.

In some cases, uræmia shows itself by much slighter symptoms; by transitory *trismus* perhaps, or, frequently, as a premonitory symptom, by short attacks of *clonic spasm* in some of the facial muscles, or in those of the eyeballs, or of a limb; the patient retaining consciousness, or being at most a little confused or dull of intelligence.

But the most remarkable of all the effects of uræmia is, perhaps, *amaurosis*. This not infrequently occurs along with eclampsia; the patient, when he recovers consciousness, finds himself blind. Sometimes, according to Wagner, it precedes the convulsions. But more often it is the only symptom, except headache. It sets in suddenly, is bilateral, and is almost always complete, the patient having not the slightest perception of light. Dr Gowers says that the pupils generally still react to light; but Wagner says that they are sluggish, and that they may in some cases be altogether insensible and widely dilated. The ophthalmoscope does not show any change in the optic discs or retinæ.* This alarming affection rapidly subsides, so that the patient regains sight within twelve or twenty-four hours, or at the longest in the course of a few days. Wagner says that such a favourable termination may occur even when the pupils have lost their sensitiveness to light. The fact that, as a rule, the pupils react, seems to show that the seat of uræmic amaurosis must be above the corpora quadrigemina. It is said that in some instances a transitory defect of hearing, or even complete deafness, has been observed as a sequela of a uræmic seizure.

b. In striking contrast with these varied forms of what may be termed acute uræmia are some which are described as chronic. The latter are not, like the former, always obviously cerebral in their character. The lungs or the digestive organs may appear to be the parts affected. But there is good reason to believe that even in such cases the starting-point of the symptoms is generally, if not always, in the brain.

The *cerebral symptoms* usually consist of headache, giddiness, or drowsiness, any of which may go on for weeks, or for months continuously, or with intermissions. The patient's aspect is often remarkably dull and expressionless; he lies in bed, taking no notice of what goes on around him, and altogether indifferent to his own condition. Ultimately he may fall into complete stupor. A patient of the editor's recently died of tubal nephritis of about three months' standing, which was passing from the acute to the chronic stage, when he became increasingly dull and apathetic until at last he lay insensible, but without stertorous breathing, for several days before his death. Sometimes the general symptoms are like those of the typhoid state, the tongue being dry and brown, and sordes collecting upon the teeth and lips. Such cases might be mistaken for enteric fever.

* Two instances, however, are cited by Dr Gowers, in which slight œdema of the discs is said to have been detected, which passed off with the amaurosis.

In other cases, the principal indication of chronic uræmia is *dyspnœa*. This is generally paroxysmal, and is apt to come on at night, like asthma. It may be expiratory, like asthma, or inspiratory, as though there were laryngeal stenosis; or, again, both inspiration and expiration may be free, but unnaturally hurried.

Another sign of uræmia may be an intense *itching* of the skin. Sometimes patients go on scratching or rubbing themselves even when they are so far unconscious that it is impossible to rouse them.

One of the most characteristic symptoms is *vomiting*. At first it may occur only in the morning, when the stomach is empty. Afterwards it may take place whenever any food is taken, and become exceedingly intractable, continuing for weeks or even for months. Urea may often be discovered in the matters rejected, and some have supposed the vomiting to be the result of its presence in the stomach. But in Voit's experiments upon animals it was found that urea ingested with the food did not set up vomiting at once, but only when there had been time for it to be absorbed into the blood, and to act upon the nervous centres. The vomited matters are sometimes alkaline, perhaps from some of the urea having been decomposed into carbonate of ammonia; but as a rule they are acid. Bartels has suggested that œdema of the walls of the stomach may sometimes be the cause of vomiting in Bright's disease; but such an affection is rarely observed in the *post-mortem* room, and to suppose it capable of producing vomiting is a mere assumption.

Hiccough is not uncommon in association with other effects of uræmia, and Wagner mentions one instance of chronic Bright's disease in which hiccough and slight œdema of the lower limbs were the sole symptoms.

Diarrhœa is of rather frequent occurrence, and often accompanies vomiting. It sometimes seems to depend upon an inflammatory affection of the intestinal mucous membrane, which may be œdematous, or ecchymosed, or even in a state of severe "diphtheritic" inflammation, like that which is met with in dysentery. Occasionally its surface is covered with large leathery patches, or extensively ulcerated. In cases of this kind the evacuations often contain blood, and mucus and pus in large quantity. There is some doubt as to whether the diarrhœa and enteritis of Bright's disease are strictly of uræmic origin. Cohnheim and other recent writers think that they are rather effects of local irritation from carbonate of ammonia produced by decomposition of urea in the intestine.

Prognosis.—Epileptiform convulsions and the other symptoms that have been grouped together under the name of acute uræmia may accompany any form of Bright's disease, and are very frequent in the nephritis of scarlet fever, cholera, and of pregnancy. They sometimes pass off; and it may be added that their occurrence is no certain proof that the renal disease which causes them is so severe or so advanced as to be incapable of recovery. On the other hand, the prolonged stupor, the typhoid symptoms, and the other phenomena of chronic uræmia almost always end fatally. They are never seen in the more acute forms of Bright's disease—as, for example, after scarlet fever, or in association with pregnancy—unless the so-called "cholera-typhoid" (vol. i, p. 285) is to be taken as an instance to the contrary.

Theory of uræmia.—In discussing the cause of the symptoms that we have grouped together under the name of uræmia, we must in the first place consider whether those which are obviously cerebral may

possibly depend on actual lesions of the nervous centres. Some years ago Traube propounded the theory that they were due to œdema, combined with anæmia of the brain. The œdema he supposed to be brought about by the action of a hypertrophied heart upon the smaller intracranial blood-vessels, assisted by a watery condition of the blood itself. But every part of his theory has since been shown to be untenable. In many instances the brain is found after death to be perfectly dry; and when it is œdematous. Bartels is probably right in thinking that this is an effect, rather than the cause, of any convulsive seizures that may have occurred—unless indeed it is a mere accidental result of wasting of the brain, as is doubtless very often the case. Sometimes minute spots of hæmorrhage are found in greater or less numbers in the substance of the brain. In two instances of Bright's disease, both with large white kidneys, the pons and the bulb were found full of such capillary hæmorrhages. It seems most likely that they also are produced by the disturbance of the intracranial circulation, which cannot but accompany the uræmic paroxysm; their occurrence is far too exceptional to admit of their being regarded as its cause.

We are therefore driven to what may be termed the chemical theories of uræmia. There are, however, considerable difficulties in accepting the most natural supposition, namely, that it depends upon the retention in the blood of urea which the kidneys have failed to excrete. Frerichs consequently suggested in 1851 that the poisonous agent was really carbonate of ammonia formed in the blood by decomposition of urea. Subsequently Treitz amended the hypothesis by supposing that the carbonate of ammonia was produced, not in the blood, but in the stomach and intestine, a vicarious excretion of urea into the alimentary canal first taking place, and the carbonate of ammonia being afterwards reabsorbed into the blood. But this theory of "ammoniæmia," though at one time it was widely adopted in Germany, is now universally abandoned. On the one hand, it has been shown that though carbonate of ammonia, injected into the blood of animals, causes symptoms somewhat like those of uræmia, the resemblance is, after all, incomplete, and that many other salts produce like effects. On the other hand, many experimenters fail to detect carbonate of ammonia in the blood of uræmic patients, and there appears to be no doubt whatever that, if present at all, it is not in sufficient quantity to account for the effects attributed to it. One clinical point which Frerichs adduced in support of his view was that by holding a glass rod moistened with hydrochloric acid near the mouth of a uræmic patient the presence of carbonate of ammonia could be recognised in the expired air by the white fumes of chloride of ammonium that were formed. Schottin, however, showed that in many uræmic patients this test completely failed, whereas it often succeeded in other patients who lay in a typhoid state from whatever cause, the carbonate of ammonia being set free from dried secretions within the mouth, and not exhaled from the lungs.

Recent observers have accordingly fallen back upon the older and simpler theory, which supposes that the symptoms of uræmia are due to the presence in the blood of urea.

The objections which had caused this view to be discarded were drawn partly from experiments upon animals, partly from clinical observations of Bright's disease. Experiments had seemed to show that urea, and even urine itself, could be introduced in large quantity into the blood of animals without giving rise to any ill-effects. Voit and Oertel, however, found

'Ztschft. f. Biol.,' 1868) that although urea when added to the food of a dog produces no symptoms so long as it can be freely excreted by the kidneys, yet if the animal is not allowed to drink any water symptoms like those of uræmia appear.

The clinical objections to what may be termed the "uræmic" theory of uræmia were taken by Frerichs from a work which had shortly before been published by Dr Owen Rees; they were briefly that the occurrence and the severity of the paroxysms bore no necessary relation to the quantity of urine secreted, and that the blood was sometimes loaded with urea without any such symptoms appearing.

In fact the characters of the urine in cases of Bright's disease at the time when uræmia develops itself differ in different cases. As a rule the renal secretion is very much diminished in quantity for several days before the symptoms set in; it may even be completely suppressed. But sometimes there is a normal flow of urine, although it contains much less than the due amount of urea; and sometimes the quantity of urine is above normal, as in a case of Wagner's, in which the patient, a man named Richter, for three successive days passed seventy ounces daily. In that instance, however, its specific gravity ranged only from 1006 to 1010, so that after all the excretion of urea and of the other solid constituents of the urine was probably defective.*

Upon this difficult question of the relation of uræmic symptoms to the excreting action of the kidneys some fresh light has been thrown by certain observations made by Fleischer ('Deutches Arch.,' xxix, 1881). He instituted careful analyses of the urine passed by persons affected with Bright's disease, comparing them in each instance with analyses of the urine of healthy persons placed under exactly the same conditions as regards diet. He found, as a rule, that the amount of urea excreted by those who had Bright's disease was much diminished; but when uræmia set in, the amount of urea became increased far beyond the normal, either on the day of the seizure or else a day or two later. The explanation which he suggests is that when the accumulation of urea (and of other urinary constituents) reaches a point at which the system ceases to be indifferent to their presence, so that uræmia results—they at the same time stimulate the heart and the kidneys to expel them. Henceforth, therefore, it must be borne in mind that the fact of an abundant elimination of urea taking place during or after a uræmic fit, is no proof that it may not previously have been deficient, and this deficiency the cause of the attack.

On the other hand, even when systematic analyses of the urine show that the action of the kidneys has been imperfect, as when the amount of urea excreted is reduced to 200 or 150 grains daily, there is always the further difficulty that in many other cases it is quite as low, without any uræmic symptoms arising. Cases also occur in which the urine is more or less completely suppressed for a week or longer before uræmia develops itself. After all, however, such facts are entirely in accordance with clinical

* The man had not been œdematous, and there was therefore no reason for supposing that a reabsorption of dropsical fluid had anything to do with the large amount of urine poured out by his kidneys. Wagner, however, remarks that the tissues of the dead body may be found to be distinctly œdematous, when there had been no clinical evidence of it. It seems, therefore, not impossible that the absorption into the blood of such a latent accumulation of fluid may sometimes be the real cause of an excessive flow of urine previous to the development of uræmia, especially as the subsidence of dropsy is known to be frequently followed by uræmic symptoms.

experience in general. The effective operation of all "causes" of disease is liable to be interfered with by unknown conditions, of which the resistance of the patient's tissues is perhaps the most important. It is remarkable that uræmia is seldom met with in persons advanced in years; perhaps this suggests that a "predisposition" on the part of young subjects is one factor in its ætiology.

All doubts as to the occurrence of imperfect elimination by the kidneys in uræmia might be set aside if it were known that the blood in this state invariably contains more urea than in health. The earliest analysis of the blood in chronic Bright's disease seems to have been made by Dr Guy Babington, who states that in a case under the care of Dr Bright himself there was as much urea in the circulating blood as in the urine, a thousand grains of blood yielding fifteen grains of urea! Recent observers, however, have found much smaller quantities than this. Wagner says that, instead of the normal proportion of 0·16 or 0·2 parts per 1000, there may be 0·4 or 0·6 parts, or more. He further cites an observation of Hoppe-Seyler's, who, in the blood-serum of a cholera patient with uræmia discovered 1·27 parts of urea per 1000. But elsewhere he says that the quantity of urea in the blood has several times been found to be small, "so that an overloading of the blood with this substance certainly cannot be in all cases the cause of uræmia." But before we accept this important conclusion, we ought to know exactly at what period of the disease the analyses have been made in which no excess of urea has been detected. If Fleischer's observations are correct, it seems quite possible that in the course of a uræmic seizure, or afterwards, the blood might contain no excess of urea, and yet that a great excess might previously have been present, and have given rise to the attack.

Urea may also be discovered in considerable quantity in the various secretions. Its presence in the gastric and intestinal contents has been already incidentally mentioned. In one case in which there were bronchitis and extensive pneumonia, Fleischer found it in the sputum to the amount of about thirty grains in the thirty-seven ounces expectorated during twenty-four hours. But the most interesting fact of all is that in some uræmic patients urea is excreted by the skin. This seems only to occur shortly before death, and scarcely ever without the urine being completely suppressed. Schottin first observed it in 1852 in cholera patients. The 'Guy's Hospital Reports' for 1874 contain a report, by Dr Frederick Taylor, of a patient with Bright's disease, in whom, two days before death, there appeared on the face, neck, and hands white masses which adhered pretty firmly, and which, when removed, were found to be irregularly shaped, with crystalline spiculæ and prisms projecting from them. They yielded the several reactions of urea. The patient's face is described as having looked as though flour had been sprinkled over it. In some other cases the appearance is said to have been just as though a lather of soap had been allowed to dry on the surface, or as though the beard were frosted.

Most recent writers on the subject endorse an opinion which was first expressed by Voit ('Zeitschft. f. Biol.,' 1868), namely, that uræmia is not due to the poisonous action of any one ingredient of the urine—whether urea, uric acid, kreatinin, or other extractives. Voit himself was inclined to attribute a considerable share of the uræmia to the salts of potass. He believed that they may be produced by "any substance which is not a normal constituent of the body if it accumulates in large quantities and is not eliminated."

This vague statement, however, is opposed to one clinical fact, for a clear recognition of which we are indebted to Roberts, and which is of the highest importance both to the physiologist and to the physician. It is that symptoms altogether unlike those of uræmia, and holding a completely different course towards a fatal issue, are presented by cases in which the failure to eliminate urea and the other ingredients of the urine is absolute, but in which the cause of the suppression of the renal secretion is not an affection of the cortex of the kidneys, but obstruction of the ureters. These clinical symptoms are met with in what is called "obstructive suppression" of urine (*v. infra*, p. 668). The absence of uræmia in these cases seems clearly to show that where there is healthy kidney-substance, with an active circulation through it, the waste products which should be excreted in the urine undergo some chemical changes that render them incapable of producing uræmia, notwithstanding that they are retained in the body. Nor do the results of experiments upon the lower animals appear to be inconsistent with this view. Obstructive suppression is, of course, easily produced by ligature of the ureters; the effects of this operation are spoken of as identical with uræmia, but it is perhaps not to be expected that distinctions between different groups of symptoms should be so obvious in animals as in man. On the other hand, it is by no means easy to bring about non-obstructive suppression in such a way as to afford a satisfactory comparison. There are two ways in which one ought to be able to annul the activity of the secreting substance of the kidneys. One is by ligaturing the renal arteries, the other by excising the kidneys. Now, ligature of the renal arteries has been shown by Hermann ('Sitzungsbericht d. Wien. Acad.,' 1861) not to be effectual in arresting the blood supply to the kidneys, which may continue to pour forth urine. And excision of the kidneys is an exceedingly severe operation, very apt to produce vomiting and other ill-effects that make it unfair to contrast it with so simple a procedure as ligature of the ureters.*

Roberts maintains that the cause of uræmia is the accumulation in the blood of products intermediate between urea (or uric acid) and the albuminous substances from which it has its origin, such as kreatin and kreatinin, hypoxanthin and xanthin, or leucin (amido-caproic acid), or aspartic (amido-succinic) acid and tyrosin.

In conclusion, there seems to be no doubt that uræmia is produced by the poisonous action upon the nervous centres of materials accumulated in the blood as the result of defective excretion by the kidneys. But it is still uncertain whether this action is excited by one substance or by more; and in many cases the actual outbreak of convulsions is immediately due to some obvious disturbance of the balance of the bodily functions, which may be supposed to have been previously unstable. Thus Bartels relates a striking instance in which the production of profuse sweating in a dropsical patient by a hot bath, followed by hot packing, at once brought about a series of uræmic attacks; next day the dropsy was gone. Sometimes, perhaps, the immediate cause of uræmic symptoms is the sudden failure of the heart to keep up an active circulation through the renal vessels, so that the excretory function of the kidneys, which may for a long time have been more or less impaired, now becomes altogether ineffectual.

* After excision of the kidneys the quantity of urea which is found in the blood is not so great as after ligature of the ureters, but as Salkowski points out, the cause of this may very well be the vomiting that follows the former operation.

We now pass on to consider the several forms of Bright's disease enumerated at p. 594.

I. PARENCHYMATOUS NEPHRITIS.*—As remarked at p. 592, this name of Virchow's is adopted as the most suitable, but not as implying that the pathological changes in this form of Bright's disease are strictly limited to the secreting cells of the renal cortex. On the contrary we shall presently find that the glomeruli and even the connecting tissue are in many cases markedly affected.

Ætiology.—Parenchymatous nephritis is often due to obvious causes. It may be definitely traceable to *cold*. Bartels cites three well-marked examples of this; one is that of a patient who was taken ill as the direct result of going to sleep half undressed by an open window on a winter's night, after having spent the evening in dancing; another is that of a man who, while perspiring freely, left his smithy and went out into the open air in his shirt, getting wet through with a sleety rain; the third is that of a person who was skating, when he broke through the ice, and had much difficulty in extricating himself.

Very many cases arise from *scarlatina*. And some in which no definite cause can be found may be really due to a latent attack of scarlet fever, particularly during childhood. Cholera, erysipelas, enteric fever, smallpox, measles, all more or less frequently give rise to albuminuria and to some degree of nephritis, but it seems doubtful whether the renal affection in any one of these diseases is sufficiently definite or prolonged to justify the title of Bright's disease. In countries in which *ague* is endemic, it is said to be a frequent cause of Bright's disease. In women, *pregnancy* is often the cause, as the late Dr Lever showed many years ago, especially in primiparæ, and, above all, when there are twins. Sometimes the disease appears to recur in successive pregnancies. It generally manifests itself during the months of gestation. How it is brought about is not at all clear. It certainly is not due to pressure by the gravid womb upon the renal veins; and the most plausible view seems to be that it results from the kidneys having extra work thrown upon them in the elimination of effete matters. Its onset is often insidious, but those writers who separate acute from chronic Bright's disease include it under the former head.

Among the causes that are more or less slow in their action, the writer would be inclined to place indulgence in *alcohol* as a very important one, and this was the original opinion of Bright, although it is disputed by later observers. For example, a solicitor, usually moderate in his habits, some years ago acquired temporary albuminuria as the result of drinking sherry in large quantities to induce sleep, at a time when he had a great trouble weighing upon him. Another patient's urine was for many years albuminous, apparently as the effect of habitual excess in stimulants; he changed his ways, and two years later no evidence of any renal affection could be discovered, and he seemed to have regained his usual health.

Anatomy.—The appearances presented differ a good deal in different cases, even at the same period of the disease.

(1) *The large red and speckled kidney.*—Should it prove fatal during the first two or three months, the kidneys are either found of nearly natural size, or more often enlarged up to twice their natural weight. It may be noticed that they are rounded in shape, stretching their capsule, which is not thickened,

* *Synonyms.*—Tubular or tubal nephritis—Desquamative nephritis—Néphrite albumineuse—Croupöse nephritis—Acute and chronic epithelial catarrh of the kidney.

and can be as easily stripped off as from a normal kidney. Their colour is usually a dull greyish red, or a paler grey, the pyramids having a much deeper reddish-purple tint. Sometimes, especially if there has been complete suppression of urine, and if death has been due to convulsions attended with great pulmonary congestion, all parts of the kidneys are found gorged with blood, and of a dark chocolate colour (see Dr Dickinson's 5th plate). In most cases red points are scattered over the cortex; some of them are blood-filled glomeruli, others are punctiform hæmorrhages. But in some instances, particularly among those attributable to scarlet fever, the kidneys show scarcely any deviation from their natural appearance. In other cases the scarlatinal kidney is swollen, bright red, and dripping with blood when cut.

The principal morbid change affects the convoluted tubes of the cortex, the epithelium of which first becomes cloudy and granular, and afterwards proliferates, so as to fill them with masses of irregular or rounded cells. These are seen in sections blocking up the tubules and greatly increasing the thickness of the cortex. In the acute stage the epithelium and granules are mingled with blood-discs and leucocytes; as the affection becomes chronic, minute oil-drops appear and render the tubes black by transmitted light (see Dr Stewart's 3rd plate, fig. 1). Osmic acid shows this change early.

Recently, however, special attention has been paid to the histology of scarlatinal cases by Klebs, and in this country by Klein and by Greenfield; and these observers are agreed that the most constant lesions are those that concern the glomeruli and their capsules. Not only do the nuclei of the capillary tufts of the glomerulus proliferate, but there is also an abundant growth of nuclei within the capsule, leading to adhesion between it and the glomerulus, and ultimately to compression and atrophy of the latter. The peri-glomerular connective tissue also becomes crowded with nuclei, which ultimately develop into fibroid tissue; and the afferent vessel of the glomerulus, as well as its capillaries, undergoes a peculiar hyaline change. Sometimes these lesions are limited to a few of the glomeruli only; sometimes they are very widespread. It is obvious that the obliteration of the space naturally existing between the tuft and the capsule that encloses it must completely abolish the functions not only of the glomerulus itself, but also of the whole length of the convoluted tube that corresponds with it. And, further, the changes in the tufts themselves may fairly be supposed to obstruct the blood supply to the convoluted tubes, and to affect the nutrition of their epithelium. Consequently, some pathologists are now disposed to see in the "glomerulo-nephritis," as they term it, the fundamental morbid process that follows scarlet fever, and to regard the lesions of the tubal epithelium as secondary and relatively unimportant. If these views should be confirmed in their entirety, the name of parenchymatous nephritis may ultimately have to be given up. At present, however, it is uncertain whether the observations made on the early stages of Bright's disease consecutive to scarlet fever apply also to that which arises from cold, or which is attributable to pregnancy, or which begins insidiously without any assignable cause. Glomerulo-nephritis is certainly not peculiar to scarlatinal cases, for Cohnheim met with a typical example of it in the case of a man who died some weeks after having his skin rubbed all over with petroleum for four days consecutively.

(2) *The large white kidney.**—In cases that have been of somewhat longer

* Including the pale, marbled, or mottled kidney and the large, granular, smooth kidney of Bright—granular describing the appearance to the eye, not the feel to the hand. (See his 1st, 5th and 7th plates in the 'Guy's Hosp. Reports' for 1843.)

duration, the kidneys present appearances deviating still more markedly from the normal. The cortex has now an opaque white or whitish-yellow colour, both on its surface and on section; and this contrasts very strikingly with the red colour of the pyramids. The organs are sometimes very large, much more so than are ever the reddish-grey or chocolate-coloured kidneys of an earlier period. In three cases the weight of a pair of kidneys was twenty-eight and a half or twenty-nine ounces, and in a fourth Dr Moxon found them weighing within half an ounce of three pounds. On the other hand, it sometimes happens that histologically similar kidneys, still retaining their smooth surface, are of natural size, or even slightly smaller; but such cases are rare exceptions. The shortest time within which we have seen the kidneys assume the "large white" character is five or six weeks; more often the disease has lasted several months. In each of the two cases just mentioned of great enlargement the patient had been ill for fourteen months. On the other hand, in one instance the kidneys were still of a brick-red colour at the end of six months. It is to be remembered that a considerable proportion of cases of tubal nephritis fatal at this stage are of insidious origin and slow development, so that their duration cannot be fixed. And no doubt many cases that have commonly been classed as examples of the "large white" variety of Bright's disease really belong to the lardaceous form. Kidneys from the bodies of syphilitic patients, or of those who had phthisis or other suppurative affections, should never be set down to primary parenchymatous nephritis, until the absence of the lardaceous change has been determined by microscopical examination.

Histologically, the most striking appearance in most specimens of the large white kidney is the accumulation of immense quantities of fatty epithelial cells in the renal tubes. It is this that gives the opaque yellow colour with reflected light; in thin sections, viewed by transmitted light, the tubes often appear quite black. The stroma of the cortex also is closely studded with fat-granules. Hence such kidneys are sometimes spoken of as "fatty," but very improperly, since that designation ought to be reserved for cases in which a fatty change is primary, as in obese persons who also have fatty liver, or in cases of poisoning by phosphorus. Again, it must not be assumed that all large white kidneys are fatty. In those which are translucent and greyish white the amount of fat is often very small.

Dr Greenfield, in his summary of renal pathology in the Sydenham Society's 'Atlas,' speaks of cases, especially in pregnant women, in which the microscope shows that the lesions are almost entirely "interstitial." One distinguishing character of such kidneys is their toughness, almost like that of caoutchouc, contrasting strongly with the soft pulpy texture of those in which the principal changes affect the tubal epithelium. But the fact is that in almost all, if not in all, cases of advanced "parenchymatous" nephritis, interstitial lesions are really present to a greater or less extent. This has been insisted on by Mahomed and Saundby in this country, and in Germany by Weigert, who says that he has for years vainly sought for a specimen altogether free from interstitial changes. They are not uniformly diffused through the renal substance, but (as in many other forms of Bright's disease) consist of patches of nuclear growth, afterwards developing into tracts of connective tissue, in which the nuclei are less numerous. The glomeruli also have their capsules thickened, and pass through every stage in the process of conversion into structureless globular cysts.

(3) *The contracted white kidney.*—If tubal nephritis runs on long enough, the kidneys become shrunken, small, and rough on the surface, though they still (and perhaps always) retain more or less of the whitish-yellow colour. This continues to distinguish them from kidneys affected with the cirrhotic form of Bright's disease; but the distinction is lost in those cases in which organs primarily cirrhotic become the seat of secondary parenchymatous changes. The occurrence of a granular stage as the ultimate issue of nephritis arising from scarlet fever or of any other cause that commonly produces a large kidney, was at one time denied; but it has now been clearly established, and a typical example is figured in Plate 3 of the Sydenham Society's 'Atlas of Pathology.' The case was that of a girl, aged ten, who, rather more than two years before her death, became dropsical as the result of scarlet fever. After four months she recovered, but a year later the face began to swell from time to time, and she died at last with cerebral symptoms. At the autopsy the kidneys were found to be very small indeed, with thick opaque capsules, hard, tough, and puckered on the surface, presenting on section yellowish-grey masses. The records of *post-mortem* examinations at Guy's Hospital contain a good many cases, in young subjects, of a more or less similar kind, the weight of the pair of kidneys being from four to eight ounces. In most of them the history affords no clue as to the date at which the disease had begun. But in some instances it is recorded that there had been an illness, attended with dropsy, several years before death. A case in point, occurring in a young woman of twenty-four, a patient at the London Hospital, is figured in the same Plate of the 'Atlas.' She was said to have been ill for only three months; but her kidneys were found by Dr Sutton to be reduced to about half their normal size, to be "very granular, and of a reddish colour, everywhere mottled with a yellowish or purplish or greyish substance."

Symptoms.—This parenchymatous form of Bright's disease varies in its mode of onset in different cases. When it is due to scarlet fever, or to a definite exposure to cold, it may begin with a rigor and pyrexia.

More commonly, the earliest indication of the patient's illness is the occurrence of *dropsy*. This may first appear in the loose tissue round the eyes, and in slight cases it is especially noticeable before the patient gets up in the morning; the lower eyelid is œdematous, and the conjunctiva also This conjunctival œdema produces "the bright eye" of renal disease, and "the tear that does not run over." Generally dropsy also affects the limbs; and a favourite seat for it is the lower part of the back, as far down as the sacrum—"the lumbar cushion." The external genitals, too, are very apt to become swollen, and the prepuce is sometimes so stretched and twisted as to interfere with micturition. In extreme cases almost the whole body is distended with fluid, so that it has a bloated appearance, while the extreme anæmia which rapidly develops itself gives the skin a pale yellow, wax-like colour.

The appetite is often bad, the tongue furred, and the bowels constipated. Vomiting is frequently a marked symptom, and there may be much headache. Pain in the loins is often altogether absent, but sometimes it is severe, radiating to the groins and down the thighs, and accompanied with tenderness to pressure.

The *urine* in the acute stage is always scanty, and sometimes entirely suppressed. Though the patient is constantly striving to micturate, a few

drops of blood-stained liquid may be all there is to pass. Complete suppression is a very grave symptom, and generally points to a fatal issue, which is seldom long delayed, even if the kidneys should afterwards, to some slight extent, resume their functions. When some ounces of urine are secreted daily, it is commonly of high specific gravity (1025—1030) and of dark colour, more or less red or brown from the presence of blood. Its appearance may often be compared with that of strong tea or of porter. It is turbid, and throws down a chocolate-coloured deposit, containing altered blood-discs, swollen epithelial cells, and casts, some of which are hyaline, while others may themselves be full of blood-discs or epithelial cells.

Albumen is of course always present in the urine when there is blood. But the quantity of albumen is often not so large at this period as it is a little later when the hæmaturia has passed off. The late Dr Mahomed described, in the 'Med.-Chir. Trans.' for 1874, what he termed a *pre-albuminuric* stage, in which, while no albumen can be detected, the guaiacum test reveals the presence of colouring matter of blood and the sphygmograph shows a marked increase of arterial tension. Bartels also mentions cases in which, when scarlatinal dropsy first set in, the urine, though exceedingly scanty, was non-albuminous, and he cites a case of Henoch's in which no albumen could at any time be detected except on the day before death, when the patient was cyanosed and almost pulseless as the result of an attack of convulsions. Commonly the amount of albumen ranges from 2 to 5 per cent. The total quantity passed in the twenty-four hours is said to be from 80 to about 400 grains. The excretion of urea is greatly diminished, falling to half the normal amount, or even much less.

When parenchymatous nephritis ends in recovery in the course of a few weeks—as occurs in the large majority of scarlatinal cases, and in many of those due to other causes—the dropsy and the other symptoms subside, and the urine gradually recovers its normal characters. It is more abundant, of lower specific gravity, and paler; it no longer contains blood, which, indeed, is generally present only at the very commencement of the attack; the quantity of albumen in it becomes less and less, until at length there is none.

On the other hand, when the disease runs on for months, the dropsy continues, or becomes worse. So bloated and anæmic is the appearance of the patient that he is sometimes spoken of as having a "large white" body, as well as large white kidneys. He lies helplessly in bed, his back propped up with pillows, his legs stretched stiffly out before him, or supported by a pillow beneath the knees. The swelling of the external genitals is often extreme. The scrotum looks like a bladder full of water, and is so large that there is no room for it between the thighs; the prepuce is described by Bartels as "curled up like a posthorn." Sometimes the cuticle over some of the distended parts cracks and the dropsical fluid oozes out in such quantities as to soak through the bedding. This may cause considerable tracts to become excoriated, and ultimately to be covered with pale granulations which, when they skin over, give the surface a warty appearance. Or inflammation may set in, attended at first with a crimson redness, like that of erysipelas, and leading to more or less extensive gangrene of the skin and even of the subcutaneous tissues. Erythematous or exfoliative forms of dermatitis are not uncommon, independently of the irritation of urine or exuded serum, or of tension of the integuments (cf. p. 611).

In this more advanced stage of parenchymatous nephritis the state of the urine is very variable. It may still be scanty and of rather high specific

gravity. But more frequently it gradually becomes abundant and pale, and then its specific gravity falls considerably below the normal, to 1010 or 1005, or even lower. It may still be highly albuminous; indeed, the quantity of albumen in it may reach 5 per cent., or even more, so that on the application of heat the whole contents of the test-tube set into a solid mass. On the other hand, as disease progresses this amount of albumen often lessens considerably. Blood in small quantity may be present from time to time, but this is rather exceptional. Casts are commonly found in abundance, some of them hyaline, others containing leucocytes, epithelium, and fat-granules, others completely opaque and granular. The excretion of urea is at all periods of the disease much below the normal amount. Even when the flow of urine becomes more abundant, the total quantity of urea contained in it in the twenty-four hours, instead of increasing, is commonly still less than before. Albuminuric retinitis, in all its forms, is of frequent occurrence (p. 609).

Event.—In some cases of parenchymatous nephritis, at whatever stage of the disease, death occurs as the result of the intensity of the dropsical and other symptoms; especially from accumulation of fluid in the great serous cavities of the chest, or (though very rarely) from œdema of the larynx. In many instances it is the result of œdema of the lungs, or of pneumonia which has commonly more or less of an œdematous character. In others it is brought about by the supervention of acute pleurisy or pericarditis. Many cases, again, end by uræmia; while in a few, failure of the heart, from dilatation of the left ventricle, is the direct cause of the fatal issue.

It can but rarely happen that complete recovery takes place when the disease has lasted many months, still more rarely when it has been prolonged for years. But it is surprising how symptoms will sometimes subside and disappear, and how, even after the patient has been waterlogged and has had alarming uræmic attacks, he may regain what appears to be a fair state of health. A striking instance of the removal of an albuminuria of long standing by treatment is the case of a medical man, aged twenty-six, whose urine after an attack of scarlet fever was continuously albuminous after meals for more than six years. By the advice of Dr Johnson he was strictly dieted, and at the end of nine months the urine became normal, and remained so eighteen months later ('Brit. Med. Journ.,' 1879, ii). In that case, however, the general health remained good all along.

II. Lardaceous Disease of the Kidney.*—From the time of Rokitansky the lardaceous affection of the kidneys has been described as one of the forms of Bright's disease, and Wilks has pointed out that there is good reason for supposing that one of the specimens figured by Bright himself as a "large white kidney" was lardaceous, inasmuch as the liver of the same patient, which is still kept in the museum of Guy's Hospital, presents that morbid change. For a long time the view which prevailed with regard to the relations between lardaceous kidneys and those affected with diffused inflammatory lesions was rather that they were clinically allied as all alike giving rise to albuminuria and to dropsy than that there was any close pathological connection between them. This view doubtless received support from the fact that in the liver and in the spleen the lardaceous change is never associated with diffused inflammation; but even in other

* *Synonyms.*—Rokitansky's Speckniere—The waxy kidney of the Edinburgh pathologists—Virchow's amyloide Nierenentartung—Depurative nephritis of Dickinson.

circumstances neither of those organs seems to be liable to such lesions. The true reason why it is impossible to exclude the lardaceous kidney from Bright's disease is, that the lardaceous change is so commonly associated in the same kidney with the lesions belonging to the other forms of this disease. Whatever the nature of this association, it gives to the lardaceous affection of the kidneys a clinical importance which does not belong to the like affection of any other organ. And therefore the general bearings of that morbid change may be better discussed here than elsewhere.

*The lardaceous degeneration.**—The first step in the study of this disease was the discovery by Meckel of the fact that the tissues affected give a peculiar reaction with iodine, turning a walnut or mahogany-brown colour, whereas healthy tissues remain pale yellow: he thought this due to the presence of cholesterine. Virchow afterwards stated that on the addition of dilute sulphuric acid a more or less distinct blue or purple tint can be detected, to which he attached great significance, regarding it as an indication that the lardaceous material was chemically related to principles of vegetable origin, such as starch and cellulose. But many observers have failed altogether to obtain any blue tint, and most will agree with Ziegler that this reaction is at best imperfect. Unfortunately, it led Virchow to give to lardaceous disease in general the name of "amyloid." In Germany this name is still used, though in France and England it never replaced the older term, which embodies no erroneous theory. It applies not to the fat (*lard*, *lardacé*, a comparison made by Portal) but to the firm, elastic, translucent rind of bacon. It was shown by Kekulé that the "lardacein" (as it is now called) contains nitrogen, and afterwards by Kühne and Rudnett that it is really allied to albumin.†

Tests.—It is best to employ an aqueous solution of iodine and iodide of potassium as a test for the lardaceous material. The cut surface of the organ to be tested must be first washed free of blood, which, if present in large quantity, makes it impossible to speak with any confidence as to the presence or absence of the reaction. The surface is then lightly brushed over with the solution. A very considerable degree of lardaceous change becomes apparent in a few seconds, by the formation of brown or black spots or streaks. If, however, the change is very slight in amount, it may be perceptible only with the microscope, and after the application of the iodine to a thin section. In 1875 Jürgensen made known in 'Virchow's Archiv' the fact that methyl violet, prepared by the action of iodide of methyl upon aniline, gives a beautiful colour-reaction with lardaceous tissues. Those parts which present the lardaceous change slowly become red, whereas the unaltered parts are stained blue. The main advantage of this over the iodine test appears to be the greater definiteness with which the reaction remains limited to certain elements in a complex structure. Thus it is invaluable in determining whether the secreting cells of an organ, or the

* The earliest term applied to this condition was "lardaceous" (*speckartig*, like bacon-rind) by Rokitansky and the Vienna school. In Edinburgh it was named "waxy" by the late Professor Sanders. Budd and other English writers called it "albuminous," or "scrofulous enlargement." Schrant, and afterwards Oppolzer, used the vague epithet, "colloid." Any of these is better than the misleading term "amyloid" (starch-like); but lardaceous is used in France and America, and is adopted in the Nomenclature of the Royal College of Physicians.

† Kühne's plan was very ingenious. Lardacein is insoluble and unaffected by reagents, alkalies, acids, or digestion. He therefore submitted a lardaceous liver to artificial gastric digestion, and when everything else was dissolved away, analysed the residue.

adjacent capillaries, are the seat of the morbid change. Another important point is that it enables permanent preparations to be made and preserved in glycerine. On the other hand, it cannot be used as a rough test in the *post-mortem* room; and even as applied to microscopical sections of the tissues it seems to be in no way superior to the iodine test when the question is merely as to the presence or the absence of the lardaceous change in an organ, or even as to its amount.

Without the addition of any colouring matter, the lardaceous change is plainly recognisable with the microscope in thin sections of an organ. The affected parts of the tissue have a swollen, homogeneous, glistening appearance, which cannot be mistaken by an experienced eye.

Theory.—As to the nature of the process by which lardacein is substituted for the natural proteids of the tissues, nothing is as yet known. Whether this material arises *in loco* as a "degeneration" of the normal structures, or whether it is a "deposit" or an "infiltration" derived from the blood, is still uncertain. Formerly, most writers on pathology expressed a confident opinion in favour of the latter view; now the current of opinion is in favour of the latter. However, in the blood itself it cannot be detected. Another point in favour of its local origin is the fact that hyaline casts that have been retained within the tubes of the kidney sometimes give the reaction with iodine; so that one can hardly doubt that a chemical change occurs in the albuminoid substance of the casts subsequent to their formation. What makes the question a peculiarly difficult one is that in every organ the parts most apt to become lardaceous are not the proper elements of its structure, but the capillaries and the small arteries. On the "infiltration" theory, it is extremely hard to understand why the change should be limited to the vessels in certain organs, and not equally distributed over those of the entire body. Moreover, even in a single organ—such as the kidney—it is apt to be very partial, occurring only in certain glomeruli, or only in some loops of the same glomerulus. Surely this looks more like a local "degeneration." On the other hand, it may be urged that there are obvious analogies between the lardaceous change and the process of calcification, which is due to the deposition of lime-salts from the blood.

Locality.—The lardaceous change has from time to time been met with in strange situations, where its occurrence certainly would not have been anticipated; and what is still more remarkable is that in these cases it has often been absent from its usual seats. As examples may be cited, Burow's case of lardaceous degeneration of laryngeal tumours, Ziegler's case of lardaceous nodules at the base of the tongue, Birch-Hirschfeld's statements as to the presence of lardaceous material in mesenteric glands after enteric fever, and, above all, the curious examples of a lardaceous change in the vessels of the conjunctiva recorded by Sämisch, by Leber, and by Kyber. References to all these observations may be found in a paper by Kyber in vol. 81 of 'Virchow's Archiv.' It has also been shown that by carefully searching the various structures of the body, even when the lardaceous change has its usual distribution, one can often detect its presence in many other parts besides those in which it is commonly recognised.

As an important pathological process, lardaceous degeneration may be regarded as limited to a small number of organs, these being the kidneys, the liver, the spleen, the intestine, the thyroid, and the adrenal bodies; and in the last two, so far as is at present known, it gives rise to no symptoms and has no clinical significance whatever

Ætiology.—Many writers, Bartels for example, speak of scrofula, chronic tuberculosis, and syphilis, as being all alike constitutional diseases which predispose to the development of lardaceous degeneration, probably in consequence of their liability to induce chronic ulcerations of the bones, skin, or the mucous membranes. He also admits, however, that it may show itself as the result of chronic and protracted suppuration apart from constitutional disposition. Now, such a method of statement appears to be incorrect in two important respects. In the first place, it is certain that syphilis in some mysterious way induces the lardaceous change in cases in which formation of pus has occurred only to the most insignificant extent, if at all. In the second place, it is more than doubtful whether "scrofula" or chronic tuberculosis (in other words the presence of tubercle in the lymph-glands or other organs) has any influence in the same direction, apart from its tendency to cause suppurative lesions. We may therefore reduce the known causes of the lardaceous change to two—chronic *suppuration* and *syphilis*—which may of course be combined, but either of which is capable of acting separately. Some pathologists also mention the cachexia caused by ague, but without adequate evidence; and the possibility that the cases might also be syphilitic must not be forgotten. It is of course possible that there may be other causes beside the two above enumerated; for instance, there is some reason for thinking that the lardaceous change occurs in association with Hodgkin's disease without there having been sufficient suppuration to account for it.

In 1876 the author brought before the Pathological Society a tabulated statement as to what appeared to have been the ætiology of 244 cases of lardaceous disease of the viscera, collected for him by Mr Lancaster from the *post-mortem* records of Guy's Hospital over a period of twenty-one years. In 154 there had been prolonged suppuration; 67 of these were cases of phthisis; in 51 there was disease of some joint (generally the hip- or knee-joint) or caries of the spine, or of some other bone; in the remaining 36 there were a variety of affections, amongst them empyema, dysentery, calculous and scrofulous pyelitis, ulcerating, cancerous, or sarcomatous tumours, cystitis from stricture, and bedsores of long standing as the result of disease of the spine. There were also five other cases in which there had, indeed, been suppuration, but in which it seemed doubtful whether this had been enough to afford a reasonable explanation of the lardaceous change. Thus in one instance there had been chronic discharge from one ear as well as from the nose; in another, one testicle had been inflamed and suppurating as the result of a blow two months and a half before death, but with open discharge only for a fortnight; and in a third there was merely tubercular peritonitis with caseous disease of the mesenteric glands. In five or six of the 154 cases that were clearly due to suppuration, this had had a definite starting-point, so that some idea could be formed of the length of time required for the development of lardaceous lesions. One patient had had a carbuncle eight months; another had had pelvic cellulitis for exactly the same period; a third had had a bedsore seven months; a fourth was affected with a sarcomatous growth, which had been discharging for four months. In a fifth case there had been fracture of the spine three months before death, bedsores two months and a half, and also a double empyema; but syphilis was probably also present, inasmuch as the testes presented fibroid changes and there was a scar in the groin. A sixth patient had suffered amputation of the leg three months and a half before death, on account of a compound

fracture with abscess; in that instance it is particularly noticed that both in the liver and in the spleen the lardaceous change was just beginning.

On the other hand, among the 244 cases of lardaceous disease there were 76 in which there was satisfactory proof (either from the history, or from *post-mortem* appearances, or from both together) of syphilis; and in three others there was at least a suspicion of its presence. In about 34 of these 76 cases there was evidence of there having been bone disease or suppuration leaving 42 to be ascribed to syphilis alone. In no instance is it stated that the syphilis had been inherited; but Bartels speaks positively of having seen lardaceous affections in cases of inherited syphilis attended with ulceration of skin and bone.

Of the 244 cases, there are thus left only six examples of lardaceous disease that were not accounted for by the presence either of syphilis or of suppuration; and in some of them the notes of the autopsy are incomplete, the state of the testes, in particular, being unrecorded. Now, it seems far from unlikely that in each of these six cases the cause of the lardaceous change was really syphilis. When we consider how slight and accidental are often the lesions on which we rely as proofs of the presence of syphilis in the dead body it appears almost certain that there must be other cases in which no syphilitic lesions were observed, but in which syphilis was really present. Six cases among 244 are not perhaps too numerous to be covered by this plea; even if we ought, in fairness, to add to them some of the five other cases in which, although suppuration had occurred, there was a doubt whether it was adequate to the production of the lardaceous change.

Age and sex.—The period of life at which lardaceous lesions due to syphilis are most apt to occur is from thirty-one to forty. At Guy's Hospital there have been fourteen fatal cases in persons between those years against six in persons between twenty-one and thirty. As the result of protracted suppuration, on the other hand, the affection has been slightly more frequent during the earlier period; it occurs, too, in adolescents, and even in children; there has been one case at Guy's Hospital in a boy only four years old. Above the age of fifty lardaceous disease is decidedly uncommon, but we have had one instance of it in a man of sixty-five.

Among our cases male patients have been more numerous than females in the proportion of two to one.

Anatomy of the lardaceous kidney.—The appearances presented by a kidney affected with this disease vary widely in different cases. Sometimes the lardaceous degeneration of its vessels is the only change in the organ discoverable, whether by the naked eye or with the microscope. This, however, is only the case when the immediate cause of death has been some other disease; generally one of the various maladies to which, as we have seen, the lardaceous change is commonly secondary. No instance can be recalled in which, at this stage of the renal affection, the patient has succumbed to cachexia from the simultaneous development of the lardaceous change in several organs. How rare it is in the *post-mortem* room for a kidney to be seen which presents no other lesions than the lardaceous change, may appear from a collection of more than sixty cases of lardaceous kidney taken from our records at Guy's Hospital; among them there seem to be only three or four uncomplicated examples.

A kidney thus affected is of the natural size and looks smooth and healthy, except that a keen eye may perhaps perceive the glomeruli to be somewhat more

distinct and more translucent in appearance than is natural. On the addition of iodine the change is generally most marked in the glomeruli; and sometimes it is entirely limited to them. Nor does it affect all of them at once, being, on the contrary, often confined to a few of them only, and even in the same glomerulus to one or two of its coils. Not unfrequently the afferent arteries are involved as well, their middle coat being the first part to suffer. But sometimes the reaction is obtained, not in the cortex, but in the straight vessels of the pyramids. And sometimes it seems to begin in both the cortex and the pyramids simultaneously. When the change is very far advanced, the capillaries round the renal tubes are also affected, as well as the basement membrane of the tubes, and perhaps the epithelium lining them.*

In the great majority of instances lardaceous kidneys are the seat of other lesions likewise, by which they are greatly increased in size. In about a quarter of the sixty cases the weight of the two organs together was from twenty to twenty-seven ounces. The appearance of such kidneys is sometimes very peculiar, and fully justifies their being termed *waxy* or *butternieren*. Their surface is smooth and pale yellow, with conspicuous stellate veins; their section is shining and polished, of a semi-translucent grey or yellowish colour, sometimes showing obviously the grey swollen glomeruli, sometimes mottled more or less thickly with creamy opaque spots and streaks, where fatty granules are present in abundance. What causes the enlargement of the organs in these cases is undoubtedly the accumulation of inflammatory products, not only in the tubes, but also in the interstitial tissue. But as to why such a diffused nephritis should arise there have been all possible differences of opinion.

Cohnheim is inclined to believe that it is an independent result of the same cause which produces the lardaceous change, and which we shall presently find to be generally either syphilis or prolonged suppuration. But the association of the two morbid processes occurs far too frequently to admit of such an explanation, especially as no pathologist will maintain that diffuse nephritis, apart from the lardaceous change, is a common result of syphilis or of suppuration.

The known ætiology of the lardaceous change clearly excludes the view of Cornil and Ranvier that it is preceded by the nephritis.

We have therefore no alternative but to suppose that in some way lardaceous degeneration must cause the nephritis. And the objection that no similar inflammatory process is found to arise in a lardaceous liver or spleen loses its force when we consider that neither of these organs is subject, under any circumstances, to an affection comparable with Bright's disease.

Weigert observes that the peculiar position of the glomeruli, between the arterioles of the renal cortex and the capillary blood supply to the tubes, may well render any obstruction to the flow of blood through the glomeruli the cause of damage to the tissue beyond. He believes that the first result of the lardaceous change is to produce fatty degeneration of the epithelium, and that this degeneration, whatever its origin, is apt to set up diffused interstitial nephritis. But certainly fatty degeneration of the kidneys is not uncommon, without the least trace of consecutive nephritis.

* Dr Dickinson's 8th plate gives an excellent picture of the appearance of the kidney to the naked eye, and his 9th and 10th of its histology; the latter is also well illustrated by Dr Stewart's 5th and 6th plates.

Sometimes lardaceous kidneys are found after death to be smaller than natural. This occurred in about one out of five or six of the sixty cases collected from our *post-mortem* records. As a rule the loss of size was not very obvious, the two organs weighing together not less than seven and a half or eight and a half ounces; but in one instance the weight (of what happened to be a "horseshoe" kidney) was only three and a half ounces. There was always, however, much irregularity and shrinking of the surface —a more or less markedly "granular" condition; and the loss of substance was no doubt far more considerable than was indicated by the weighing machine, inasmuch as the kidneys still retained large quantities of inflammatory exudation, as well as the lardaceous material itself. Such wasted organs clearly represent the most advanced stage of the lardaceous affection, and it is fair to assume that its course has been more than usually slow and protracted. But there is no evidence that kidneys that ultimately become thus reduced in size have passed through an earlier stage in which they were enlarged. Some observers have tried to account for such cases by supposing that the kidneys were already contracted and granular (from gout or some other cause) before they began to be affected with the lardaceous change, as the result of syphilis or protracted suppuration. And there seems to be no doubt that in 1866 we had at Guy's Hospital an instance of the accidental association of the two morbid processes in the case of a woman who died at the age of forty-five of cerebral hæmorrhage, and whose kidneys, besides being lardaceous, were granular and full of minute cysts. Weigert remarks that there ought not, with the microscope, to be any difficulty in distinguishing mixed cases of this kind, since the glomeruli would doubtless be shrunken and atrophied (after the manner described at p. 642, *infra*), whereas in the most advanced stages of a lardaceous kidney they continue to present the appearances characteristic of that affection. The hypothesis of an independent granular change could, however, be hardly applied to two of our cases, each of which occurred in a young man of twenty-four, for at that age such a change in the kidneys is almost unknown; in one of these cases the organs weighed seven and a half, in the other six and a half, ounces.

Symptoms.—The characters of the urine secreted by lardaceous kidneys were first studied by Traube in 1858; and his account of them has been confirmed by the labours of Grainger Stewart and others who have since taken up the subject. But although cases doubtless sometimes occur, in which the state of the urine may alone suggest to an experienced observer the idea that the renal affection from which the patient is suffering is lardaceous, and not one of the other forms of Bright's disease, such cases are rare. Indeed, considering how constantly the lardaceous change becomes associated with other lesions in the kidneys, and how diverse these lesions may be, it is very unlikely that the urine will always present the same characters. The cases in which the influence of the lardaceous affection should be most clearly traceable are, of course, those in which it is uncomplicated with inflammatory changes in the renal cortex. But there is much force in Cohnheim's remark, that the existence of such "simple" lardaceous kidneys can scarcely ever be determined, except in patients who have fatal disease of other organs which causes them to be wasted and anæmic, and to suffer from pyrexia, diarrhœa, and other symptoms that must in themselves disturb the balance of the nutritive changes in the body. Thus Bartels appears to be right in declaring that there is no evidence that the lardaceous change in itself interferes with the excretion of urea; if this is diminished it is a result

of a general lowering of the activity of the chemical processes throughout the body. Indeed, the effect of this change in the glomeruli appears rather to be that their walls are rendered more permeable than before.

With regard to the *quantity* of urine secreted by lardaceous kidneys, different writers make very different statements. According to Grainger Stewart, unless an extreme degree of nephritis is present, it is excessive, ranging from fifty to two hundred ounces daily. He also maintains that an increased flow of urine is in many instances of great clinical importance as an early symptom of the lardaceous affection, preceding albuminuria.* Bartels, however, gives much smaller average amounts—from fifty to sixty or seventy-six ounces—and he expressly remarks that there is never such a degree of polyuria as occurs (for example) in some cases of granular disease of the kidneys. He mentions a case in which the average daily secretion amounted to less than seventeen ounces. Wagner also declares that in the majority of cases—almost constantly in the last few weeks, but sometimes in the whole course of the disease—the urine is more or less scanty, sometimes with intervals of a few days in which it is normal or increased in quantity. No doubt these discrepancies of opinion depend mainly upon the degree of care taken by different observers to recognise an underlying lardaceous change in kidneys that would formerly have been set down as examples of parenchymatous nephritis. Wagner, for instance, says that in the *post-mortem* room the lardaceous form of Bright's disease is seen more frequently than any other —"much more frequently than chronic parenchymatous nephritis or cirrhosis of the kidney;" and the author must confess that his experience at Guy's Hospital accords very much with this.

The specific gravity of the urine varies with the quantity; it may be as low as 1003 or 1005, or it may occasionally reach 1025. For several days before death the urine is often very scanty, and of high density.

In all but exceptional cases *albumen* is present. Lecorché has maintained that so long as the lardaceous change is uncomplicated with nephritis the urine is non-albuminous. But Bartels denies that this is correct; and one at least of our cases at Guy's Hospital affords proof to the contrary. On the other hand, there can be no doubt that Bartels himself goes too far when he declares that albuminuria is always present, except perhaps at the very commencement of the affection, when the change in the vessels is just beginning. Many observers have noted that even in cases in which albumen is sometimes to be detected, it may at other times be absent from the urine. And Cohnheim speaks of having made autopsies in several cases in which albuminuria was said to have been altogether wanting. The quantity of albumen varies greatly. There may at first be only a little; but the rule is that the quantity is large, and it may reach from 1 to 3 per cent. Whether or not the presence of such considerable amounts of albumen may be taken as evidence that there is nephritis as well as the lardaceous change seems as yet to be doubtful.

Blood is very rarely present, even in small amount. The statement made by Senator some years ago that the presence of paraglobulin in large quantities is characteristic of this rather than of other forms of Bright's disease, has not been confirmed since the introduction of Hammarsten's method of separating paraglobulin from serum-albumin (p. 595).

In some exceptional cases there may be found in the urine glistening

* Dr Johnson supports him in both these statements, and so does Dr Dickinson, who puts the daily amount at from 50 to 90 ounces.

epithelial cells which turn reddish brown with iodine, from having undergone the lardaceous change before being shed. The discovery of them is probably conclusive as to the original nature of the renal affection.

The urine is generally pale and transparent, throwing down no deposit, or at most a few *hyaline casts* and epithelial cells.

On the other hand, when there is much nephritis, the urine may be high coloured, and may give an abundant precipitate containing lithates, as well as numerous hyaline and granular casts, to which epithelial and fatty cylinders may be added (see Dickinson's pl. vii, fig. 2).

The other symptoms which accompany lardaceous disease of the kidneys are very variable and uncertain. It must not be supposed that cachexia and wasting are necessarily present. Bartels speaks of having had patients who were able to follow laborious occupations at a time when there was unequivocal evidence of the existence of this renal affection. And one of our cases at Guy's Hospital was that of a man, aged thirty-six, who was admitted with a fractured spine, the result of a fall while he was carrying a sack of barley on his shoulders; lardaceous changes were found in the liver, the spleen, and the adrenals, as well as in the kidneys, which were enlarged, and weighed sixteen ounces.

Dr Stewart lays stress upon a pasty or waxy complexion, with a little pigment about the eyelids, and distension of small blood-vessels upon the cheeks, as being suggestive of this form of renal disease.

Dropsy is sometimes wanting as a symptom of lardaceous disease of the kidney, especially when there is diarrhœa from a coincident affection of the intestinal mucous membrane. But this symptom is frequently present in an extreme degree, affecting the face and the arms and the whole body, exactly as in cases of the "large white kidney" for which (as we have seen) cases really lardaceous have so often been mistaken, both at the bedside and after death. Bartels declares that when dropsy does occur it is ordinarily confined to the lower limbs and to the abdomen. But though reports of some few cases, taken by themselves, might seem to support this statement, our experience on the whole at Guy's Hospital does not bear it out. When such a limitation of the dropsy is observed, it is an evidence that the immediate cause of the effusion was failure of the heart's action (cf. *supra*, pp. 606, 607).

Now, it has been noticed by all recent writers on the subject that the heart is seldom found enlarged in cases of lardaceous disease of the kidneys. Cohnheim asserts that even when there is wasting and shrinking of the kidneys the heart invariably remains small unless the lardaceous change in them was preceded by the cirrhotic. Among over sixty cases observed at Guy's Hospital there were four in each of which the heart was found after death to weigh from eleven to thirteen ounces; and in two of these the kidneys also were enlarged, weighing fourteen ounces and eighteen ounces respectively. The frequent absence of cardiac hypertrophy seems to be sufficiently explained by the cachectic and anæmic condition of the immense majority of those whose organs present the lardaceous change; and it does not prove (what is very unlikely) that when interstitial nephritis affects lardaceous kidneys it has no effect upon the heart. But if this view is correct, and if there is really some degree of increase of arterial tension, there is certainly nothing to be wondered at in the occurrence of cardiac dropsy, as a result of the failure of the heart to respond to the increased calls upon it.

Retinitis and *uræmia* are said to be rarely observed in cases of lardaceous affection of the kidney. But in three cases at Guy's Hospital death was ushered in by convulsive seizures. One patient, who had been in the hospital nine months previously with dropsy, was readmitted four days before death in a state of collapse, with cold and blue extremities, the result of severe diarrhœa and vomiting. In several instances acute peritonitis, or acute pericarditis, was what immediately brought the disease to a fatal termination.

Diagnosis.—From what has been stated with regard to the clinical effects of the lardaceous affection of the kidneys, it is evident that, apart from other circumstances, neither the characters of the urine nor any other symptoms can be relied on to suggest its real nature, as distinguished from the other forms of Bright's disease. What, in fact, generally enables us to make a diagnosis is either (1) that indications of lardaceous change are presented by the liver, or by the spleen, or by both; or (2) that one or other of the known causes of this change is present. The liver is always enlarged when the seat of well-marked lardaceous disease; but diffused lardaceous infiltration of the spleen and lardaceous disease of its Malpighian follicles forming the "sago-spleen" may both be well marked without the bulk of the organ being increased. It must therefore be borne in mind that the spleen does not always become increased in size. Further, even when both spleen and liver are considerably enlarged, there may be no possibility of detecting the enlargement in patients who are fat or flatulent or dropsical. Knowledge of the causes of the affection is therefore all-important.

Prevention and treatment.—In regard to the *prevention* of lardaceous affections, it clearly is of great importance that all inflammatory diseases likely to lead to protracted suppuration should be so treated as to shorten their course as much as may be. And experience may perhaps hereafter show that in cases of syphilis it is desirable, on this account, as well as with the direct object of eradicating the obvious symptoms of the disease, to continue the administration of mercury and of iodide of potassium longer than might otherwise seem needful.

It must not, however, be supposed that in all the cases which after an autopsy are positively set down as dependent upon syphilis the presence of this disease has been, or could have been, recognised during life. On the contrary, there are a great many instances in which it is only by the discovery in the dead body of such lesions as fibroid degeneration of the testes, or small gummata or cicatrices in the liver, that the syphilitic character of the case is made out. In this respect there is a wide difference between the two great causes of the lardaceous change. For the occurrence of suppuration to an extent adequate to produce lardaceous affections can hardly ever be overlooked, except perhaps when it takes place from the intestinal canal (as in dysentery) or in those exceptional cases in which there is no external discharge of pus at all, but merely a large deep-seated abscess, the contents of which undergo slow inspissation and caseation.

It may possibly be thought that the clinical diagnosis of lardaceous disease of the kidneys from other forms of Bright's disease is, after all, a matter of no great moment, since it is to the diffused nephritis which is present that most of the symptoms are really due. But there is some reason to believe that, both as regards prognosis and with a view to treatment, it is important not to overlook the possible presence of

the lardaceous change. When general dropsy sets in, the downward progress of cases with lardaceous kidneys is often more rapid than that of other forms of Bright's disease. On the other hand, it not infrequently happens that albumen is detected in the urine of syphilitic patients several years before serious symptoms appear. There is also satisfactory evidence that such cases, even when dropsy occurs, sometimes end in recovery, the urine gradually resuming its normal characters. Two instances of this are recorded by Dr Dickinson in vol. xxx of the Pathological Society's 'Transactions.' One of his cases is that of a patient who contracted syphilis in 1861, and who in 1874 began to suffer severely from periosteal nodes; soon afterwards his legs became œdematous, and his urine loaded with albumen, so that the coagulum filled one fourth or one half of the test-tube. Under systematic treatment, assisted by a residence on the Riviera during four winters, the disease slowly subsided, and by June, 1879, the urine was quite natural and the patient himself apparently in a state of perfect health.

As to what changes take place in the kidneys when recovery occurs, nothing is yet known. It seems not impossible that the glomeruli and other vessels that were lardaceous may return to a normal condition, at any rate in cases in which the albuminuria is only of short duration. But in protracted cases, such as Dr Dickinson's, it is far more likely that the affected parts of the kidneys shrink and undergo atrophy, and that parts which had escaped the disease take upon themselves the whole renal function, possibly undergoing hypertrophy to enable them to perform it.

In practice the only way in which one can escape the danger of overlooking among one's cases of Bright's disease some of those that are lardaceous, is to give the iodide of potassium to many patients in whom the cause of their renal affection is obscure, especially if there is reason to suspect dissipated habits at an earlier age.

Cirrhosis of the Kidney.*—The view with regard to this form of Bright's disease generally held by English pathologists is that those cases which are secondary to parenchymatous nephritis or to lardaceous degeneration should be distinguished from those which are the results of a primary morbid process. One proof of the reality of this distinction is afforded by the difference in the appearance of granular kidneys at different periods of life. In autopsies on children, and even in adults under the age of twenty, twenty-five, or perhaps thirty years, we do not meet with the red or brown granular kidneys which are so frequent in middle-aged and in old people. When contracted and granular kidneys are seen in young subjects, they are full of opaque whitish-yellow spots or patches. It is true, doubtless, that these tend to become fewer and less conspicuous as the affection becomes more and more advanced; so that it is quite possible that in some exceptional instances they may disappear, and leave the organ in a condition like that which is usually met with at a later age. But even if the means of distinguishing them should thus sometimes fail, it would not at all follow that we should forget the obvious differences between the two diseases which are presented by the large majority of cases.

* *Synonyms.*—Chronic interstitial nephritis—Red granular atrophy—Granular degeneration, producing the small red kidney of Bright—Chronic gouty nephritis.—*Fr.* Maladie de Bright, forme chronique aux reins ratatinés. *Germ.* Schrumpfniere (the contracted kidney).

On the other hand, in older patients the criterion afforded by the appearance of the kidneys not infrequently ceases to be applicable. The reason is that when a part of the renal substance has been destroyed by cirrhosis the remainder is apt to become affected with the parenchymatous change. Thus the records of *post-mortem* examinations at Guy's Hospital furnish no fewer than thirteen cases in which the kidneys of patients who had been the subjects of gout, and whose joints contained urate of soda, were wasted and granular, but at the same time whitish yellow in colour. Of most of these cases the clinical history given in the *post-mortem* notes is too imperfect to enable one to determine satisfactorily the period at which the second affection supervened. But in 1873, a woman, aged forty-four, a gin-drinker, died after an illness of seven weeks' duration, which she said began one day with pains in the loins while she was working in a cold wash-house. On the following day the face was swollen, vomiting then set in, and afterwards the legs and the abdomen became swollen. The urine was albuminous, of sp. gr. 1012. On *post-mortem* examination the kidneys were found mottled with yellow; but they weighed only eight ounces, they were granular on the surface, and their cortex was much narrowed. The arteries were rigid. The heart weighed twelve ounces, the left ventricle being hypertrophied.

No doubt the pathologist, if he has no clinical history to guide him, is unable to distinguish such "mixed" cases as these from cases of primary parenchymatous nephritis in its most advanced or granular stage; though even without any history he may be guided to the right conclusion if he discovers the gouty origin of the disease from the presence of urate of soda in the joints. But in this country the "mixed" cases after all form an insignificant minority in comparison with the very numerous cases in which the kidneys are purely cirrhotic.

Ætiology.—What proportion of cases of renal cirrhosis are associated with gouty deposits in the joints it is impossible to say, for unfortunately the joints are still too often forgotten in *post-mortem* examinations. But we have very often found the joints affected in the bodies of those who were not known to have had any gouty attack. At the meeting of the International Congress in 1881, Dr Ord stated the results of autopsies made at St Thomas's Hospital, on twenty-four or twenty-five cases of granular disease of the kidneys; in sixteen gouty deposits were present in the joints, in eight or nine they were absent.

It is not unlikely that the rarity of gout in Germany brings with it a corresponding rarity of renal cirrhosis, and this may be the chief reason why some German writers fail to recognise the latter affection as distinct from other forms of Bright's disease. But they speak of a "senile atrophy," which they regard as devoid of clinical importance; and probably under that name they put aside many of the less marked instances, especially when the cause of death is not obviously dependent upon the state of the kidneys. Few pathologists would admit that in this country the kidneys are naturally liable to any purely senile change, except slight shrinking, corresponding with the loss of weight in the tissues generally that comes with advancing years. At the same time we may admit that granular degeneration of the kidneys is associated with vascular and other changes which belong to the latter periods of life, and in the exceptional cases in which it occurs under thirty-five years of age there may often be found other indications of premature old age.

Next to gout, the best known cause of cirrhosis of the kidneys is chronic poisoning by *lead*. The disease is accordingly seen in painters, in printers, and in type-founders, and sometimes in those whose tissues have accidentally become impregnated with the metal. But lead-poisoning also produces gout, and therefore it is difficult to prove the extent of its direct effect in leading to renal cirrhosis.

Most observers think that excessive indulgence in *alcohol*, altogether apart from its tendency to set up gout, is a common cause of granular atrophy of the kidneys. Although Dr Dickinson gives reasons for doubting this conclusion, the general impression is strongly in its favour. The reports of *post-mortem* examinations at Guy's Hospital do not bear out the statement sometimes made that the renal affection is frequently found in association with cirrhosis of the liver, in spite of the apparent resemblance between the two diseases from an histological point of view ('Guy's Hosp. Rep.,' 3rd series, vol. xx, p. 196). Moreover, if lead produces gout, so does drink. Perhaps one of the best arguments in favour of intemperance leading to the more chronic forms of Bright's disease is that it has been so generally assumed without argument by men of experience, including Bright himself. There is no belief, as there is no evidence, that syphilis is a factor.

Chronic interstitial nephritis, secondary to affections of the excretory apparatus of the kidney, will be treated separately as *consecutive* cirrhosis (p. 648).

Age and sex.—This form of Bright's disease is almost unknown in youth and early adult life. Out of 121 cases at Guy's Hospital death occurred in eighteen between 31 and 40 years, in thirty-nine between 41 and 50, in thirty-six between 51 and 60, in twenty-four between 61 and 70, in four between 71 and 80. Below the age of 30 typical instances are rarely, if ever, met with, but several of the patients between 35 and 40 had suffered from gout, and presented the disease in a perfectly characteristic form. These figures correspond pretty closely with those given by Dr Dickinson. They are, of course, very unlike those of Wagner, who mixes up together all forms of "contracted kidney." Eichhorst, however, gives 40 to 60 as the usual age.

The proportion of males to females in the Guy's Hospital cases was almost exactly as two to one; in Dr Dickinson's cases it was 165 to 85.

*Anatomy.**—In its earlier stages renal cirrhosis is by no means a conspicuous morbid change. The kidneys may be of natural size and colour; and beyond the fact that the normal arrangement of the pyramids of Ferrein is no longer visible upon the cut surface of the cortex, and that the capsule is thick and too adherent, there may be nothing to suggest disease, so that one is sometimes surprised to find with the microscope to what an extent the cortex has been destroyed.

In advanced stages the case is very different. The capsule is greatly thickened, and cannot be stripped off without the cortex tearing and coming away with it. The surface of the kidney is covered with little projections or granulations, which, in uncomplicated cases, are of a red or reddish-grey colour. Hence the phrase "raspberry kidney" applied to this granular degeneration. They no doubt consist (like the granules of a cirrhotic liver) of portions of the cortex which are less altered and wasted than the rest. Kelsch and Charcot maintain that they correspond in position with the summits of the "medullary rays" or columns of straight tubes that traverse the cortex,

* The following account is based upon notes of considerably more than 100 examples of primary and uncomplicated renal cirrhosis inspected by the author.

the depressions between them answering to the intervening tracts of convoluted tubes; but Weigert and Wagner dispute this statement. The reduction in the thickness of the superficial parts of the cortex is often extreme. The bases of the pyramids may be separated from the surface of the kidney by a layer of tissue only a line or so thick. The inter-pyramidal portions of the cortex are not generally wasted to the same degree; indeed, in the author's belief, they undergo a compensatory hypertrophy during the early stages of the process. The weight of the two kidneys is often reduced from eight or nine ounces to five, four, three, and sometimes even to two and a half ounces. The smallest pair of kidneys we have seen at Guy's Hospital were found by Dr Wilks to weigh thirty grains short of an ounce and a half. Yet neither the diminution in size, nor the loss of weight, gives an adequate idea of the destruction of the renal cortex; for the pelvis is proportionately wider than in the healthy organ, and is filled with what appears an excessive amount of adipose tissue, over which the substance of the kidney is spread out as a thin shell.

Histologically, the tubular structure of the renal cortex is found to be replaced by connective tissue in various stages of development. At first there is merely a "small-cell infiltration," which forms foci scattered here and there through the organ, especially round the capsules of the glomeruli but with prolongations between the tubes immediately adjacent to them. Gradually the beautiful pattern of the renal cortex is disturbed. The tubules are squeezed here and dilated there; they lose their epithelium and become obliterated, or converted into minute cysts. The glomeruli are at first less affected than the rest of the vessels, and the Malpighian capsules less than the tubules, so that they may sometimes seem little altered, though crowded together by the atrophy of the tissue between. The intertubular blood-vessels and lymphatics, with remains of shrunken tubes, form the elements of the hard and bloodless new tissue. Ultimately the exuded leucocytes undergo conversion into new tissue, in which there are generally very few blood-vessels, though these are sometimes remarkably wide. More or less numerous relics of tubes are usually to be seen embedded in this tissue, and are filled with cells unlike normal renal epithelium, or with hyaline casts. There are also areas in which the tubes are comparatively unaltered, except that they are too wide and patent, with flattened epithelium.

But sometimes, in extensive tracts, hardly a trace of renal structure can be detected. Here the glomeruli and Malpighian capsules are more or less completely destroyed. The capsules often become thickened by the formation of concentric layers, giving them a fibrillated appearance. The tufts degenerate into a structureless material, containing only a few scattered nuclei. By the shrinking of the intervening tissue these "glassy globes" are often drawn close together, so that a large number of them are seen in the same microscopic field. Ultimately nothing may be left but round translucent masses, of which the diameter is not more than one half or one third of that of the normal glomerulus. On the other hand, in those parts of the kidney which still retain a tubular structure the glomeruli may be considerably larger than natural.*

It is held by some pathologists that degeneration of the glomeruli is the primary change in the tissue of the kidney in this disease, and that it

* For instructive figures illustrating renal cirrhosis see Dr Dickinson's 5th plate, Dr Coats's fig. 274, and figs. 65 and 66 in Dr Woodhead's 'Practical Pathology.' Also Dr Saundby's and Dr Greenfield's plates in the 'Path. Trans' for 1880, with their comments

is itself dependent upon an *arteritis obliterans* affecting the smaller branches of the renal artery in the cortex, and even the afferent vessels of the tufts. The thickening of the larger arterial twigs, indeed, constitutes one of the most obvious morbid appearances in a red granular kidney; they stand out upon the cut surface of the organ with patent mouths, like so many little quills. Thoma has shown ('Virchow's Archiv,' 1877) that, when a glomerulus becomes obsolete, its afferent artery may remain pervious, and may open straight into the efferent vessel or into the interlobular capillary network.

In many cases the histological characters of a cirrhotic kidney are further complicated by the presence of innumerable minute *cysts;* certain observers describe cases of this kind under a separate name as "micro-cystic" kidneys. The immense majority of them are invisible by the naked eye, but some may be of all sizes up to that of a millet-seed, or even of a pea. They may make up by far the larger part of a microscopical section. Their contents are often a yellowish-brown jelly-like substance, which can be turned out of the larger cysts as a solid mass. With regard to their nature there was at one time some controversy. Mr Simon suggested that they might be formed out of overgrown epithelial cells; others have thought that they arose out of Malpighian capsules. But the former mode of origin seems very improbable, nor can any analogy be cited in support of it; the latter would not account for more than a fractional part of the numberless cysts that are often present. Accordingly, all pathologists are now agreed that they consist of portions of tubes that have become cut off, and have assumed a spherical shape. Not only are they sometimes arranged in rows, like beads in a necklace, but intermediate forms are often met with—cylindrical cavities, with constrictions here and there in their course.

Another appearance that is observed in cirrhotic kidneys is due to the deposition of *lithic acid* or of its salts in the renal tissues. It consists in the presence of minute whitish-yellow grains scattered through the cortex, or arranged in lines in the pyramids, the summits of which occasionally appear to be encrusted by them. Some of these deposits are amorphous, some are made up of bundles of acicular crystals. They appear to lie partly between the tubes, partly in their interior. Dr Garrod seems to think that such deposits are the results of gout, and that, acting as foreign bodies, they produce albuminuria and set up the renal disease in association with which they are found after death. But they are found in cases in which there is no other evidence of gout, and they are commonly seen in Germany, where gout is rare.

The urine.—In the slighter degrees, or in the earlier stages, of cirrhosis of the kidneys, the urine may be normal in every respect. Dr Grainger Stewart, for instance, relates a case of a man of sixty-five, who died of phthisis, and who passed forty ounces daily, the specific gravity being 1020. But when the disease is advanced, the quantity of urine is often greatly increased and its specific gravity is very low.

The *quantity* amounts to a total of 70 to 200 ounces daily; in a case recorded by Bartels it was measured on a single occasion from 8 p.m. to 8 a.m., and was found during that period to reach 210 ounces. It is commonly secreted more abundantly at night than in the day. Bartels had the urine of one of his patients measured for twenty-six days, that passed from 10 p.m. to 7 a.m. being separated from that passed from 7 a.m. to 10 p.m.; during the nine hours of night the average quantity

was found to be seventy-seven ounces; during the fifteen hours of day only forty-eight ounces.

The specific gravity varies from 1004 to 1010. The urine is faintly acid, pale and clear, depositing no sediment, or only a very slight cloud.

It usually contains only a very small quantity of *albumen*, less than ·5 per cent; the whole amount of albumen in the twenty-four hours is said by Wagner to be not more than from twenty to ninety grains. Indeed, unless care be used in applying the tests, the presence of albumen may be altogether overlooked, even when the urine really contains it; after pouring nitric acid through the urine one must not forget to allow time for the opalescent zone to appear. But it may happen that albumen is entirely absent for days or weeks together. Or the urine passed during the night may be constantly free from it, while it is as regularly present in that passed during the day. That the urine is ever ex-albuminous throughout the whole course of the disease seems to be extremely improbable. There have been cases in which no albumen has ever been detected, but the doubt is whether the urine has been tested sufficiently often and carefully. When albumen is more or less constantly present it is often more abundant in the day urine than in the night urine; and in that passed after meals than in the urina sanguinis of the early morning, which the patient is often told to bring. This is the case even when the patient is kept always in bed.

Casts are often entirely absent; when any are found they are commonly hyaline and narrow, but sometimes opaque and granular.

The amount of *urea* excreted daily seems, according to recent observations, to be not so deficient as used to be supposed. It is true that the proportion in a given quantity of urine is generally not more than 1 or 2 per cent., but so abundant is the urine that Bartels found in four of his cases that the daily average of urea ranged from 296 to 522 grains.

Gouty persons, who have been acccustomed to pass high-coloured urine, becoming thick with urates, are often deluded by the idea that the kidneys are performing their functions much better than before, when the secretion becomes pale and clear as the result of the development of cirrhotic changes. Again, when the flow is much increased, with consequently great thirst, patients sometimes take alarm and seek advice for what they imagine to be diabetes. When the urine is tested and no sugar is found in it, this supposition is of course negatived. But, as observed at p. 562, it sometimes happens that if albumen happens for a time to be absent, the diagnosis of *diabetes insipidus* is wrongly given. Even when the quantity of urine passed is not noticed to be excessive, the patient may be obliged to get up out of bed three or four times every night to micturate. He sometimes will complain of this, but often one has to ask whether it is so. If nocturnal irritability of the bladder has existed for a considerable time, it may be fairly inferred that the renal affection is of at least as long standing.

Other symptoms.—The character of the *pulse*, and the state of the *heart*, form very important elements in the diagnosis of cirrhosis of the kidney, especially when no albumen is found in the urine. These points have been already fully discussed at p. 612, *et seq.* All that is needful to add here is that the hypertrophy of the left ventricle, the thickening of the smaller arteries and the high tension of the pulse, common in some degree to most forms of Bright's disease, reach the fullest and more constant development in the typical cases of chronic granular degeneration.

Clinical forms.—The disease comes under observation in several different ways :

1. Some patients only complain of *weakness and exhaustion ;* and the most conspicuous physical change discoverable in them is that they are anæmic and wasted, with flabby muscles. A man, aged fifty-nine, with what proved to be chronic Bright's disease, was so pallid that his disease was set down during life as idiopathic ("pernicious") anæmia.

2. In certain patients the chief symptoms are *gastric or intestinal ;* uncontrollable vomiting, or diarrhœa, or both together. It is especially in such cases that the breath is sometimes horribly fœtid, as mentioned by Bartels. Vomiting is sometimes of a uræmic character and of serious import. Diarrhœa towards the end of a chronic case is often uncontrollable, and depends upon intestinal catarrh with œdema of the mucous membrane. Occasionally ulcerative colitis is present and is the immediate cause of death.

3. In a good many instances what brings the patient under medical observation is the occurrence of *acute pneumonia*, *pleurisy*, or *pericarditis.* Peritonitis, which is not uncommon in parenchymatous nephritis, is scarcely ever seen as a complication of renal cirrhosis. The acute thoracic inflammations are often rapidly fatal, and pericarditis, when it occurs in a case of kidney disease, is attended with grave symptoms, and dangerous. But it may sometimes give rise to but little disturbance of the general health, and subsides after a while more or less completely. Even when it is still active at the time of the patient's death, the amount of lymph exuded is often but small, so that it is not always clear whether it has brought about the fatal issue. Purulent pericarditis is not uncommon.

4. *Cerebral hæmorrhage* is very often the cause of death in renal cirrhosis, and in many cases it is not preceded by any symptoms that are recognised as indicating that the patient is seriously out of health, or affected with any organic disease. Sometimes the occurrence of hemiplegia is due, not to extravasation of blood, but to softening of a part of the brain-substance, as the result of arterial changes.

5. *Uræmia* is of less frequent occurrence than in tubal nephritis. Among 120 cases at Guy's Hospital only fifteen or sixteen ended thus. Three of them occurred in patients between thirty-one and forty, six between forty-one and fifty, two between fifty-one and sixty, two between sixty-one and seventy, two between seventy-one and eighty. The statement commonly made that this form of kidney disease is the one above all others in which uræmia is apt to occur, appears to be traceable to the fact that the advanced stages of parenchymatous nephritis have been so generally mixed up with it. Headache, giddiness, and other cerebral symptoms are often complained of ; but such symptoms appear in many cases to be dependent upon the diseased state of the intracranial arteries rather than upon the condition of the blood. In 1880, a man aged forty was admitted into Guy's Hospital for pain and heat of the head. He was found to have albuminuric retinitis ; afterwards he became melancholic with suicidal tendencies.

It is important to remember that in patients affected with Bright's disease (particularly when there is extensive destruction of the renal cortex) the administration of even a small dose of opium or morphia is followed by fatal cerebral symptoms. Thus, in one case a grain of opium, prescribed for pain in the head, appeared to be the cause of convulsions and stupor that ended in death ; and in another case like results seemed to be due to the administration of a third of a grain of morphia for lead colic. A patient of the

editor's suffering from cancer of the throat died comatose from subcutaneous injection of a fourth of a grain of morphia. The kidneys were found to be the seat of chronic cirrhosis with wasting of the cortex.

6. Sometimes the patient comes under observation with "*renal dropsy*," having the characters described at p. 606. Such cases are as a rule examples of the supervention of parenchymatous nephritis upon antecedent cirrhosis and at the autopsy the renal affection is found to be of the "mixed" kind. But, unless the patient has been under observation previously, it may be impossible to diagnose the presence of any but recent changes in the kidneys; for the state of the urine is indistinguishable from that which might be produced by them alone. In all persons beyond middle age affected with renal dropsy, it is very important, with a view to prognosis, that the probability of there being chronic as well as acute lesions should be borne in mind.

7. By far the most important group of cases of cirrhosis of the kidneys is that in which the main symptoms are *cardiac*, the patient coming under observation with dyspnœa, palpitation, and dropsy of the dependent parts of the body. Dr Mahomed ('Guy's Hosp. Rep.,' 1879) stated that such cases constitute 17 per cent. of all those in which this affection of the kidneys is found after death, and the author's analysis of a somewhat larger number of cases than his yields an even higher figure. After death the left ventricle is usually found dilated as well as hypertrophied, and its walls may be considered to have yielded to the excessive strain upon them. But sometimes the *post-mortem* evidence of dilatation is by no means complete. Degeneration of the muscular tissue of the heart is probably in many cases the cause of its failure; its substance may be soft and flabby, and under the microscope the fibres may be seen to be granular and to have lost their consistency, breaking into short fragments when an attempt is made to separate them with needles. A fibroid change in the papillary muscles of the mitral valve is not uncommonly seen; they taper gradually into the tendinous cords, instead of appearing as stout fleshy columns.

Whether the mitral valve often leaks is doubtful. A systolic apex-murmur, having some of the characters of the mitral regurgitant bruit, may be heard in many cases, but dilatation of the left ventricle, without valvular lesion, is probably an adequate cause for such a murmur. It is not often that positive proof of regurgitation is afforded at the autopsy by the presence of "ripple lines" on the posterior wall of the left auricle, as in a case recorded by Dr Mahomed ('Guy's Hosp. Rep.,' 1879). Sometimes the edge of the anterior flap of the mitral valve is thickened, and can be readily bent inwards. The occurrence of marked endocarditis secondary to Bright's disease is certainly not common; but, undoubtedly, cases are every now and then seen which appear to admit of no other explanation.

Until recently cases of cardiac failure secondary to cirrhosis of the kidneys were almost always regarded during life as examples of morbus cordis. Over and over again, when the autopsy showed that the renal changes had reached an extreme point, the diagnosis sent down from the wards has been "mitral regurgitation." But it is right to add that sometimes one may be fairly in doubt as to the correct interpretation of the *post-mortem* appearances. The kidneys, perhaps, are of nearly average size, although hard, red, and glistening on section. Even the microscope, while revealing a certain amount of fibroid change, with degeneration of some glomeruli, may leave one in doubt whether this is more than an accidental

feature of the case, especially if the patient was advanced in years. Dr Mahomed pointed out, too, that mitral stenosis and other frequent effects of rheumatic inflammation of the heart are not very rarely found in association with cirrhosis of the kidneys, though apparently the relation between the two affections is only one of coincidence.

In the clinical diagnosis of the cardiac dropsy secondary to renal disease from that which is primary, the state of the urine does not always help us. In either case it may be scanty, high coloured, and loaded with urates, and may contain more or less albumen as well as casts. Wagner says, however, that in cases of renal cirrhosis, even when the urine is scanty, its specific gravity is seldom above 1012 ; and he cites an observation of Traube's that in extreme instances it may remain pale and of low specific gravity even when there is great obstruction to the venous circulation, or (on the other hand) when some febrile disease, such as acute pneumonia, develops itself. In two of our cases, however, at Guy's Hospital, the specific gravity of the urine ranged from 1020 to 1025, although at the autopsy the kidneys weighed only seven and a half or eight ounces, and were very granular ; and in a footnote to Dr Southey's translation of Bartels' work on 'Bright's Disease' a case is mentioned in which in spite of very great wasting of the kidneys the urine had a specific gravity of 1028, and deposited lithates on cooling. Indeed, whatever may be the rule as to the more advanced stages of the affection, so long as the kidneys are of good size, although the microscope may afterwards show that they have undergone extensive fibroid changes, they commonly yield a high-coloured secretion, of great density, during the time that cardiac dropsy is present.

In all such cases it is to the pulse that we must look for guidance in our diagnosis. But it is doubtful whether Dr Mahomed was right in laying stress upon a visible and tortuous condition of the temporal arteries, for this is often due to local senile changes in the coats of these vessels. Again, as this observer himself admits, in cases in which there is much cyanosis from emphysema it is not safe to rely even upon a persistent pulse at the wrist as proof of renal disease. Probably the occurrence of high arterial tension in such circumstances is comparable with the rise of blood pressure in the arteries that is observed at a certain period of asphyxia in experiments on animals (see vol. i, p. 11).

Course and events.—Renal cirrhosis is always an insidious disease, obscure in its origin and extremely slow in its course. The only apparent exceptions are cases which develop after acute scarlatinal nephritis, but probably most if not all of these are examples of secondary atrophy—"the small white kidney" described above (p. 629).

We suspect the presence of this degeneration when a patient about fifty complains of "rheumatic" pains, of muscular weakness and lassitude, of having to rise in the night to pass water, of slight nausea while dressing in the morning, or of troublesome rather than severe headache. The suspicion is much increased if he has had gout, or is exposed to lead-poisoning, or is intemperate in liquor. A pale, sallow face, a hard pulse, a weak or muffled first and a ringing second cardiac sound, with a little œdema of the eyes in the morning and of the ankles at night, make the diagnosis almost certain before we examine the urine ; and if this is abundant, pale, and of low density we may be sure that we are right, even if at first neither albumen nor casts are to be found.

Such patients, judiciously treated, often go on for many years with little

or no aggravation of their symptoms, and it may be very long before they are attacked by uræmic symptoms or by apoplexy.

But they are always in danger. Exposure to cold is likely to bring on bronchitis or pleurisy, or else a subacute attack of tubal nephritis with general dropsy. If they are attacked by pneumonia, the prognosis is serious, and if an injury befalls them or a surgical operation is proposed, the case assumes unusual gravity. If they escape these accidents, and are not cut short by intercurrent diseases, the heart may dilate and they will slowly die with cardiac symptoms.

Nevertheless, with care and good fortune, the renal degeneration often seems to cease in its gradual advance, and we may have patients with chronic Bright's disease for ten or twenty years and living on to the full term of life.

Consecutive renal cirrhosis. — Under this name may be appropriately described an affection of the kidneys which is proved by its histological characters to be a form of Bright's disease, but which is secondary to morbid changes in the renal pelvis, or in some part of the lower urinary passages. Cases of this kind have until recently attracted but little notice. The effects of pressure upon the kidneys have, indeed, long been recognised; but although it has been well known that, when hydronephrosis occurs, the pyramids are flattened and the cortex is thinned out, it seems to have been generally assumed that such appearances are only mechanical in their origin, and independent of any inflammatory process. On the other hand, both physicians and surgeons are, of course, familiar with suppurative nephritis, as the result of affections of the bladder or of the urethra. The cases now in question occupy an intermediate position. Like hydronephrosis, they are slow in their development; but, like suppurative nephritis, they are associated with very definite lesions of the renal tissue. Consecutive interstitial nephritis is often associated with hydronephrosis (*infra*, p. 672); but it may occur when the pelvis of the kidney is perfectly healthy, from urethral stricture, prolapse of the uterus, or other cause of urinary obstruction. It is often cut short in its course by the supervention of a rapidly fatal suppurative process; but it is quite capable of destroying life without any such aid. An excellent account of it is given by Mr Marcus Beck in the fifth volume of 'Reynolds' System of Medicine.'

The appearances presented by the kidneys when affected with consecutive Bright's disease vary in different cases. There is an acute or subacute form in which they are swollen, mottled, with red and white patches, and of a soft consistence. But far more often they are very tough and hard, of a dull white or opaque waxy yellow colour. The surface is generally smooth; it is often very adherent to the capsule, which is thickened, and connected more firmly than is natural with indurated adipose tissue around it. But sometimes there is a marked granulation of the surface, so that the character of the affection may be exactly like that of ordinary cirrhosis of the kidneys. In 1879, in an autopsy on a boy aged twelve, who had a hypertrophied and contracted bladder and great dilatation of the ureter and of the renal pelvis on each side, the two kidneys together, with much adventitious fat about them, weighed less than two ounces. The cortex was in most places shrunken to a thin red line; but, contrasting with the rest, there were some raised, soft, pale yellow, rounded nodules.

Even when the cause of the disease is situated in the bladder, or in the

urethra, the two kidneys are by no means necessarily affected to an equal extent. And when it is in the renal pelvis on one side only, that kidney may suffer alone. Thus, in 1876, in the case of a woman aged forty-seven, who for twelve years had suffered from calculous pyelitis of the left kidney, we found the cortex of that kidney reduced to a thin shell of white fibrous material; the other one weighed nineteen ounces, and had undergone hypertrophy, though it was also affected with recent lardaceous and other changes which had evidently been the cause of the woman's death by uræmic coma. In another case a calculus lay in one calyx of a kidney, and the corresponding part of the cortex was narrow and granular, all the rest of the organ being healthy. Moreover, when consecutive cirrhosis affects the whole of both kidneys, it often happens that the change is far more advanced in some parts than in others, so as to produce deep puckered cicatrices.

Histologically, the morbid process, as described by Mr Beck, is identical with that which characterises other forms of Bright's disease in which the interstitial tissue is mainly affected. There is first an accumulation of mmense numbers of leucocytes in different parts of the cortex, especially round the Malpighian capsules, but also between the tubes. The tubal epithelium is but little altered, being at most somewhat swollen and granular; but sometimes small extravasations of blood occur within the tubes, as well as into the intertubular tissue. The glomeruli become crowded with nuclei, and gradually shrivel into transparent bodies surrounded by thickened capsules. Ultimately the small-cell growth develops into fibrous tissue.

Consecutive Bright's disease occurs in various surgical affections of the urinary organs, such as stricture, disease of the prostate, villous disease of the bladder, stone in the bladder. But it is also seen by physicians in many different circumstances. Thus it may result from any of those diseases that will be enumerated in the next chapter as causes of hydronephrosis. It may also affect one kidney as a consequence of pyelitis; and in cases of tuberculous disease it often plays an important part in completing the work of destruction. Lastly, procidentia uteri and other affections of the female genitalia, which drag upon the ureters or obstruct the urethra, are important causes of consecutive cirrhosis affecting both kidneys.

The clinical recognition of this form of kidney disease is beset with peculiar difficulties. The urine is often so altered as the consequence of cystitis or pyelitis that its characters lend scarcely any assistance. But if it happens to be free from blood and pus it may very probably contain neither albumen nor casts, though sometimes it is albuminous, and occasionally a few hyaline casts may be discovered in it. It is generally rather excessive in quantity, and of low specific gravity. But no conclusion must be drawn from the density of a single specimen, especially soon after surgical interference with the urinary passages. Mr Beck mentions a case of lithotrity in which the specific gravity of the first sample of urine passed was only 1003, whereas that of the whole twenty-four hours' urine was 1018. The quantity of urea excreted may be little, if at all below normal; certainly it is often quite as great as can be expected if one takes into account the small amount of food which the patient can eat and digest.

Nor is the presence of consecutive Bright's disease clearly indicated by any marked general symptoms. In subacute cases there is often an evening rise of temperature to 100° or 101°, whereas the morning temperature may be constantly normal or even subnormal. The patient feels weak and languid,

and steadily loses flesh. The tongue may be very foul, with a thick white or dirty fur, and may even become dry and brown. There may be much thirst, with little appetite for food, and more or less nausea. The skin is usually moist and clammy. There is neither tenderness nor pain in the loins. The patient's mental state is often placid; and he may be drowsy, like a person slightly under the influence of opium. Death sometimes occurs by exhaustion, sometimes by the supervention of some acute disease, sometimes by deepening stupor, or by uræmia. But if the primary disease can be relieved by surgical treatment, it is surprising how all the symptoms may subside that had appeared to indicate grave renal mischief, so that after all one may be left uncertain whether such consecutive results really existed and whether they were not merely dependent upon the primary disease itself. But even restoration to a fair state of health is no proof that the kidneys have not been damaged; and in the most chronic cases of all, in which the organs become yellowish white and tough, there are often for a long time no symptoms at all; the patient remains fairly nourished, he is not anæmic, and he eats, drinks, and sleeps as usual.

It is a very important question whether this form of Bright's disease is liable, like the others, to produce cardio-vascular changes. Mr Beck says that the heart does not become enlarged, although he admits that the renal arterioles show hypertrophy of their muscular coats. Wagner, however, states that the heart was hypertrophied in each of five cases that came under his observation; in one instance there was a well-marked albuminuric retinitis, which indeed was the first symptom noticed by the patient. In two cases secondary to stricture of the urethra, we found the heart weigh sixteen and nineteen ounces respectively.

Cystic disease of the kidneys.—Among the anatomical characters of renal cirrhosis we stated that in many cases there are found in the kidneys immense numbers of cysts, of all sizes up to that of a pea. Such kidneys are sometimes termed "micro-cystic."

There are, however, other cases in which the cysts are much larger, sometimes as big as oranges, and these are usually described separately under the name of "cystic disease of the kidneys." The organs then look as if they were each made up of a mass of rounded cavities, embedded in and separated by an abundant fibrous matrix. But in this there still exist remnants of secreting tissue, by which the renal functions have, however imperfectly, been kept up. The calyces and the pelvis are little, if at all, altered; so that the affection must not be confounded with hydronephrosis. The cysts have walls of varying thickness. In the character of their contents they differ widely among themselves, even in the same kidney. Some are filled with yellow fluid, others with red, some with a gelatinous substance. The fluid always contains albumen, and sometimes blood-discs, leucocytes, or plates of cholesterine; urea and uric acid are said to be generally absent.

There seems to be no doubt that the cysts are formed out of the tubes of the cortex, exactly like those which occur in cases of renal cirrhosis, and this is a strong support of the opinion expressed by Wilks and Moxon in their work on pathological anatomy, that the affection is a form of Bright's disease, notwithstanding one important difference which exists between it and all the other forms. This is the fact that in cystic disease of the kidneys the organs are often so enlarged as to be felt during life through the abdominal

walls. Bright many years ago recorded such a case, in which a distinct tumour was detected in the left loin some months before death, and afterwards another was discovered in the right loin. Sir William Roberts relates a case in which he successfully diagnosed not only the renal nature of the two tumours that he discovered, but also the exact character of the disease by which the kidneys were affected; the tumours appeared to be soft, but not fluctuating, about as large as cocoa-nuts, but disproportionately long. After death one kidney weighed twenty-eight ounces, the other twenty-six. In many instances they have been much larger still. There occurred at Guy's Hospital in 1867 a case in which the right kidney weighed eighty-four ounces, the left fifty-three. In two other published cases the weight of the two kidneys together was six and a half pounds, and eight and three quarter pounds respectively. But the most remarkable case of all is one which was brought before the Pathological Society by Dr Hare (vol. iii), in which the left kidney alone weighed sixteen pounds, the right being in a comparatively early stage of the disease, so that it was only of about twice the natural size. During life a tumour filled the whole left side of the abdomen.

Such monstrous cystic kidneys are sometimes congenital, and may form a serious or insuperable obstacle to delivery of the fœtus (Virchow, 'Ges. Abh.,' p. 864).

The origin of this fœtal condition is very obscure; it is supposed to depend on inflammation of the straight collecting tubes of the kidney *in utero*.

In some cases in the adult also there is reason to suspect that the affection is of intra-uterine origin, and apparently it has ended its course, for there may be no symptoms of chronic renal disease, and none of its effects on the heart; moreover, microscopical examination shows plenty of healthy secreting tissue. In such cases compensatory hypertrophy has probably taken place after birth. In other cases, as in one observed by the editor in a man of fifty-three, there were symptoms which led to the diagnosis of renal cirrhosis during life.

What is most remarkable is that these hypertrophic cystic kidneys are sometimes found in association with cysts of the liver. Dr Bristowe has twice recorded this coincidence ('Path. Trans.,' vols. vii and x), and in the same seventh volume Dr Wilks relates a similar case. Rindfleisch has met with one and Frerichs with another. A sixth example was brought before the Pathological Society in 1881 by the editor ('Path. Trans.,' p. 117, vol. xxxii) with histological figures. As there stated, microscopical examination "proves that this extremely remarkable form of cystic degeneration is histologically the same as the ordinary microcystic form of kidney associated with the later stages of chronic Bright's disease." This is far more probable than Rindfleisch's suggestion that hypertrophic cystic kidneys may be examples of cysto-sarcoma.

The cysts in the liver seem to have a different origin—not to be retention cysts, but to result from vacuolation of the hepatic cells: so Dr Beale concluded in his report on Dr Bristowe's case in 1856, and the same result was independently reached from a study of the case in 1881.*

The symptoms of cystic disease of the kidneys are like those of other

* Drs Savage and Hale White have since described two cases of general paralysis in which cystic degeneration affected not only the liver and kidneys but also the brain, the lungs, and the heart ('Path. Trans.,' 1883, p. 1, with plate). In the same volume Dr Mahomed recorded another case of cystic liver and kidneys (xxxiv, p. 182).

forms of chronic Bright's disease. The urine is often rather excessive in quantity, and of low specific gravity. It seems generally to contain albumen, and occasional hæmaturia has been observed in some cases. There has sometimes been marked emaciation with great prostration of strength. But many patients have not been known to be ill until they were attacked with uncontrollable vomiting, or with uræmic convulsions and coma, or (as has happened in two instances at Guy's Hospital) with cerebral hæmorrhage.

It is obvious that, apart from the presence of abdominal tumours, the clinical effects of this affection depend entirely upon the extent to which the renal cortex is destroyed. Sometimes even the portions left between the cysts are more or less changed into fibrous tissue. On the other hand, a few scattered cysts, of various sizes, are frequently found after death in the bodies of those whose kidneys are in all other respects healthy; their presence is then unimportant.

Mutual relation of the several forms of Bright's disease.—At present there is general concurrence as to the broad divisions of the group of affections first recognised by Bright. The supposition of Frerichs that all the various anatomical forms present different stages of the same pathological process is now universally abandoned. The eight species of Morbus Brightii once described by Rokitansky, and the thirteen described by Rayer, are scarcely remembered. The three forms recognised by Bright's successors at Guy's Hospital, Barlow, Rees, and Wilks, viz. the large red kidney of acute nephritis, the large, smooth, white kidney of subacute and chronic nephritis, and the contracted granular kidney of cirrhosis, are now universally recognised. To these Virchow added the lardaceous form of disease as a fourth variety of Morbus Brightii.

The first and second forms are closely related, and may be conveniently treated together as acute and chronic stages of the same process. The fourth may either be considered, as we have considered it in this volume, independently, or may be regarded (with Bartels and Lecorché) as part of a general degeneration affecting other organs besides the kidney, and when attacking the kidney producing or complicating either the tubal or the interstitial form of Bright's disease.

Subject to these differences (which only affect nomenclature and classification) the division into acute and chronic tubal (parenchymatous) nephritis, cirrhosis or chronic interstitial nephritis and lardaceous disease is recognised by Johnson, Stewart, and Dickinson in this country, by Wagner, Niemeyer, and Eichhorst in Germany, and by Charcot in France.

The origin, sequence, and exact relation of the several pathological processes remains a difficult question. Some of the chief points still disputed are the following:—

a. Does the "coarse," hard, congested kidney (the *Stauungsniere* of German authors), so often seen in cases of chronic valvular disease of the back, go on to become either a large, rough, white, or a contracted granular kidney? Probably the true answer is that it may be an early stage of interstitial cirrhosis, but that this result is very rarely reached because the primary cardiac disease goes on much more quickly.

b. Is acute glomerular nephritis distinct from the first stage of tubal nephritis, and may it go on to produce the large white kidney? (cf. p. 625.)

c. Does the renal affection of diphtheria and other specific febrile diseases ever lead to a chronic structural lesion with the clinical symptoms of

any form of Bright's disease? There is no reason to suppose that it does: the nephritis which follows scarlatina is probably a distinct process throughout.

d. Is the large white kidney always preceded by an acute stage, or may it be produced by a process of tubal nephritis which begins insidiously? While the possibility of such a process cannot be denied, the more carefully cases of renal dropsy are investigated the less is its likelihood.

e. Does the large white kidney ever become contracted and granular? This question, of the existence of a third atrophic stage of tubal nephritis, may now be considered as definitely decided in the affirmative. It is less frequent than recovery and less frequent than death in the second stage, but it undoubtedly occurs and can be distinguished from primary cirrhosis (p. 627).

f. Is the contracted granular kidney always the result of a slow and insidious process from the beginning, or does it ever originate in an acute form of nephritis, such, for instance, as that which follows scarlatina? That the latter pathological sequence is extremely rare is certain, but that exceptional cases occur there is reason to believe (cf. p. 626).

g. May lardaceous disease of the kidneys produce symptoms of Bright's disease without tubal catarrh or interstitial inflammation? Such cases must be very rare, and slight lardaceous degeneration without secondary changes in the kidneys is often found where no symptoms of Bright's disease have been present during life. The rule is for a lardaceous kidney to become a large white one; when the lardaceous change coincides with cirrhosis it is probable that the former is subsequent in date to the latter. Just as a contracted granular kidney is often complicated by tubal nephritis, so it may be complicated by lardaceous changes.

h. Is consecutive cirrhosis of one kidney pathologically identical with chronic Bright's disease? We have seen reason to affirm that it is (p. 648).

i. Are hypertrophic cystic kidneys to be ranked with microcystic specimens as exceptional cases of chronic Bright's disease? This also may be answered with some confidence (so far as adults are concerned) in the affirmative (p. 650).

Geographical distribution.—The acuter forms of Bright's disease are most common in temperate Europe, in Australia, and in the United States, rare in colder climates, as Iceland (according to Dr Hjaltelin, quoted by Dickinson) and Canada, in the South of Europe and at the Cape, in India, and in the tropics generally.

Lardaceous nephritis, being almost always secondary to suppuration or syphilis, is found wherever these conditions occur. Hence it is relatively more common in India than in Europe.

Cirrhosis of the kidneys is decidedly more often met with in England, Scotland, and America than on the Continent. Its prevalence is probably connected with that of gout, and both directly and indirectly with intemperance.

Occurrence in animals.—Acute tubal nephritis with hæmaturia is well known as "red-water" among lambs and horned cattle.

Chronic interstitial nephritis is, according to Mr Bland Sutton ('Path. Trans.,' vol. xxxvii, p. 579), a recognised disease in veterinary medicine, and its association with arterio-capilliary fibrosis and cardiac disturbance is recognised. He reports, from his own observation, changes in the medium-sized arteries and hypertrophy of the left ventricle concurring with interstitial nephritis in horses.

Treatment of Bright's disease.—This must depend upon the stage of the malady, and upon the symptoms present at the time.

Acute cases.—In the early period of parenchymatous nephritis the patient should be kept strictly in bed. It is often wise to place him between blankets, and he should always wear a flannel gown with sleeves down to the wrists, so that the arms may not get cold when they are put outside the bed-clothes. The diet should consist mainly of milk, but farinaceous food may be allowed and small quantities of broth. Where there is severe lumbar pain, it may often be relieved by leeches or cupping-glasses, followed by hot poultices; and this local bloodletting sometimes relieves threatening suppression of urine.

Some writers lay great stress upon keeping up an active flow of water through the glomeruli, so as to wash out the tubes and empty them of the cell masses and casts by which they are blocked up. Thus, Dr Dickinson recommends his patients to drink water freely, and Dr Grainger Stewart prescribes the diuretic salts of potass, or the spiritus ætheris nitrosi, and inhalations of oil of juniper.

More commonly we order such medicines as are supposed to be diaphoretic in their action, especially the liquor ammoniæ acetatis (the use of which has been traditional at Guy's Hospital since the days of Addison), with or without small doses of antimonial wine.

Sir William Roberts believes that the administration of the acetate or citrate of a fixed alkali, such as potass, is of benefit, because the salt is converted into a carbonate which tends to diminish the acidity of the urine, and so to prevent its irritating the kidneys as it passes through them. There seems to be no objection, at any stage of the disease, to the use of such vegetable diuretics as broom, horseradish, juniper, and uva ursi, but not much good is to be looked for from them.

Digitalis might be supposed to have an injurious effect by augmenting arterial pressure, but this, according to Mahomed, is not found to be really the case to any appreciable extent.

The importance of setting the skin freely in action is generally recognised.* It is best effected by *baths*. One plan, advocated by Liebermeister, is that of placing the patient in a bath at about 100°, and then gradually adding more and more hot water until it reaches 104° or 106°; he is left in the bath for half an hour, or even an hour, and is afterwards closely packed in a sheet and warm blankets for two or three hours longer, during which time profuse sweating occurs. It is said that after such a bath the weight is often reduced by from two to four pounds, and even in children by half a pound or a pound. Johnson, Ziemssen, and other writers advise packing the patient in a sheet wrung out of hot water, and in one or two well-warmed blankets. We often employ "hot-air baths," a lamp being placed in the bed in which the patient lies, while the bed-clothes are raised by means of a cradle so as to keep a confined space of air around his body. Any one of these methods may be repeated every day or every other day. Sometimes, however, they cause faintness or headache, and a feeling of oppression, or a rise of temperature, and they have been known to bring on uræmic convulsions. It is especially in cases attended with severe dropsy that they require to be used frequently and energetically. Their effect upon this symptom is often very striking, and they may be followed by an increased secretion of urine as well as

* It was strongly advocated by Dr Osborne, of Dublin, as early as 1835.

of urea; but if there is prostration or great dyspnœa, the hot bath may be dangerous.

Of late years the pharmacopœia has been enriched with a powerful diaphoretic in *pilocarpin*, the active principle of jaborandi, of which from one sixth to one third of a grain may be injected subcutaneously, or about twice that quantity taken by the mouth. It gives rise to a great flow of saliva and also to abundant sweating, but the latter effect is not always so well marked in persons who have Bright's disease as in those who are healthy. In some cases, however, it produces unpleasant symptoms, such as nausea, vomiting, or even collapse. Wagner says that these may be obviated by the administration of a little brandy, or wine, or coffee before the pilocarpine is injected.

The regular administration of *purgatives* is also of importance. The most usual practice is to give a dose of compound jalap powder twice or three times in the week. But if this causes sickness some other preparation may be substituted for it. It is essential that mercurials should not be employed systematically, for comparatively small quantities are apt to set up salivation in persons who have diseased kidneys. This caution was enforced by Osborne and by Bright himself ('Guy's Hosp. Rep.' for 1836, p. 375).

When dropsy cannot be otherwise got rid of, it becomes necessary to resort to *acupuncture*. This is generally done with a needle, which is pushed into the subcutaneous tissue of the thigh, ro leg, or foot, and moved about before being withdrawn, so as to make an open channel, through which the fluid may find its way and escape. The surface should be first well greased, in order that inflammation of the skin may be as much as possible prevented. It is surprising how freely the fluid will often trickle away. It should be soaked up by blankets wrapped round the limb; and these must be frequently changed and well washed before being used a second time. If four or six punctures are made, there is often a great diminution of the anasarca within a day or two; and the abdomen too may appear to be much less swollen than before. Some physicians prefer to make small incisions, or to employ Southey's tubes. The great objection to the latter methods, and, in a less degree, to puncturing the skin at all, is the danger that an erysipeloid inflammation may be set up in consequence, which may cause sloughing and prove fatal by setting up fever or exhaustion.

For the more acute forms of uræmic seizure, *venæsection* is often the best remedy; the abstraction of ten or even twenty ounces of blood may be followed by a strikingly rapid subsidence of the symptoms. A couple of leeches on each temple is one of the most efficient modes of relieving uræmic headache, and probably of warding off more dangerous symptoms. In other cases the inhalation of chloroform proves effectual.

It is generally advisable to give a drastic purge; and for this purpose we may prescribe gamboge or elaterium, a drug which, on account of its irritating properties, is better avoided in other circumstances. Sometimes cold affusion to the head may be employed with advantage. If further experience should confirm the idea that the occurrence of uræmia often leads to a greater activity on the part of the kidneys, the conclusion would seem to be that it should not be treated by diaphoretics, such as pilocarpin or hot-air baths.

When an acute attack of Bright's disease is passing off, the greatest care must be taken to prevent the occurrence of a relapse. The patient should be kept warmly clad, the use of flannel next the skin being especially insisted on. He must avoid exposure to cold, as well as bodily fatigue.

The diet should be restricted in quantity, and the amount of animal food and stimulants must be very small.

At this stage, and also when the disease becomes chronic and is attended with marked anæmia, *ferruginous compounds* are very useful. The tinctura ferri perchloridi is most usually prescribed, but sometimes it is not well borne, so that some milder preparation must be substituted, such as the ferri et ammoniæ citras, or the tinctura ferri acetatis. Digitalis may be given as well; and if there is headache, the bromide of potassium is very useful. It is often of great advantage to the patient to spend the winter in a hot dry climate, such as that of the Riviera, or of Egypt.

The diet must still be carefully regulated, as regards both solid food and alcoholic drinks. Dr Johnson has published some remarkable cases of recovery in grave cases under a purely milk diet continued for weeks or months.

Chronic cases.—In renal cirrhosis, the first indication is to control the arterial tension. At first sight the physiological explanation given at p. 456, according to which this is part of a system of compensation for the renal disease, might seem to involve the conclusion that it should not be interfered with by treatment. But even from that point of view it must be desirable to relieve the circulating fluid, as far as possible, of any substances which the kidneys may find difficulty in removing, and the accumulation of which renders the excessive pressure necessary. As a matter of experience, the regular administration of *purgatives* is believed by Dr Broadbent, as by Dr Mahomed, to be of great value, and even to be capable of warding off for a time the occurrence of cerebral hæmorrhage. *Nitro-glycerine*, again, is sometimes serviceable; especially in relieving uræmic dyspnœa. Or recourse may be had to inhalations of nitrite of amyl, the effect of which is, however, too temporary to be of much assistance. When sedatives are needed, chloral-hydrate is safe and usually efficient.

In cases of cardiac dropsy secondary to renal cirrhosis, *digitalis* is often invaluable. By it the disease can often be kept at bay for a considerable time, and the patient may even be restored to a state of apparent health.

By such treatment with drugs, combined with strict diet and, if possible, removal to a warm climate during winter and spring, cases of Bright's disease, both parenchymatous and interstitial, may be greatly alleviated and not infrequently cured. The statement made thirty years ago, that parenchymatous nephritis when once established is as hopeless as tuberculous phthisis, is certainly untrue now, even with the better prognosis of phthisis which pathology and clinical experience alike have taught us.

URINARY CALCULI AND THEIR EFFECTS

CALCULI.—*Lithic or Uric acid—Lithate of Soda—Oxalate of Lime—Cystine—Xanthine—Indigo and other rare substances—Phosphate of lime—Carbonate of lime—Fusible calculus—Ætiology of calculi.*

NEPHROLITHIASIS.—*Lumbago—Hæmaturia—Renal Colic—Obstruction and suppression of urine—Atrophy—Hydronephrosis—Pyelitis and renal abscess—Solvent and operative treatment of calculi and of their results.*

THE materials of which calculi are constructed are the same as those of the various inorganic deposits from the urine, described in an earlier chapter (p. 570). We have first to consider how they undergo aggregation into calculi, a question which has lately been studied by Dr Vandyke Carter, Dr Beale, and Dr Ord.

It has long been known that a calculus is generally made up of concentric layers which may differ among themselves in composition, and that the nucleus or central part may be quite distinct in character from the rest of its substance. Lithic acid forms the chief ingredient of most calculi; according to Sir William Roberts five out of six consist chiefly of this material; and until lately it was thought to be the most frequent constituent of their nuclei. This, however, has now been shown to be a mistake. Even when the apparent nucleus really consists of uric acid, the microscope generally shows that the very centre of all has a different composition. It consists either of an aggregation of spheroids or dumb-bells of oxalate of lime, or else of hedgehog-crystals of urate of soda. In countries where *Bilharzia hæmatobia* is endemic, its ova frequently form a nucleus. In other cases it consists of inspissated mucus, or of a small blood-clot, or of casts of the renal tubes. Ebstein, in 'Ziemssen's Cyclopædia,' mentions that he once found in the urine a deposit of epithelial cells from the renal pelvis beautifully encrusted with uric acid; the patient, a woman, afterwards had symptoms of renal colic and passed concretions, but he does not state whether their nuclei were formed upon epithelial cells. As regards the spheroids of oxalate of lime which form the nuclei of most calculi, Dr Beale maintains that they often have their origin within the tubes of the kidney. Not only has he found dumb-bells in the substance of casts, but he describes and figures microscopic calculi, already laminated, which he says he has many times seen in the renal tubes after death. Lastly, vesical calculi are sometimes moulded on foreign bodies introduced into the bladder from without.

In the laminæ which generally make up the body of a calculus the materials are often laid down in a definite manner. Thus lithic acid appears in the form of rods or columns, piled upon one another or arranged side by side; the lithates are seen to form globules, with concentric rings; oxalate of lime may form spheroids, and dumb-bells of this substance are sometimes embedded in laminæ consisting mainly of urates. Clear crystalline layers, however, make up a large part of some concretions. These facts, which

have been pointed out by Dr Vandyke Carter, do not seem to indicate that calculi are anything else than "precipitates or aggregations of ordinary crystalline and amorphous deposits held together by mucus," though he thinks such a theory inadequate. It is true that when the principal ingredients of a stone are removed by solvents, a small quantity of organic matter is left; but to speak of this as an "organic basis" or "matrix" is to suggest misleading analogies with the very different processes of true growth seen in bones and in shells.

Dr Beale describes a remarkable instance in which a smooth, oval urethral calculus, two inches and a quarter in length, was composed entirely of very small concretions of calcic and triple phosphates, united by a whitish material; this, however, was altogether an exceptional specimen, for it is said to have lain in the urethra for fifty years before it was extracted by operation. In vol. xiii of the 'Pathological Transactions' is a full account of this case by Mr Haynes Walton.

Few calculi consist entirely of one substance. Still conspicuous chemical distinctions exist between calculi; and there are corresponding differences in their physical characters.

1. *Lithic acid calculi* ($C_5H_4N_4O_3$).—These are the commonest of all stones. They occur as minutely hard bodies, of round or oval shape; smooth on the surface or tuberculated; of a yellow, fawn, or reddish colour. They are formed in the pelvis of the kidney. Sometimes they are passed in enormous numbers while still very small, from the size of poppy seeds up to that of mustard seeds or split peas. Not infrequently several lithic acid stones, perhaps as large as marbles, are found in the pelvis of the kidney, or in the bladder. They then often have flat surfaces or smaller facets, produced by contact with one another; the presence of such surfaces is important as an indication that the concretion is not solitary. Calculi of uric acid sometimes weigh three or four ounces.

2. *Lithate of soda* ($C_5H_3NaN_4O_3$).—These are soft concretions, of rare occurrence, which appear never to reach a large size, except by the addition of lithic acid, or some other substance. Like crystalline deposits of lithates they occur chiefly in children.

3. *Oxalate of lime* (CaC_2O_4).—These calculi are characterised by their extreme hardness and by their rough irregular surface, whence the name of "mulberry calculi." They are, however, often passed safely while still small; they then appear as smooth, rounded, greyish, or brown bodies, which are compared to hemp seeds; or they may be covered with glistening crystals. As a cause of hæmaturia, pyelitis, or renal colic in middle-aged people, such little calculi are more frequent, in the author's experience, than those of any other kind. The larger "mulberry" stones are generally of a blackish-brown colour, and irregularly rounded form; Roberts, however, says that stones of the same composition are sometimes oval, smooth, and of a bluish-grey colour. When crushed, they break into sharp, angular pieces. Mulberry calculi are usually solitary, and never present in large numbers. It seldom happens that more than two of the small hemp-seed concretions are passed by the same patient, and then they are passed at long intervals.

The four following kinds of calculus are all so rare as to be little more than pathological curiosities.

4. *Cystine* (CHNSO).—Calculi of this substance are very rare. They

are usually egg-shaped; their surface is granular and glistens with minute crystals; they are of a honey-yellow colour, and on section look semi-translucent, like beeswax, and show indications of a radiating structure. It is curious that when exposed to daylight for a long time they often slowly acquire a delicate green hue, due to the growth of a microscopic fungus. They are of rather soft consistence. They may reach a considerable size, weighing as much as three or four ounces, notwithstanding the low specific gravity of the material of which they are composed. Roberts describes two specimens which were passed per urethram; one was cylindrical and an inch and a quarter long, and weighed twenty-seven grains. They usually consist of pure cystine; but one mentioned by the same author had a nucleus of uric acid, with an outer layer made up of a mixture of uric acid and cystine. In the 'Path. Trans.' for 1880 (p. 182), Dr Shattock described a cystine calculus, which in all parts contained a minute proportion of oxalate of lime, and had in its interior a defined thin layer, consisting entirely of that salt: the nucleus was of cystine. In the Guy's Hosp. museum there are ten cystine calculi successively passed by the same patient (Prep. 2144, *seq.*).

5. *Xanthine* ($C_5H_4N_4O_2$).—This substance differs from the constituents of calculi hitherto mentioned in not being known as a urinary deposit.*

Xanthine was first discovered by Dr Marcet about the year 1817, in a calculus, and only five instances of its occurrence in the form of concretions are on record altogether. Its chemical composition is that of uric acid, less one atom of oxygen; hence it was at one time termed "uric oxide." The characteristic test for it is analogous to the well-known murexide test for uric acid: when moistened with nitric acid it dissolves without effervescence, and on evaporation there is left a bright yellow residue; this, when cool, becomes violet red (not purple) if treated with a solution of caustic potass. It is insoluble in cold and almost insoluble in hot water, but is readily soluble in liquor ammoniæ or liquor potassæ.

Hitherto xanthine calculi seem never to have been found in the renal pelvis, only in the bladder. In their physical characters they resemble uric acid calculi. One removed by Langenbeck from a child weighed 339 grains (Guy's Hosp. museum, 2145^{90}).

6. *Indigo* (C_8H_5NO).—This substance, again, can hardly be said to be known as a urinary deposit, though it sometimes colours lithates. As a concretion it has been found only once in the renal pelvis of a middle-aged woman, by Dr Ord ('Path. Trans.,' 1878). It formed a flat broad cake, like a lozenge in shape and size, weighing forty grains; its surface was partly dark brown, partly blue black; its section grey and polished. On paper it left a blue-black mark.

7. *Urostealith.*—This name was given by Heller ('Heller's Arch.,' Band ii) to certain soft elastic concretions, like india rubber, which were passed by a young man. Dr Moore, of Dublin, has since ('Dubl. Quar. Journ.,' xvii) met with similar specimens. And in the museum of the College of Surgeons there are two such which belonged to Hunter's collection. They were taken from the bladder, and the suggestion has been made that the fatty salts of lime, of which they are entirely made up, were formed by the decomposition of

* Dr Bence Jones alone, in 1862, found on two occasions what he believed to be xanthine as a sediment in the urine; the crystals were like those of uric acid, but they were at once dissolved when the fluid was warmed.

a solution of soap, which might perhaps have been injected for therapeutical purposes. This theory, however, seems not to be applicable to the other cases.

8. In the 'Med.-Chir. Trans.' for 1872 Mr McCarthy has described certain calculi of peculiar form, eleven in number, which were taken from the left kidney of a woman after death. When first removed they felt soft and greasy, and they each consisted of a central globular body, with long tapering spines projecting from it. None of these appear to have been analysed, but a somewhat similar concretion from the right kidney was found by Dr Tidy to contain 36·6 per cent. of fat and of cholesterine, the other chief ingredients being lithates (35 per cent.) and oxalates (9 per cent.). Similar specimens in the museum of the College of Surgeons are said to consist of oxalate of lime. Mr Benjamin Duke, of Clapham, once showed the author a number of similar calculi removed after death.

9. *Phosphate of lime or bone earth* ($Ca_3 2PO_4$).—All the varieties of calculi hitherto described appear to occur in acid urine; certainly this is the case with the two of clinical importance—uric acid and calcic oxalate. There is, therefore, a broad distinction between those and three varieties which remain, for these can only be formed when the urine is alkaline. One, which is very rare, consists of phosphate of lime. Concretions formed of this substance are described by Roberts as being white and chalky in appearance, and rather smooth on the surface, with an earthy fracture. Their texture is sometimes loose, sometimes very compact. They vary in size from that of a pea to that of a hen's egg. In the museum of the Manchester Infirmary there is a laminated specimen in which bone earth alternates with uric acid. A peculiarity of calculi composed of pure phosphate of lime is that while they require alkaline urine for their production it yet must not be alkaline from ammonia, since, if it were, the triple phosphate could not fail to form, and to make up a large part of their substance. But even in urine which is alkaline from fixed alkali they arise very seldom.

10. *Carbonate of lime* ($CaCO_3$).—Concretions of this substance are very rarely seen in the human subject. Roberts says that when they do occur they are generally small very hard bodies, varying from the most minute size up to that of a hazel nut; grey, yellowish, or bronze coloured; smooth on the surface, sometimes with a metallic lustre. Dr Haldane, of Edinburgh, once found a number of little calculi consisting of carbonate of lime in the dilated pelvis of the kidney of a man who had died from spinal abscess. Some years before Roberts had met with a case in which immense numbers of precisely similar bodies were passed during life. The largest of them were of the size of mustard seeds, they were translucent and of an amber colour, and showed a laminated structure under the microscope. They were probably also of renal origin. The urine in which they were found was ammoniacal.*

11. *Mixed calcic and ammonio-magnesian phosphates* ("*fusible calculus*").—We saw how the decomposition of urea into carbonate of ammonia during the alkaline fermentation of urine inevitably leads to the formation of the "triple" phosphate of ammonia and magnesia; and how this salt and the amorphous phosphate of lime come down together as a precipitate, which has a strong tendency to agglomerate into a mortar-like mass, and sometimes

* In vol. xix of the 'Pathological Transactions' a large renal calculus is described as composed of carbonate of lime, but in vol. xxviii it is stated that on a re-examination of the specimen it proved to consist of other materials.

concretes upon the surface of the inflamed mucous membrane of the bladder. It also accumulates upon any foreign body which is exposed to its action, and particularly upon calculi, of whatever nature, which are washed by putrid urine. It rarely forms the starting-point of a stone, whether in the kidney or in the bladder; but it often rapidly converts a small nucleus into a concretion of enormous size. It is a soft friable substance; in the blow-pipe flame it fuses into a kind of enamel, whence the term "fusible calculus." In the bladder it may form stones weighing as much as twenty, thirty, or even forty ounces. In the kidney it is often moulded into the shape of the pelvis and calyces, each branch having a funnel-shaped expansion at its extremity.

Dr Gee has recorded (vol. xxxix, 'Med.-Chir. Trans.') a case in which such a concretion weighed thirty-six and a quarter ounces; it, or rather the remains of the kidney stretched over it, had been felt during life as an abdominal tumour of stony hardness. As is often the case, its surface was covered with brilliant crystals of pure triple phosphate, but these were of exceptional size and beauty, some of the prisms being half an inch long. The whole of the calculus was very hard and dense. It had a dark brown nucleus, which consisted mainly of oxalate of lime. Whether this also had a branched form is not stated. Such "coral-like" masses, sending prolongations into the several renal calyces, and probably always composed of the mixed phosphates, are only found as a result of ammonical decomposition of the urine in the renal pelvis.

Ætiology.—It will be apparent from this account of the different kinds of urinary calculi that in a patient whose urine is, and has been, acid, the only kinds that are at all frequently met with are those mainly composed either of uric acid, or of urates, or of oxalate of lime, or of alternating layers of two or all of them. But the phosphatic calculi only occur when the urine is ammoniacal—by deposition on a pre-existing nucleus, and this (when not a foreign body) is either lithic acid or oxalate of lime. The ætiology of stone, therefore, resolves itself into the causes that favour the precipitation of uric acid or calcic oxalate from the urine.

The eastern counties of England, and especially Norfolk, are well known to yield a much larger proportion of cases of stone—at least of stone in the bladder—than any other districts of the country. But, in his address on surgery, delivered before the British Medical Association, Mr Cadge, of Norwich, estimated ('Brit. Med. Journ.,' 1874, ii) that lithic acid and the lithates make up nine tenths of the calculi observed there, whereas for the whole of England a lower figure (usually five sixths) is generally given. Consequently, it seems not improbable that these ingredients may alone be concerned in producing the excessive number of cases observed in the eastern counties, the frequency of calculi formed of oxalate of lime, cystine, and other substances being perhaps no greater there than elsewhere. Mr Cadge could only call to mind three cases in which he had removed an oxalate of lime stone from an adult. In one of these cases only the outside of the stone consisted of that substance, the central part being uric acid; the patient had been recently residing in North Wales, having previously left Norfolk, probably with a uric acid stone already in his bladder. Another case occurred in a soldier who had only been for a short time in this country. The third was in a Norfolk man, but he also had resided elsewhere.

Why calculous (lithic) deposits should occur so frequently in East Anglia is unknown. One point on which Mr Cadge insists is that in young children

it is almost entirely confined to the poor, and he was inclined to think that a deficient supply of milk as food had to do with it. The only young child of well-to-do parents whom he had treated for stone was said by the mother to have differed from all her other children in having never been able to take milk. He believed that another important factor was the hardness of the drinking water.

The hereditary influence sometimes seems to show itself in a very marked way. But persons who come to reside in Norfolk are said sometimes to form a calculus very rapidly, while others who leave the district lose the proclivity.

Stone is far more frequent in males than in females; but this applies especially to stone in the bladder.

As regards age, vesical calculi are well known to be much less common in adults than in children and in old people, but it is not certain whether the same rule applies to renal calculi.

NEPHROLITHIASIS.—The morbid conditions produced by the presence of gravel or calculi in the kidney are commonly classed together under this name. They depend upon irritation of the renal pelvis, or upon mechanical obstruction of the ureter, and may be arranged as follows:

1. *Pain in the loins*, or renal lumbago.
2. *Hæmaturia*, the most important and most constant effect of calculi.
3. *Renal colic*, produced by the passage of a stone into the ureter.
4. *Obstructive anuria or suppression of urine*, caused by impaction of a calculus in one ureter, when the opposite kidney is from previous disease incapable of secreting urine.
5. *Unilateral atrophy of the kidney*, from obstruction of its ureter.
6. *Hydronephrosis*, from the same cause.
7. *Pyelitis*, with *pyonephrosis* and *perinephral abscess*.

It is to be observed that there is scarcely one of these affections that may not sometimes be due to other causes than nephrolithiasis. Nevertheless, to save repetition, we will, in the present chapter, discuss each of them fully, without attempting to limit our view to those cases in which calculi are present.

1. *Lumbago.*—In the mildest form of nephrolithiasis the principal symptom is a dull aching pain in the loins, such as is commonly called *lumbago*. Such a pain is most frequently the result of irritation of the kidneys by the urine, or by something deposited from it. It is often supposed that myalgia (or as it is vaguely called, muscular "rheumatism") affecting the lumbar muscles—like that which so commonly occurs in the shoulder—is as frequent a cause; and one is often tempted to take this view by the patient's complaining that his back feels stiff, and that stooping greatly aggravates his discomfort or suffering. But even in such cases more rapid relief is given by rendering the urine alkaline than by any other kind of treatment. Often, in the author's experience, the pain has been at once removed by a few full doses of the citrate of potass, repeated at short intervals. It may be a question whether the complaint is due to an over-acid state of the urine alone, or whether there is formed in the renal pelvis an actual sediment of uric acid, which is afterwards redissolved when the secretion becomes alkaline. One point in favour of the latter opinion is that the dull aching

sensation is sometimes experienced only when the patient first wakes in the morning, and ceases when he gets into the upright posture.

2. *Hæmaturia.*—That urine contains blood may be obvious by its appearance; though, strictly speaking, one often cannot be sure that a case is not rather one of hæmoglobinuria until one has ascertained with the microscope that blood-discs are present (cf. p. 583). When the bleeding is very profuse the urine may look like pure blood. From this every gradation of colour may be observed down to the palest pink or the faintest brown tinge. In many instances, when the colour is not in itself distinctive, there is a sediment which cannot be mistaken: on tilting the chamber-vessel from side to side, a granular-looking reddish-brown substance may be seen lying just within the edge of the fluid, and following its movements. When no such sediment is visible, the microscope will at once reveal the presence of blood in a drop of urine taken from the bottom of a vessel in which it has been standing for a little while, or sometimes from the bulk of the urine directly after it is voided. It is impossible to insist too strongly upon the importance of microscopical examination in all cases in which hæmaturia may be suspected; without it the absence of blood should never be asserted. Theoretically, albumen must be supposed to be always contained in urine in which there is blood. But the ordinary tests often fail to show it, when with the microscope blood-discs are at once seen. The only circumstances in which the results of a microscopical examination cannot be trusted are when the urine is of very low specific gravity or in a state of ammoniacal decomposition, for in such cases the blood-discs may be rapidly dissolved. In urine of acid reaction and a normal density they remain visible for days.

Blood-discs in urine do not always retain their biconcave form: sometimes they are shrivelled, with crenated edges, and sometimes they show protrusions; while in dilute urine, they are often represented by delicate globular corpuscles slightly larger than natural. The only things that can be mistaken for blood-discs in urine are compressed spirilla, minute discoid forms of oxalate of lime, and perhaps nuclei of renal epithelium. Dr Beale speaks of cases in which spirilla looked so like blood-discs that great care was required to distinguish them.

In the exceptional cases in which the microscope fails to reveal the presence of blood, the spectroscope may be used (see p. 584) or chemical tests may be applied. One test is known as Heller's; it consists in rendering the urine alkaline by the addition of caustic potass or soda, and then boiling, so as to precipitate the earthy phosphates, which carry down with them any blood-colouring matter that the fluid may contain. Salkowski says that this reaction is very delicate, but that it also occurs with the colouring matter of rhubarb or senna. Another test which is commonly employed in this country is that of adding to a small quantity of urine a drop of freshly prepared tincture of guaiacum and shaking it up with a few drops of ozonic ether. If blood is present, a brilliant blue colour appears in the layer of ether that collects on the surface of the fluid when it has stood for a minute or so. The chief sources of fallacy lie in the fact that saliva, nasal mucus and iodide of potassium give the same reaction. According to Dr Mahomed ('Med.-Chir. Trans.,' lvii) this guaiacum test is even more delicate than that of the spectroscope.

It must not be forgotten that blood may be added to urine for purposes

of deception. In one such case the microscope showed that the corpuscles present were the oval blood-discs of a bird.*

Its causes.—In some cases hæmaturia is merely a symptom of a general disease such as purpura, scurvy, smallpox, or malarial fever. In some it is possibly vicarious to menstruation† or supplementary to the hæmorrhoidal flux, or to asthma; though for instances of such occurrences one has to go back to writers of the earlier part of the century. In most cases, however, hæmaturia is a symptom of some disease of the kidneys or bladder. The most frequent of these are calculi or gravel. Next to these come acute inflammation, tubercular ulceration, and new growths. But it must not be overlooked that when blood is poured into the urinary passages in sufficient quantity to coagulate *in situ*, symptoms of various kinds may result, which simulate effects of some antecedent disease to which the hæmorrhage was due. Thus a clot in the ureter may produce an attack of "renal colic" just like that caused by a calculus; and clots in the bladder may give rise to dysuria, or to complete retention of urine.

Seat of hæmorrhage.—When coagula are passed in the urine, they often clear up all doubt as to the place of the hæmorrhage. Sometimes they are flat, having evidently been formed upon the floor of the bladder; sometimes cylindrical, having come from a ureter. Blood-casts of the uriniferous tubes prove of course that the hæmorrhage is derived from the renal cortex. They are rarely found except in acute tubal nephritis.

Bleeding from the substance of the kidney is seen when the organ has been lacerated by violence, in cases of poisoning by oil of turpentine or by cantharides, and in very rare cases in which quinine, by a curious idiosyncrasy, produces a like effect ('Brit. Med. Jour.,' Jan., 1870). The renal affection which in some patients is set up by the application of a blister sometimes produces hæmaturia; sometimes only fibrin is exuded, but in such quantity as to form transparent gelatinous clots in the bladder, and obstruct the outflow of urine. Two cases of this kind are related by Bartels.

Another circumstance that may aid in the diagnosis of the origin of hæmaturia is the way in which the blood escapes during micturition. Should bleeding occur into the urethra, the blood precedes the stream of urine, and is washed out by it. On the other hand, when the bladder is the seat of disease, it is towards the end of micturition that the urine is most deeply discoloured. Dr Beale speaks of cases in which persons, apparently healthy, day after day pass small quantities of blood, just as micturition is ceasing; it would seem that "the effort required to expel the last drop of urine causes the rupture of a few capillaries about the membranous part of the urethra or the neck of the bladder." In some instances this affection has been caused by undue sexual indulgence. It usually ceases after a time if the patient rests. When there is hæmorrhage from the renal pelvis, the blood is always intimately mixed with the urine; but so it is likewise in many cases in which its origin is from the bladder.

When the urine is of red or pink hue this is due to the reaction being alkaline, and hence the seat of hæmorrhage is most likely the bladder.

If the urine, instead of being bright red, has a brownish hue ("like tea"),

* I once checked an attempt of this kind, by remarking that it was necessary for me to see the urine passed; the patient, who had had a railway accident, subsequently confessed that he mixed with his urine blood that came from a cut upon the wrist.—C. H. F.

† It is obvious that blood may be present in a healthy woman's urine during menstruation.

this shows that the blood-corpuscles have been acted on by acid and their hæmoglobin turned to acid hæmatin : the probability is that it came from the kidney. But whenever the hæmorrhage is profuse, the urine remains red ; for it then neutralises the smaller amount of acid urine.

From what has been stated it must be evident that in a large proportion of cases, including those in which the bleeding is most severe, there are no certain indications as to the seat of the disease, except from other symptoms, such as pain or dysuria.

In practice, however, almost the only vesical affections that give rise to profuse hæmaturia, as their sole symptom, are villous tumours and other forms of new growth, generally malignant. Prof. Humphry believes that the occasional cessation of hæmaturia for a long period affords an indication in favour of the diagnosis of villous disease of the bladder. Such diseases occur chiefly in patients who have already reached middle age or passed beyond it ; but in 1865, a child, only eighteen months old, died in Guy's Hospital, of the effects of a polypoid growth from the right side of the neck of the bladder. The editor had once sent him from Mr Bryant a minute clot which was passed with the urine of a little girl in apparent health and only five years old. On microscopic examination a beautiful tuft of villous growth was apparent. In 1877 an autopsy was made in the case of a man aged thirty-four, who said that ever since he was twelve years old he had suffered from hæmaturia, recurring at intervals of weeks or months, with greater or less severity. There was found to be a soft spindle-cell sarcoma, growing as a flat, slightly-lobulated tumour from the base of the bladder on one side. It is a point of some importance that in a case of Murchison's ('Path. Trans.,' 1869), villous growths from the pelves of both kidneys were associated with a like affection of the bladder. Mr Davies-Colley once succeeded ('Clin. Soc. Trans.,' 1881) in removing through a perineal incision a villous growth from the bladder of a man aged thirty-two, who had suffered for eight years from hæmaturia, and was completely cured by the operation. This patient sometimes passed blood at the beginning of micturition, sometimes at the end of it, the explanation no doubt being that the villi were sometimes nipped in the prostatic part of the urethra. Sir Henry Thompson has published several similar cases.

Hæmorrhage from the renal pelvis may be due to various causes. When a patient passes blood in the urine without there being other symptoms to throw light upon the nature of the disease, the presumption is generally in favour of the presence of a renal calculus ; but the possibility that tubercle or cancer of the kidney may be developing itself must never be left out of consideration. It is, however, surprising how often one meets in practice with instances in which profuse hæmaturia causes for the time the utmost alarm, without there being any clue as to its source, and ceases after two or three days, leaving the patient as well as ever.

In 1881 a man aged sixty-three was admitted into Guy's Hospital with extreme wasting and cachexia. After a few days he was attacked with severe hæmaturia. This, however, quickly subsided, though afterwards pus appeared in the urine, and three weeks later he died. At the autopsy it was found that the cause of the wasting was cancer of the œsophagus. In one of the calyces of the left kidney a calculus was impacted. The lining of the renal pelvis was much thickened, œdematous, and of a deep purple colour from ecchymosis, looking like velvet. Probably a like condition is generally present when hæmaturia is the main symptom.

It is an interesting question whether bleeding ever occurs as a result of the irritation produced by mere granular deposits of uric acid or oxalate of lime, or whether the presence of a larger concretion is necessary. A certain answer to this question can hardly be expected; but Sir Benjamin Brodie and others have taught that "red sand" (uric acid) is capable at times of causing hæmaturia. A point of considerable clinical importance is that jolting movements of the body are exceedingly apt to bring on, or to aggravate, hæmaturia in persons who have even small calculous concretions in the kidneys. Not only does this occur after horse exercise, but also after riding in a carriage with springs. Even when no blood is obvious to the naked eye, it is always worth while to make a microscopic examination of the urine passed under such circumstances by a patient in whom renal calculus is suspected. In most cases of this kind there is lumbar pain, or pain referred to the front of the abdomen on one side, or to the groin. Another circumstance, to which Brodie drew special attention, is that the symptoms are sometimes referred mainly to the bladder. Micturition may be frequent, and accompanied by a cutting pain in the neck of the bladder and in the urethra, so that the presence of a vesical calculus is suspected.

Treatment.—Whatever the cause of hæmaturia, rest in bed is essential. Cold should be applied as near the seat of hæmorrhage, as possible; if it be from the kidney, ice-bags to the loin; if it be from the bladder, ice-bags to the hypogastric region of the abdomen, or injections of iced water into the rectum or into the bladder itself. Prout found the injection into the bladder of a solution of alum (twenty to forty grains in a pint of water) very effectual when the hæmorrhage was vesical. As internal styptics, gallic acid, acetate of lead, ergot or alum may be employed. In some cases the tincture of perchloride of iron is particularly serviceable. Oil of turpentine, too, may succeed when all other drugs have failed: it would of course be wrong to give it should the blood come from the renal cortex, but in cases of hæmorrhage from the pelvis of the kidney there is no objection to its use. The hæmaturia caused by cantharides or by turpentine is treated by cupping to the loins, warm poultices, diaphoretics, and purgatives.

On the whole, drugs have less effect on hæmaturia than on most other forms of hæmorrhage, less, for example, than ergot in hæmoptysis, or opium and lead in bleeding from the bowels.*

Hæmaturia, though so important as a symptom, is after all not a serious occurrence in itself. It is far less dangerous than intestinal gastric or pulmonary hæmorrhage; indeed, few physicians have seen a fatal case of bleeding from the kidneys.

In the treatment of recurrent or persistent hæmaturia attributed to renal calculus, the first thing is, if possible, to get rid of the cause of the disease. This will be considered at the close of the present chapter (p. 683).

* "I do not lay any great stress upon the use of internal astringent remedies (for hæmorrhages) because it does not appear likely from reasoning, that they should do any service, and I am far from being convinced by experiment that they ever do, except perhaps in hæmorrhages of the primæ viæ. They may sometimes have appeared to be attended with success, because there is but a small proportion of hæmorrhages, not owing to internal violence, which would prove fatal though no means were used to stop them" (Heberden). The latest expression of opinion agrees with that of this wise physician more than a hundred years ago: Dr Saundby says of the use of drugs in hæmaturia: "My experience has been that they are all very untrustworthy, and I hesitate to give the preference to any one." ('Brit. Med. Journ.,' Dec. 17th, 1887).

3. *Nephralgia.*—The process by which a calculus passes down the ureter into the bladder is attended with special symptoms, which are commonly known as renal colic. They often set in with extreme suddenness and violence, and constitute one of the most painful of diseases. The patient is sometimes awakened by them from sleep; sometimes they are brought on by the jolting of a vehicle, or by some muscular effort, such as sneezing, coughing, running, jumping, or riding on horseback.

The *pain* is generally described as running from one loin downwards in the direction of the ureter, but it may also spread over the whole of the abdomen, or radiate to the chest or to the shoulder-blade, or appear to run along the costal cartilages or the iliac crest. Very commonly it extends down into the corresponding testicle, which becomes drawn up towards the inguinal canal, and is distinctly swollen as well as tender. There may be pain, too, along the inner side of the thigh, with numbness and tingling of the skin there. The suffering is often intense; the patient grows faint and cold, and breaks out into a profuse sweat; the pulse becomes very rapid and small,* the breathing is quickened, and the temperature may presently rise.

Nausea and *vomiting* are marked symptoms; the matters rejected from the stomach often becoming bilious after a time. Epileptiform convulsions have sometimes been observed. In pregnant women abortion frequently takes place; cases are even recorded in which successive pregnancies have been brought to a termination at considerable intervals of time by the supervention of attacks of renal colic.

In the vain hope of relieving pain the patient adopts the most extraordinary positions. A medical friend of the author found that the only attitude which seemed to give him any comfort was kneeling with the head bent over so as to touch the gound. But some persons prefer to lie on the side with the knees drawn up. Movements of the body generally increase the pain, and yet the restlessness is so great that it is often impossible for the patient to remain long in one position. The paroxysm, if it lasts long, is generally interrupted by remissions of the pain, which before long becomes as bad again as ever. Its duration is very variable; it may be over in the course of a few hours, or it may last for several days. Its termination is sometimes quite sudden; the patient, perhaps during a violent fit of retching, may experience a sensation as though he were stabbed, and from that moment the acute suffering ceases, for the stone has slipped into the bladder.

Micturition during an attack of renal colic is generally frequent, and sometimes there is severe strangury, with burning pain in the urethra, or at the end of the meatus. Often only a few drops of urine are voided at a time, and they are often deeply coloured with blood. But if the kidney on the opposite side is healthy, it may go on pouring out a normal secretion. Ebstein remarks that in cases of calculous pyelitis, in which the urine is habitually discoloured by blood and pus, the fact that it becomes normal when a stone is impacted in the ureter affords valuable evidence that the other kidney is not affected in the same way. But it often happens that calculi are present in both kidneys, so that the urine from that which is unobstructed is purulent, or blood-stained, or perhaps of low specific gravity and albuminous, as the result of consecutive Bright's disease. Those

* Traube, however, recorded a case in which during repeated attacks of renal colic, the pulse was slow, full, and remarkably tense.

cases in which at the time when an attack of renal colic occurs the opposite kidney is absent, or so atrophied as to be unable to secrete any urine, will be specially described in the next section.

It is doubtful whether the passage of a calculus is dangerous to life when the other kidney is healthy. Ebstein speaks of the possibility of its ulcerating through the walls of the ureter and escaping, so as to set up fatal peritonitis. But the case which he cites from Allan Webb ('Pathologia Indica,' 1846) was not uncomplicated, for "the vermiform appendix and the ureter were found ulcerated and adherent to one another and to the surrounding structures, and a large amount of pus had escaped from the ulcerated ureter into the abdominal cavity."

On the other hand, it does not seem that the subsidence of an attack of renal colic is a complete proof that the stone has passed into the bladder. Sometimes perhaps it undergoes disintegration in its course downwards, and the fragments escape unnoticed in the urine. In other cases it remains permanently impacted, while the kidney undergoes atrophy or passes into a state of hydronephrosis, as will be presently described.

The fact that the pain may cease while its apparent cause is still there, affords an argument in favour of an opinion held by Traube that nephralgia really is due, not so much to the direct irritation of the mucous membrane by the calculus, as to the peristaltic movements which take place in the over-distended ureter above. Roberts, however, relates a case in which the track of a calculus could be followed along the whole length of the tube by the abrasions which it had produced.

A ureter which has given passage to a stone may be found considerably wider than natural when death occurs from some other cause, after the lapse perhaps of many years; in one case the vesical orifice of the tube was large enough to admit a thick probe. The occurrence of such dilatation of the tube explains the fact that when attacks of renal colic are frequently repeated, the later ones are comparatively slight and of much shorter duration; but of course a great deal depends upon the shape and the size of the calculus on each occasion. Sometimes the affection recurs at tolerably regular intervals. Sometimes a calculus is passed by a patient who never before showed any symptoms of urinary disorder; and this is especially frequent when the concretion consists of oxalate of lime. An attack of renal colic sometimes puts an end to pyelitis which had existed for a considerable time; in such a case one may infer that the renal pelvis contained a solitary stone, which was the cause of the previous symptoms, and which has now escaped into the bladder. Prout, however, remarks that after the passage of an oxalate of lime calculus painful sensations often continue to be experienced for some time, so that he had found difficulty in convincing his patient that other calculi did not remain behind.

In making a *diagnosis*, we must not suppose that the occurrence of renal colic is in itself proof of the passage of a stone into the ureter. In some cases (as, for example, in one recorded by Traube, in which the pain for some time returned every night, lasting about five hours, but in which no calculus was ever voided) it seems more likely that the attacks are due to the presence of a stone in the renal pelvis, just as hepatic colic seems sometimes to arise from a gall-stone which has never left the gall-bladder. Again, precisely similar seizures may arise from the ureter becoming blocked in other ways; as, for instance, by small hydatid cysts, or even by clots of blood. Thus, in cases in which there is profuse hæmaturia, the occurrence

of violent pain along the course of the ureter must not be taken as establishing the fact that the bleeding is due to a calculus rather than to cancer. Lastly, in a remarkable case recorded by Dr Dickinson in the 'Pathological Society's Transactions' for 1875, an abdominal aneurysm over which the ureter was stretched gave rise for a long period to paroxysms of pain exactly like those of renal colic, and once attended with swelling of the testicle. In the immense majority of cases, however, renal colic is a symptom of stone in the pelvis of the kidney.

The *treatment* of renal colic consists mainly in the administration of anodynes, though something may perhaps be done to facilitate the release of the calculus. Thus, if the abdomen is not too tumid, friction may be used in the course of the ureter; or, while the affected side is being rubbed, the patient may be held head downwards, as was done by Sir James Simpson with success in two cases, the concretion apparently falling back into the pelvis of the kidney. Venesection was formerly had recourse to, and numerous leeches were employed; but it is better to avoid such methods of relieving what is essentially a transitory affection. A hot bath is often of service, or hot fomentations of the loins. But our chief reliance must be placed on the free use of opium. The hypodermic injection of morphia is to be preferred on account of the rapidity and certainty of its action, but if this should be objected to, and vomiting prevent a draught being retained, fifteen or twenty minims of tincture of opium may be thrown up into the rectum. The dose will probably have to be repeated at rather frequent intervals. The inhalation of chloroform often answers better than anything else.

4. *Suppression of urine.*—In the chapter on cholera (vol. i, p. 283) the fact was mentioned that the secretion of urine may, for a time, be completely suppressed, the patient voiding none, and none being found in the bladder when a catheter is passed. Poisoning by turpentine may produce a like effect; and in some remarkable instances it has been observed after an operation upon the urethra, or even after passing a catheter. It is also a symptom of suppurative nephritis, and of the most acute forms of Bright's disease. Such cases of "*non-obstructive suppression*" (as Roberts terms them) end fatally in a few hours or in a day or two, unless the kidneys resume their function. When the affection is recovered from, the urine that is first passed is scanty, high coloured, and generally albuminous or even bloody. The best treatment appears to consist in the use of the hot bath, or the application of hot mustard poultices to the loins, and the injection of hot gruel into the rectum.

In other cases, a temporary suppression of urine, without obstruction of the ureters, occurs as part of the general *shock* or collapse produced by the action of corrosive sublimate or of some other irritant poison, or by sudden lesions, such as perforation of the stomach, or rupture of the uterus. The renal affection is then relatively unimportant, passing off whenever the patient rallies, and generally leaving no ill-effects behind it.

According to Charcot, hysterical women are liable to a very different kind of suppression of urine, which he terms *hysterical ischuria*, and which may continue, almost without interruption, for a long time without seriously disturbing the health. He describes one of his patients as voiding less than a teaspoonful on an average each day for weeks together, whereas in the matters which she vomited urea was present. It is difficult to believe that

fraud was not practised in this and other like cases, although Charcot is convinced that he completely guarded himself against it.

A complete contrast to these cases is presented by *obstructive suppression*. When this occurs, the patient, instead of dying within a day or two, goes on for seven or eight days without obviously grave symptoms, so that both he himself and his friends find it difficult to imagine that there is danger. He is calm and free from distress, with an unclouded intellect, and with natural pulse, respiration, and temperature. He may be able to take food, the tongue may be clean, and there may be neither nausea nor vomiting. The muscular strength, however, begins to fail, and there is often marked sleeplessness. There is no desire to micturate, and sometimes no urine at all is voided. Generally, however, at very irregular intervals, the bladder discharges a few ounces, or sometimes a pint of urine. This is always pale and watery and of very low specific gravity; and, unless blood is mixed with it, it is usually free from albumen. At the end of about a week symptoms appear, which are commonly followed by a fatal termination within two or three days at the latest. The most distinctive of these are muscular twitchings, which Roberts says are never wanting. Contraction of the pupils also constantly occurs. The muscular weakness now rapidly increases; and, as a result of its involving the respiratory muscles, the breathing is slow, panting, and laborious. The appetite is entirely lost, and the tongue and the palate become dry. There is increasing drowsiness, with short fitful snatches of sleep, and a little rambling delirium. Convulsions and coma rarely set in, the intellect being commonly preserved to the last, so that the patient has in more than one instance spoken sensibly the instant before his death. Diarrhœa is of quite exceptional occurrence; and so is severe vomiting. The skin is moist, and often sweats profusely; there is never any ammoniacal or urinous odour from the surface of the skin or with the breath; nor does the body give off such odours after death. In one instance slight general anasarca was observed when the suppression first took place, but it passed off entirely on the third day.

The duration of life is stated by Roberts to be, as a rule, from nine to eleven days, and he remarks that the passing of a few ounces, or even of two or three pints, of a dilute urine does not seem to prolong it by more than a few hours. He knows of only three instances in which the patient survived beyond the eleventh day. In one of those cases, that of a man aged sixty-four, recorded by Rayer, death did not occur until the lapse of twenty-five days; another, that of a man aged seventy-three, recorded by Sir James Paget ('Clin. Soc. Trans.,' vol. ii) did not prove fatal for twenty-one days; the third, observed by Roberts himself, in a woman aged fifty-six, ended in death on the fifteenth day. The age of the patient does not appear to have any influence in accelerating or retarding the progress of the affection. Recovery has been known to occur in two or three cases in which there had been nearly complete suppression of urine for nine or ten days; in one of them the pupils had become contracted, or there was some mental confusion, but muscular twitchings had not made their appearance.

It is to Sir Wm. Roberts that we are indebted for the first complete account of the symptoms and causes of "obstructive suppression" of urine. But such cases had, of course, been observed before, although their characters had not been distinguished from those without obstruction. The case recorded by Sir Henry Halford, and cited in 'Watson's Lectures,' must have belonged to this category, although it was much more rapid in its course,

having apparently proved fatal in about three days. Sir Thomas Watson notes that patients affected with suppression of urine are chiefly persons who are advanced in life and inclined to corpulency.

The only instance at Guy's Hospital which was known to the author was that of a man aged forty-six, who in the year 1876 received a blow on the left side of the abdomen. This was followed by hæmaturia; two days later the urine became entirely suppressed and remained so until he died, seven days after the injury. In the course of the last twenty-four hours the muscles of his face were noticed to twitch, a profuse sweat broke out, and he became unconscious. At the autopsy one unusual feature was observed, namely, suppurative nephritis; and probably this accounts for its having reached a fatal termination more rapidly than those recorded by Roberts. But the cause of the suppression of urine was found to be exactly that which he believes to be almost always present, viz. obstruction of the ureter of one kidney by a calculus, when the other kidney is already incapable of secreting urine, owing to some antecedent lesion.

It is conceivable that both ureters might simultaneously be plugged with calculi; but the only other condition that can interfere with the flow of urine through both ureters at once (the two kidneys being healthy) and so cause an obstructive suppression of urine, is obliteration of their channels by pressure from without, as by cancer of the uterus or by some other disease of the pelvic organs, such as we shall find to produce hydronephrosis. Roberts relates a few cases of this kind which ended fatally, and one in which after no urine had been secreted for seven days it flowed again naturally during the remaining four weeks of the patient's life. But in most cases due to external compression, the renal cortex becomes atrophied or destroyed by hydronephrosis or consecutive Bright's disease before complete obstruction of the ureters occurs, so that the symptoms and course of obstructive suppression are seldom typical.

In the regular form of obstructive suppression dependent upon blocking of the ureter of the only functionally active kidney possessed by the patient, it is to be noted that the renal pelvis does not become dilated to a considerable extent, and that the quantity of urine accumulated by it is by no means large. The substance of the kidney was in one of Roberts's cases found to be much congested, but in another it was rather anæmic looking, though dotted on the surface with numerous blood-spots. No pathologist seems to have noticed in the human subject œdema of the kidney, and a deeply ecchymosed state of its pelvis, which Cohnheim described as the usual consequences of ligature of the ureter in animals. The kidney, however, is generally of about twice the normal size, having undergone hypertrophy as the result of the overwork thrown upon it by the obsolescence of the opposite kidney during the months or years that may have passed since the latter became unable to share in the excretion of the urine.

As already remarked, whatever urine is formed by a kidney of which the ureter has been blocked, is pale, of low specific gravity, and contains but a small percentage of urea. This is, perhaps, contrary to what one might have imagined to be the probable effect of such an occurrence, but it accords perfectly with the results of the experiments of Hermann upon animals. He showed that in dogs the secretion of urine appears to cease entirely under a pressure of 2·4 inches of mercury, and that when the pressure is removed the result is that a large quantity of watery urine is poured out, in which very little urea is present. Bartels relates the case of a young man

who had suffered from previous attacks of renal colic, and who in one such attack had suppression of urine for five days. When this passed off, he voided in twenty-four hours more than 3000 c.c., having a specific gravity of 1009, and containing numerous hyaline casts as well as albumen. Most observers seem now to think that the cessation of the activity of the kidney as soon as the pressure in the ureter and renal pelvis reaches a certain point is more apparent than real, the urine being really secreted, but re-absorbed as fast as it is formed. We have seen (p. 623) what an important bearing this view has upon the theory of uræmia, when taken in connection with the absence of the usual uræmic symptoms in obstructive suppression.

In the *treatment* of suppression of urine, when it appears to be due to impaction of a calculus in one ureter, recourse should be had to those measures which we have seen to be sometimes effectual in aiding its expulsion downwards into the bladder, or its return upwards into the pelvis of the kidney, when there is renal colic. The abdomen in the course of the ureter may be well rubbed and kneaded, while the patient is in various positions,—standing, or lying, or inverted with his head downwards. But it is to be feared that the absence of pain in such cases means that the peristaltic movements of the ureter itself have ceased, and therefore that there is little chance of success from such means. In two of Roberts's cases it is expressly noted that soon after the secretion of urine ceased, the pain of which the patient had been complaining disappeared entirely. Consequently, it does not appear hopeful to employ hot baths, or chloroform inhalations, or anodynes of any kind, for the purpose of relaxing spasm.

There is, however, one method of treatment which seems never yet to have been attempted, but which deserves a trial. It is that of cutting down upon the kidney in the loin, and incising the ureter in the renal pelvis, so as to allow whatever fluid may be collected there to escape. The removal of pressure would probably at once be followed by an abundant secretion, and it is possible that a permanent fistulous opening in the loin might be created. Such an operation may not indeed be justifiable during the first few days after the suppression of urine has set in, on account of the possibility of spontaneous recovery; but there certainly can be no objection to it when at the end of a week muscular twitchings begin to appear. It is also a question whether it may not be practicable for the surgeon to remove an impacted calculus from the ureter. In the case that occurred in 1876 (see p. 671) the stone was found on *post-mortem* examination impacted four inches from the kidney. But in two cases recorded by Roberts it lay just within the vesical orifice of the ureter. Might it not be felt in such a position *per rectum*, and possibly be set free by an incision?

5. *Unilateral atrophy of the kidney.*—It is not at all uncommon in the *post-mortem* room to find one kidney shrunken to a mere thin flat relic, scarcely if at all bigger than the adjacent adrenal body, and weighing about an ounce or an ounce and a half. Twenty cases of this kind were collected by the author from our records at Guy's Hospital; and no doubt this number has since considerably increased. In several instances, the cause of death was some disease entirely unconnected with the urinary organs. The secretion of urine, in fact, takes place quite naturally under such circumstances, because the opposite kidney undergoes a compensatory enlargement, becoming as heavy as the two organs together normally should be.

The nature of the process by which this enlargement is effected has been studied by different observers with discrepant results. Perl ('Virchow's Arch.,' vol. 56) found increase in size of the convoluted tubes and epithelium but not of the glomeruli. Beumer (*ibid.*, vol. 72) could find no demonstrable increase in size, whether in the glomeruli or in any of the tubes, so that according to the strict terminology of Virchow, the compensatory change would be a *hyperplasia* rather than a *hypertrophy*.

However, the presence of an atrophied kidney is not without its effect, even when the kidney on the opposite side has thus become enlarged, so that it can secrete the full normal quantity of urine. For, if the ureter of the latter should, from any cause, become obstructed, the necessary result, as we have already seen, is suppression of urine, instead of a mere attack of renal colic. Again, laceration of the region by violence is very likely to be followed by fatal results, as happened some years ago, in the case of a boy, admitted into the accident ward of Guy's Hospital, although during life it appeared a mystery why a unilateral injury should have such a serious effect. Moreover, the tissue of a kidney enlarged by compensating hypertrophy seems to be unduly liable to Bright's disease; at any rate, in about one fourth of the above twenty cases there has been chronic nephritis of the hypertrophied kidney. Perhaps the compensation is not always perfect, and so the enlarged organ cannot for an indefinite length of time do the entire work of secreting urine without danger. The period of life at which the atrophy occurs might naturally be supposed to make a difference in the completeness of the hyperplasia; and, indeed, it is to be observed that Beumer's observations were made in a case in which one kidney was congenitally absent, so that their applicability to cases of acquired atrophy may after all be disputed. Among forty-eight instances of congenital absence of one kidney, collected by Beumer from different sources, there were no fewer than twenty in which the opposite kidney was found diseased. It most often was the seat of "chronic inflammation," but in many instances it contained calculi in the renal pelvis. It is worth noting in this connection that congenital absence of the kidney is probably much more rare than an acquired atrophy; it is accompanied by absence of the ureter and the renal artery.

Acquired unilateral atrophy of the kidney is due to various causes. In three of our twenty cases a calculus was found impacted in the corresponding ureter, and in two other cases calculi were present in the renal pelvis. In none of the remaining cases was any concretion found, nor was there any obstruction to the outflow of urine from the renal pelvis. Yet the pelvis and the calyces were dilated in no fewer than nine of them, and in two the ureter was dilated and thickened all the way down to the bladder. It seems difficult to avoid the inference that there had at some former period of the patient's life been a renal calculus, which either escaped through the natural passages or underwent disintegration, but which had deranged the kidney sufficiently to cause it to waste. This conclusion is greatly strengthened by the fact that in two other cases there was a history of lithotomy many years previously; in one of them the operation had been performed by Sir Astley Cooper when the patient was aged thirteen, forty-five years before his death. Lastly, in one instance in which neither the renal pelvis nor the ureter was enlarged, the vesical orifice of that tube was considerably lower than that on the opposite side, and lay nearer to the prostate, as though it had been forced downwards in the expulsion of a concretion.

A very much rarer effect of the presence of a calculus in the renal pelvis is the replacement of the substance of the kidney by a mass of adipose tissue, having the shape of the healthy organ, and of about the same size, as in a case described and figured by Dr Rickards, of Birmingham, in the 'Brit. Med. Journ.' for July 7th, 1883.

6. *Hydronephrosis.*—We have seen that plugging of a ureter by a calculus, or obstruction of both ureters as the result of morbid processes of various kinds, does not necessarily lead to any considerable accumulation of fluid in those tubes, or in the renal pelvis. There are, however, cases in which such an accumulation occurs, and for these the name of *hydronephrosis*, originally suggested by Rayer, appears to be the most suitable, especially since it serves to distinguish them from cases of cystic disease of the kidneys, such as will be described afterwards.

Anatomy.—The earliest indication that the organ has been subjected to pressure from within is a diminution in the mammillary apices of the pyramids, which, instead of nearly filling the calyces, become separated from each other by broad intervals, and ultimately flattened, or even hollowed out. With the microscope it may be seen that the tubes in the remains of the pyramids are bent into a regular series of wave-like curves. As this change in the pyramids goes on, the calyces and the renal pelvis undergo dilatation. Sometimes the calyces stretch out of the hilus of the organ, so that the pelvis forms a sac situated nearer to the middle line of the body than the kidney itself, and sending finger-like processes into it; in a case that occurred at Guy's Hospital in 1876 such a sac lying beyond the kidney was found to hold a pint of fluid. Much more frequently as the calyces and pelvis yield before the pressure of their contents, each calyx forms a somewhat egg-shaped cavity, communicating with the pelvis by a smooth orifice, and separated from the adjacent calyces by a tough fibrous membrane; the surface of the organ acquires a lobulated appearance, the lobules corresponding in number with these cavities; or, if the sac is very large, the septa between them may become perforated, so that they may at last break down and form a single cavity.

In the meantime the secreting substance of the organ passes into the condition described as consecutive Bright's disease, or undergoes atrophy, until at length no trace of it can be discovered, or at most only a few scattered relics here and there in the walls of the sac. The ureter, too, may be dilated until it is as large as the finger of a glove, or a coil of small intestine. In one or two instances it has actually been felt during life as an abdominal tumour.

Secretion.—The nature of the fluid contained in the sac of hydronephrosis varies in different cases. When the enlargement is but slight, as in most instances in which both kidneys are affected, it is still more or less dilute urine, which, however, may contain albumen, or be mixed with pus or blood. In those extreme cases which are generally unilateral, the fluid is sometimes pale and clear, sometimes stained with blood. It is usually of lower specific gravity than normal urine, being in this respect like the fluid secreted in cases of "obstructive suppression." But in a remarkable case operated on by Czerny it must have had absolutely the same characters as the secretion of healthy kidneys, for the urine passed by the patient was in all respects natural, and yet extirpation of the hydronephrotic organ was followed by complete and fatal anuria, and on *post-mortem* examination it

turned out that the opposite kidney had undergone atrophy and that its ureter was obliterated.

The solid matters dissolved in this fluid are generally urea, uric acid, and salts of the same composition as those that are found in urine. But in a case that came under the author's observation in 1876 neither urea nor uric acid could be detected in the fluid removed by tapping from a tumour believed to be hydronephrotic; and Sir Spencer Wells and Mr Cooper Rose ('Lancet,' 1868) have also met with instances in which urea has been absent. Mr Henry Morris ('Med.-Chir. Trans.,' 1876) cites cases in which the contents of hydronephrotic sacs in the fœtus have been devoid of urea. Albumen is commonly present in greater or less quantity. In some cases the fluid has been purulent, as in one described by the editor ('Path. Soc. Trans.,' xxiii) in which six and a half pints of an opaque reddish fluid were drawn off by a trocar: the disease may then be called pyonephrosis, if a special name is needed. Dr Dickinson ('Path. Soc. Trans.,' xiii) has recorded a case in which a very large sac contained a gelatinous or colloid substance.

Ætiology.—As already observed, the causes of hydronephrosis are often such as affect *both kidneys* simultaneously. Among these may be enumerated stricture of the urethra, enlargement of the prostate, and various vesical affections (including villous disease of the bladder); also pregnancy, prolapsus, or retroflexion of the uterus, and pelvic tumours, especially cancer of the womb involving the surrounding tissues or the iliac glands.

Cohnheim has recorded a remarkable case in a rachitic boy of eleven with contracted pelvis, in whom double hydronephrosis was produced by the pressure of an enormously dilated rectum and sigmoid flexure. In such cases the renal affection is usually more marked on one side than the other. But there is almost always so much interference with the secreting action of the two kidneys that death occurs from such interference (if not from the primary disease) before the sac has become large enough to constitute an abdominal tumour capable of recognition during life. Mr Morris, however, relates a case of villous disease of the bladder in which a rounded swelling, of the size of the head of a small fœtus, was felt in the right loin. As a rule, the only clinical evidence of the renal affection is a pale watery condition of the urine, until perhaps convulsions or other uræmic symptoms set in, and rapidly bring about a fatal termination. For example, in 1871, a woman aged thirty-six was lying in the uterine ward of Guy's Hospital with cancer, when she began to complain of severe headache; after two days she screamed out violently in the night, and became unconscious; and in this state she remained until her death three days later. The autopsy showed that the cause of her symptoms was not cerebral hæmorrhage (as had been suspected), but uræmia: each kidney had its pelvis greatly dilated, its pyramids flattened, and its cortex pale, though not decidedly narrowed. In 1869 a woman aged thirty-eight was admitted into our clinical ward shivering violently and very cold, with a dry brown tongue and other typhoid symptoms, but with her mind clear. She was said to have had prolapse of the uterus for a year, and her urine was found to contain pus. She died two days later, her temperature having been very low throughout. On *post-mortem* examination it was found that the womb had dragged down the vesical extremities of the ureters, and compressed them against the pubic arch. There was hydronephrosis on both sides, and the cortex of each kidney was greatly atrophied.

If the cause of the hydronephrosis is so situated as to affect the ureter

leading from one kidney only, it may produce a tumour of very large size. The opposite kidney then undergoes hypertrophy, and, as it may carry on the secretion of urine perfectly, there is nothing to prevent the development of the hydronephrosis to any conceivable extent.

Of the lesions that may affect one ureter so as to cause a *unilateral* hydronephrosis, the most obvious is obstruction by a calculus. Thus in 1877 a man aged forty-six died in Guy's Hospital of dropsy due to Bright's disease affecting a hypertrophied left kidney; in the right ureter there was impacted a mulberry concretion an inch and three quarters in circumference; the right kidney was converted into a shining loculated cyst, with a smooth lining, upon which there was one little patch of renal substance about as large as a shilling still remaining. It is of course impossible that, after both ureters have been completely blocked by stones, the patient should live long enough to admit of the development of double hydronephrosis. But in 1874 a boy aged six was in the hospital for stone in the bladder, when he died of tonsillitis. Each ureter was greatly dilated and also the pelvis of each kidney. The right ureter was blocked by a second small calculus about an inch above its orifice; the left was free, so that the distension on that side had to be attributed either to interference with the downward flow of urine resulting from the vesical calculus, or else to the passage of that calculus at a time when the right ureter was free, or at least not entirely obstructed. In 1857 there died in the hospital a woman aged fifty-six, who had a large fluctuating swelling in the left loin, and a smaller one in the right loin. Hydronephrosis was found to be present on both sides, and the pelvis of each kidney contained calculi, but it was only on the left side that impaction of a calculus in the ureter had taken place. This patient had been passing blood and pus in her urine all the while she was in the ward; but in most cases of hydronephrosis due to impaction of a calculus the urine is perfectly normal, though a history of former attacks of renal colic may perhaps be elicited, sometimes very far back. Rayer recorded a case of hydronephrosis in a man aged sixty-four, who had for a long series of years enjoyed perfect health, but who at the age of twenty-two had had an illness attended with pain in the right kidney and along the ureter, and with hæmaturia.

Another cause of unilateral hydronephrosis is compression of the ureter, generally near the brim of the pelvis, by a thickened peritoneal band, the result of inflammation of the serous membrane. And sometimes the ureter is thickened and narrowed by changes in its own coats, the origin of which is no longer discoverable when the case comes to an autopsy, probably many years after their occurrence. A case of the editor's ('Path. Trans.,' xxiii) in which the ureter was found to be obliterated about an inch and a half below the pelvis of the kidney, appeared to be clearly traceable to a kick from a horse about two years previously; the injury had been followed at the time by hæmaturia. A similar instance in which hydronephrosis in a boy of twelve was directly traceable to a fall, has been recorded by Mr Croft ('Trans. Clin. Soc.,' xiv). In 1873 an autopsy in the case of a boy aged four, who died with a calculus in his bladder in Guy's Hospital, showed the ureter as thick as a lead-pencil, and completely occluded by an oblique cicatrix about an inch from its origin.

There are, however, many instances in which no cause for the hydronephrosis can be made out, the ureter appearing perfectly free from obstruction in its whole course from the renal pelvis to the bladder. In most such

instances there has probably been at some former period a calculus, which has in the meantime undergone disintegration or has been voided.

With regard to this question, the facts adduced at p. 672, in reference to the origin of atrophy of the kidney, are of much importance. It is possible that some conditions regarded by Roberts and other observers as occasional causes of hydronephrosis are not really so, but that here also the true cause is a calculus. One of these hypothetical causes is compression of the ureter by a supernumerary renal artery. Another is obliquity of the origin of the ureter from the renal pelvis, causing a valve-like impediment. That such an appearance is not infrequently met with is certain, and Dr Hare has recorded ('Med. Times and Gaz.,' 1857) a case in which the ureter on each side was coiled on itself—like a turn and a half of a corkscrew brought closely together—and adherent to the lower part of the sac. There can be no doubt that this arrangement of the ureter is the cause of the "intermitting" character of many hydronephrotic tumours, as well as of the fact that after paracentesis the ureter sometimes becomes for a time pervious. What seems doubtful is whether cases of this kind are congenital, or whether the twisting of the tube is not a secondary result of its distension, just as one finds the duct of the gall-bladder distorted to an even greater extent and bound down by adhesions as the result of the passage of gall-stones through it. The author has twice seen such a valvular condition of the upper orifice of the ureter when there was obstruction of the lower urinary passages; once in the case of an old man who died of the effects of stricture of the urethra, and in whom, although the ureter was not dilated, the pelvis of the left kidney formed a large pouch full of dark-brown fœtid fluid; and again in a fatal case of lithotomy, complicated with stricture.*

A point upon which Cohnheim lays stress is the origin of the ureter from the side of the renal pelvis instead of from its lower end. The result of this, he says, is that so long as the patient is in an upright position the bladder receives only so much urine as overflows from the kidney. And he mentions the case of a woman so affected who for a long time passed scarcely any urine during the day, whereas she voided large quantities at night. This, however, surely proves too much, unless indeed the hydronephrosis was very considerable; and it must not be forgotten that in renal cirrhosis (to which the consecutive Bright's disease of hydronephrosis is very analogous) the nocturnal flow of urine is often excessive. Cohnheim himself observes that before such cases come to an autopsy the conditions are so altered by the dilatation of the pelvis of the kidney as to render it impossible to say how the affection began. That hydronephrosis itself is sometimes congenital is well known; some cases in which the abdomen has been large from the time of birth have been prolonged for years, although they far more often terminate fatally within the first few days or weeks. But congenital hydronephrosis is traceable to some definite malformation, such as closure of a ureter, or more rarely of the urethra. The cases in question do not lend support to the view that obliquity or twisting of the upper end of the ureter, occurring as a malformation, can give rise to hydronephrosis. One point worthy of notice about the congenital form of the disease is that it is often associated with harelip, imperforate anus, club-foot, and other defects of development. The fact that closure of the outlets of the kidneys causes during intra-uterine life an accumulation of fluid seems

* Dr Sainsbury has described a case of hydronephrosis from two valvular folds of mucous membrane at the origin of the ureter ('Path. Trans.,' 1886, p. 296).

to show that the secreting function of those organs must already be in a state of activity; and in a paper read before the Royal Medical and Chirurgical Society in 1876 ('Med.-Chir. Trans.,' vol. lix, p. 98) Mr Morris has argued for the view that they normally pour urine into the *liquor amnii*, whence it is absorbed into the blood of the mother, to be afterwards re-excreted by her urinary organs.

In a case that occurred at Guy's Hospital in 1868, Dr Moxon suggested a cause for hydronephrosis that, so far as I know, has not been observed by other pathologists. The left kidney was found by him after death to have the pyramids flattened, the pelvis and the calyces dilated. The patient was a man aged twenty-two, who suffered from a lumbar abscess, and lay constantly on his left side with his pelvis raised upon an air-pillow, so that the tendency of fluid to gravitate within the ureter must have been from the bladder to the kidney, and not in the reverse direction.

Diagnosis.—Many cases are on record in which *single hydronephrosis* has been mistaken for a large ovarian tumour, or even for ascites. The most remarkable of them all is perhaps one related by Mr Glass in the 'Philosophical Transactions' for 1747. The patient was a woman aged twenty-three at the time of her death, who had been dropsical from birth; the abdomen then measured 6 feet 1 inch in circumference, and the sac contained thirty gallons of fluid. In several other instances many pints have been taken from a hydronephrotic tumour during life, or have been found in it on *post-mortem* examination. Among the points which should distinguish such a tumour from an ovarian cyst are its having first made its appearance in the loin rather than near the pelvic brim, its having no pelvic connections, the presence of the colon in front of it, and the absence of resonant intestine in the loin. It has, in fact, all the characters of a renal tumour. Fluctuation is generally well marked, and the outline of the swelling is sometimes lobulated. It may occupy a large part or the whole of one side of the abdomen, extending across beyond the umbilicus, and downwards into the iliac fossa.

In a patient under the author's care in 1883 there was a prominence in the epigastric and in the left hypochondriac region, while in the loin the bulging was but slight; and as there was obvious pulsation, with an audible bruit, the case looked like one of abdominal aneurysm. In that instance the history given by the patient himself contained one point which, if duly attended to, would have cleared up the diagnosis. He said that on more than one occasion after the first appearance of the swelling, it had undergone a great diminution in size; he had not, however, noticed that at those times there was any increased flow of urine, nor that the urine was altered in appearance. This spontaneous subsidence or disappearance of the tumour, when it is observed, is by far the most important clinical character of hydronephrosis. If associated with an excessive discharge of fluid from the bladder, it may be said to be pathognomonic. Even without that corroborative evidence, the only cases in which a similar occurrence is likely to be met with are those in which an ovarian cyst ruptures into the uterus or into the intestine; and such events are probably always indicated by the escape of fluid through the vagina in the one case, or the entrance of air into the cyst in the other.

Two other affections may be mistaken for single hydronephrosis, namely, hydatid of the kidney and renal cyst. Each of them is very rare, at least as giving rise to a palpable swelling. The former is almost certain to be set down to hydronephrosis, unless its nature is revealed by the escape

of daughter-cysts through the urethra, or by the characters of the fluid removed by paracentesis. The latter could probably be distinguished only after extirpation or on *post-mortem* examination. Two striking instances are recorded by Mr Cæsar Hawkins ('Med.-Chir. Trans.,' xviii) and by Dr Hare ('Path. Soc. Trans.,' iv); in each of them the tumour filled the right side of the abdomen. Three or four other cases are cited by Czerny in his list of cases of nephrectomy ('Trans. Internat. Congress,' 1881).

Neither pain nor tenderness is necessarily present in hydronephrosis, though when the swelling is large it often causes a distressing sensation of fulness or distension. In some cases pricking or shooting pains are complained of, which are perhaps due to local inflammatory changes in the peritoneum covering the sac. The colon is sometimes tightly stretched over the tumour, in such a way as to interfere with the free passage of its contents; thus in a case recorded by Roberts the chief symptoms were at first those of intestinal obstruction, which recurred again and again during several years.

Double hydronephrosis can only be surmised when the conditions which produce it are present, along with symptoms like those of advanced Bright's disease. Two lumbar tumours are very rarely felt.

Prognosis.—When hydronephrosis is bilateral, the patient is always in danger, since the structure as well as the functions of the secreting tissue of the two kidneys is inevitably interfered with; and in many cases the primary disease that has caused the obstruction to the escape of urine would in itself prove rapidly fatal, even without any such complication.

On the other hand, the course of unilateral hydronephrosis is commonly very chronic, and it scarcely ever in itself brings life to an end. In the case recorded by Mr Glass, death was apparently due to pressure on the diaphragm and displacement of the thoracic viscera. In one observed by Mr Thompson, of Nottingham ('Path. Trans.,' xiii), it resulted from peritonitis set up by escape of the contents of the sac through an ulcerated aperture. In the editor's case (p. 675) there had been communication with some part of the intestine, for the sac which had suppurated contained a mass of vegetable fibre, with bits of apple-core and part of a clove. In other fatal cases that have been recorded the cause of death has generally been either an independent disease (as, for example, acute tuberculosis in a case of Dr Hillier's) or else the supervention of some morbid process in the hypertrophied kidney on the opposite side of the body. Consequently, it is not advisable to interfere actively with hydronephrosis, until the patient is unable to bear the pain and discomfort to which its presence may give rise.

Treatment.—In some few instances one can succeed in emptying the sac by rubbing the abdomen. Dr Roberts relates the case of a girl of eight, who came under his care with a soft fluctuating tumour in the left side, of about the size of a child's head. This was diligently manipulated in every direction, with the aid of a lubricating ointment, on alternate mornings. After the third time she suddenly passed abundant urine, the tumour forthwith subsided, and did not reappear while she remained under observation. A somewhat similar result was attained in a case recorded by Dr Broadbent ('Path. Soc. Trans.,' xvi) of double congenital hydronephrosis in an infant.

But when the sac is tense little can be hoped for from such a procedure; and there is often so much tenderness that it cannot be adopted. The only treatment then is to puncture the sac with a trocar. On the left side this may be done at a spot just anterior to the last intercostal

space. But on the right side Mr Morris has shown ('Med-Chir. Trans.,' lix) that there is danger of wounding the liver, and he advises that a point should be selected half way between the last rib and the crest of the ilium, and from two inches to two and a half inches behind the anterior superior spine. After the operation, fluid like that which has been withdrawn from the tumour sometimes passes for a time with the urine, showing that the ureter has again become pervious. But the sac almost always rapidly fills again, and may soon regain the same size as before. Thus, in a case of double hydronephrosis, which was three times tapped by Fränkel, the patient did not micturate at all during from twelve to forty-eight hours after each tapping, the whole of the fluid secreted in the interval having doubtless accumulated in the two sacs. It is true that unilateral hydronephrosis is commonly attended with such extreme destruction of the renal cortex that the organ can hardly be supposed still capable of forming urine. But experience seems to shows that even in such cases fluid continues to be poured out by the walls of the sac. One of the few cases in which repeated puncture has led to permanent shrinking is that of Mr Croft already referred to ('Trans. Clin. Soc.,' xiv). In that instance, within fifty-four days of the accident which caused the disease, seventy-nine ounces of fluid had already collected. Paracentesis was performed eight times altogether, from three to four pints being removed each time. After the eighth operation, which occurred at three months' interval from the first, no further accumulation took place. In a case observed by Sir Spencer Wells ('Dubl. Quar. Jour.,' 1867) the patient, two months after a second tapping, passed two calculi per urethram, after which the tumour completely disappeared and did not return. On the other hand, there does not appear to be much fear of setting up suppuration in the sac by paracentesis, though this result has been known ('Path. Soc. Trans.,' xiii, Dr Little's case) to follow the attempt to cure the disease by making a fistulous opening. Czerny mentions ('Trans. Internat. Congress, 1881') the case of a man in whom Gustav Simon had two years previously made such an opening, and who in 1881 was continuing to act as an attendant in the wards of the surgical clinique at Heidelberg.

Czerny, in his statistics of nephrectomy (loc. cit., p. 249), gives twelve cases in which that operation has been performed for hydronephrosis or for cyst of the kidney. Seven of them ended fatally, but this high mortality may perhaps be in part attributed to the fact that in five an erroneous diagnosis of ovarian tumour had been made. According to Mr Barker a lumbar incision is in cases of this kind preferable to one in the front of the abdomen. Czerny's case referred to at p. 674, shows the importance of ascertaining that the opposite kidney retains its functional integrity. Probably this may be best done by making a preliminary opening into the sac, and allowing it to drain, so that after one can be certain that no fluid from it any longer descends the ureter, one can measure and test the urine passing into the bladder from the other organ.

7. *Pyuria and pyelitis.*—We have now to deal with those cases in which the presence of a stone in the kidney causes pus to appear in the urine, by giving rise to suppuration of the renal pelvis. But we must first digress so far as to consider the general subject of *pyuria* and briefly to indicate the various affections of the urinary organs that may lead to this symptom.

The presence of pus in urine commonly gives it a turbid opaque appearance, and on standing there is precipitated a dense whitish-yellow sediment

which can only be mistaken for one of amorphous phosphate of lime or of the mixed amorphous urates, but may be distinguished from them by its microscopical characters, and also by remaining undissolved both after the addition of an acid and also when the fluid is warmed. In urine which has undergone the ammoniacal fermentation pus assumes a different character; it forms a viscid, tenacious substance, which glides out as a coherent mass when the fluid is poured from one vessel into another.

Formerly this was spoken of as mucus, but it really always consists of altered pus. It often causes much pain and distress in passing through the urethra. One can artificially produce the same change, whenever urine contains any considerable quantity of pus, by adding solution of potass or ammonia to the sediment in a test-tube, and shaking it: the pus at once loses its opaque yellow appearance, and becomes viscid. This in fact constitutes the one chemical test for pus, a test which in this country is commonly associated with the name of Dr Babington, though Leube attributes it to Donné. It is not applicable when the amount of deposit is small. In that case the microscope at once clears up any doubt as to its nature. It is an interesting fact that leucocytes in urine, even when it is alkaline and full of bacteria, sometimes retain their amœboid movements.

Whenever there is pus in urine, there is also albumen, derived from the liquor puris. But if the quantity of pus is small, the albumen may not be discoverable by ordinary tests. It is often an important practical question to determine whether the amount of albumen observed in urine containing pus is or is not greater than the pus itself accounts for; since, if it is greater, it affords evidence of the existence of Bright's disease in addition to the affection causing the pyuria. In various surgical affections of the urinary organs, the propriety of operative interference depends largely upon this point. To help to determine it, Leube has published the following experiments made under his direction. Having added to urine 2 per cent. of pus, he found that in every microscopic field, prepared with fluid that had not been allowed to settle, there were from ten to fifteen leucocytes, and that the amount of albumen precipitated by boiling occupied about one tenth of the bulk of the urine. His conclusion is that a coagulum of even one twentieth or one twenty-fifth is more than can be attributed to pus, unless at least some few pus-corpuscles are visible in each microscopic field. Tube-casts should also be carefully looked for.

Apart from calculus, pyuria may be due to a great variety of affections. The possible presence of gonorrhœa must never be forgotton; nor, in females, that of leucorrhœa, which, however, is indicated by a large number of squamous epithelial cells, as well as of leucocytes, that are seen under the microscope. Cystitis, again, is a frequent cause, and one should remember that, as the result of inflammation of the bladder, there may sometimes be a good deal of pus in the urine without the patient complaining much of pain or having to micturate very frequently, especially if he has a stricture or an enlarged prostate.

Whatever may be the origin of cystitis, it is apt sooner or later to lead to an extension of inflammation upwards to one or both of the renal pelves. Thus, pyelitis is of frequent occurrence as part of the widespread change in the lining of the urinary organs that results from an ammoniacal decomposition of the urine within the body. It may also accompany various surgical diseases of the lower urinary passages. It may also be produced in association with nephritis by certain poisons, of which cantharides and turpentine

are the chief. And slight forms of it are seen in connection with Bright's disease, and also (it is said) as the result of diabetes, or during the course of enteric fever and of other specific diseases. Again, it sometimes follows a blow or other injury to the loin. And, as Kaltenbach has particularly pointed out ('Arch. f. Gynäk.,' iii), it may develop itself after parturition as the result perhaps of extension of inflammation from the pelvic organs. But none of these instances afford an example of the occurrence of a persistent and severe pyelitis independently of a like affection of other parts of the urinary tract of mucous membrane. So that if we exclude tubercular cases (which will be described separately) we need perhaps admit no other cause than gravel or calculus for such forms of pyelitis as require special clinical recognition. It is true that one occasionally fails to discover any concretions in cases which have ended fatally after having been of long standing, or in which a surgical operation affords an opportunity of thoroughly exploring the diseased organ. But we have already found grounds for the belief that either unilateral atrophy of the kidney or hydronephrosis may result from calculi which subsequently disappear, and the same may also be true of pyelitis.

The symptoms that characterise "calculous pyelitis" are more or less severe pain in the loin or in the abdomen, hæmaturia which generally recurs from time to time, and more or less constant pyuria. A case in point was under the author's observation for some years. The patient felt, in 1876, a slight pain or uneasy sensation in the left loin, for which no cause could be found. A short time afterwards he noticed some blood in his urine; but, on taking medicines which rendered it alkaline, the hæmaturia ceased. Yet from that time the urine almost constantly contained pus in small quantity, with apparently an excess of albumen; and crystals of oxalate of lime were usually to be detected, sometimes crystals of lithic acid. In 1880 he passed a small oxalate calculus, after which he was more free from pain than he had been for some years previously. All along the general health was good, and the patient was able to discharge responsible duties.

It very rarely happens that the disease assumes so mild a form as this. Generally speaking rigors recur from time to time; sometimes with regular quotidian periodicity. There is often considerable pyrexia, which may assume a hectic type. Diarrhœa may be persistent and intractable; or there may be obstinate constipation from adhesion of the colon to the anterior surface of the affected kidney. When pyelitis runs on for a length of time the renal pelvis often becomes dilated into a large sac, which may be felt as an abdominal tumour, and may bulge into the loin as an elastic fluctuating mass, very painful and tender to the touch. If the ureter becomes from time to time blocked this swelling may present great variations in size on different occasions, and there may be converse variations in the degree of pyuria, the urine being perhaps clear when the swelling is largest, whereas a subsidence of it may be accompanied by the escape of several ounces of pus into the bladder. In such cases of "pyonephrosis" the renal cortex probably always undergoes atrophy, or becomes shrunken by a process of consecutive Bright's disease. If there are calculi in both kidneys, as is often the case, this morbid process is of course quite sufficient to destroy life, with symptoms of uræmia. And even when the affection is limited to one side, the opposite kidney may, after undergoing hypertrophy, become affected with Bright's disease, either as the result of lardaceous changes in it, or independently of any such changes; but in other cases, after lasting a cer-

tain length of time, the inflammation subsides, and the kidney shrinks and dries up into a putty-like mass.

Again, many cases of pyelitis end fatally by the supervention of *perinephric abscess.* Inflammation probably never affects the renal pelvis for any considerable length of time, nor with any great degree of severity, without extending to the surrounding structures, which become indurated and matted together by new fibroid material; but in many instances, after a while, the mucous membrane undergoes ulceration, and perforation with escape of urine and of pus takes place into the connective tissue. When this occurs there is usually a marked increase in the pyrexia and in the other general symptoms that may have previously resulted from the pyelitis. A fluctuating swelling may appear in the loins, with extreme local tenderness; and ultimately the skin may become reddened, and the abscess, if not opened by the surgeon, may point and break of its own accord; but in other cases the course taken by the suppuration is different. It may enter the sheath of the psoas muscle and make its way downwards into the groin, and even penetrate the hip-joint. A point on which Trousseau laid stress is that when the psoas is affected the thigh is kept more or less rigidly flexed upon the pelvis; but in many instances it is impossible to determine during life the renal origin of cases of this kind. Or the pus may extend in front of the iliacus muscle, and point above Poupart's ligament. Or it may penetrate into the intestine; in such cases gas and fæcal matter often escape into the abscess-cavity, and subcutaneous emphysema may develop itself in the back, as was observed in two instances by Trousseau. Lastly, a perinephric abscess may burrow through the diaphragm and the lung, and discharge itself by the bronchial tubes. Conversely, an abscess starting from some other abdominal viscus may make its way into the urinary passages.

When there is free discharge of pus in the loin, the inflammation sometimes gradually subsides, and recovery ensues. But as a rule, the prognosis of perinephric abscess is unfavourable, the patient becoming worn out by the drain of pus, by severe pain and hectic fever, or by lardaceous disease.

8. *Suppurative nephritis.*—This consists in the presence of more or less numerous foci of inflammation within the kidney-substance, which appear upon the surface as minute round or irregular dots, and upon section of the organ as streaks, or lines, traversing the cortex to a greater or less depth, or running continuously through its whole thickness as well as through the corresponding medulla. At an advanced stage there is well-formed creamy pus; when the disease is fatal at an early period, there is often only a soft pinkish-white material, which consists of kidney-tissue infiltrated with leucocytes, but not yet completely destroyed. Surrounding the infiltrated or suppurating tracts there is much vascular injection. Sometimes only one or two points of even commencing suppuration are discoverable; so that they are not unlikely to be overlooked. There is reason to believe that the recognition of even a single point of acute inflammation of the renal cortex proves the existence of a morbid change sufficient to account for death.

The *causes* of suppurative nephritis vary. Sometimes it occurs as part of general pyæmia; and Dr Moxon noticed that in cases of pyæmia resulting from perineal section or lithotomy, abscesses in the kidneys were more apt to occur than when pyæmia was due to lesions unconnected with

the urinary organs. This observation is interesting, because some pathologists have been disposed to refer suppurative nephritis in general, even when obviously traceable to an inflammatory process spreading upwards from the bladder along the ureters, to an infection with some septic poison from without. Thus Dr Goodhart, in vol. xix of the 'Guy's Hospital Reports,' endeavoured to trace a connection between this affection and the presence of erysipelas in other cases in the same ward and at the same time. Moreover, as a matter of fact, the remarkable decline in the frequency of pyæmia in our wards has been accompanied by a corresponding decline in the frequency of suppurative nephritis.

In some cases suppurative nephritis seems to occur as a primary morbid change. Dr Goodhart's paper recorded three instances, in each of which, although some degree of cystitis was found at the autopsy, it seemed doubtful whether this was sufficient to account for so severe an affection of the kidneys; one was a case of enteric fever, another of mitral disease, and the third of extensive burns.

But in the vast majority of instances suppurative nephritis is secondary to an affection of the urinary passages; either to some one of the common surgical diseases of the urethra or of the bladder, or else to paralysis of the bladder from some spinal lesion, or to compression of the ureter as the result of cancer or other disease of the uterus. As might be expected, most cases of this kind are also marked by the mechanical effects of obstruction to the outflow of urine from the renal pelvis, on one or both sides, which have been fully described already. The suppurative nephritis itself is not always bilateral. In some instances the whole length of the urinary tract is obviously affected with inflammation, from the bladder to the mucous membrane covering the renal pyramids. But in others the lining of the ureter and of the renal pelvis is normal. Dr Dickinson is of opinion ('Med.-Chir. Trans.,' lvi) that the exciting cause of the nephritis is really the ammoniacal state of the urine resulting from its decomposition within the urinary passages, and holds that suppurative nephritis as a secondary affection never occurs except when the urine has undergone this change. How rapidly the disease may develop itself is well shown by a case which he narrates of an old woman admitted into the hospital for a fracture of the femur, who two days later became unable to pass her water, so that a catheter had to be used. The urine drawn off was then natural, but very soon afterwards the urine became offensive, and death occurred within a week of the accident, three days after the urine had changed its character. Both kidneys were found to be suppurating.

The *symptoms* of suppurative nephritis are obscure. Dr Dickinson describes rigors as an early and frequent symptom: febrile symptoms rapidly follow, with vomiting, great prostration, feeble pulse, dry, brown tongue, and sometimes profuse sweating or diarrhœa. Convulsions are rare, and the case ends in stupor rather than coma. But the disease is often latent, so that the patient may die quite unexpectedly, without febrile or cerebral symptoms.

The state of the urine throws but little light upon the *diagnosis* of suppurative nephritis. There may be a large quantity of pus in it, but this is probably the result of the pyelitis and of the cystitis which are so generally present at the same time.

It might be thought that the determination of the amount of urea in the urine would probably throw light upon the state of the kidneys in these

doubtful and obscure cases. This expectation, however, appears not to be verified by experience. Dr Goodhart ('Guy's Hosp. Rep.,' xix) records two instances in which he made quantitative analyses shortly before death, and found that the renal secretion contained thirteen or fourteen grains of urea to the ounce; a third patient passed in the twenty-four hours three pints of urine, with a total quantity of 592 grains of urea; and a fourth patient thirty ounces with 328 grains of urea. On the other hand, a man, who afterwards went out well, having had his bladder punctured *per rectum*, passed thirty ounces in the twenty-four hours with only 295 grains of urea, or less than ten grains per ounce.

Treatment of renal calculus and pyelitis.—In all cases in which there is reason to hope that renal calculi consist of uric acid, or of urates, a fair trial should be given to the *solvent method* elaborated by Sir Wm. Roberts.* This observer made a careful series of experiments with calculi, outside the human body, exposing them to the action of a slow stream of a solution of carbonate of potass, which proved to be more effective than the carbonate of soda. With a liquid containing from forty to sixty grains of the alkali to the pint, he found that stones lost from 15 to 20 per cent. of their weight in twenty-four hours. Even with liquids containing twenty or thirty grains to the pint the solvent action was considerable. But what is very remarkable is that above the strength of sixty grains it ceased, in consequence of the formation of a tenacious white crust of alkaline biurate upon the surface of the concretion. The next step was to ascertain what doses of the vegetable salts of potass would give to the urine an alkalinity equivalent to about fifty grains of carbonate in the pint; and it was found that this could be effected in adults by the administration of forty to sixty grains of the acetate or citrate, dissolved in three or four ounces of water, every three hours; in children, by about half the quantity. Some patients find that the acetate agrees with them better than the citrate, in others the reverse is the case. As the citrate of potass of the shops is apt to be impure, Roberts advises that it should be prepared by neutralising a solution of the bicarbonate with crystallised citric acid; the following formula yields sixty grains of the citrate to the ounce:—℞ Potas. Bicarb. ʒxij; Acid. Citric. ʒviij, gr. xxiv; Aq. ad ℥xij.

It is not to be supposed that the urine passed by patients taking such doses of the potass salts can be maintained at an absolutely constant degree of alkalinity. On the contrary, it varies from hour to hour, but generally within the limits which correspond with the highest solvent action upon calculi; and Roberts has found experimentally that such urine, when it is allowed to pass over a uric acid stone outside the body at blood-heat, dissolves it at the mean rate of twelve and a half grains in the twenty-four hours. Clinically, it is obvious that the best proof of the power of urine, when rendered alkaline in this manner, to act upon calculi within the body, is to be obtained in the case of vesical calculi, of which the presence and the approximate size can be determined by sounding before treatment is begun, and which can, if necessary, be removed by lithotomy afterwards. In one case Roberts, after thirty-nine days' treatment, obtained the clearest

* In the fourth volume of the 'Medico-Chirurgical Transactions' there is a report of the solvent treatment of calculi by the Rev. Stephen Hales and Mr David Hartley. The experiments, which were suggested by Cheselden, were carried on by Mr Sharpe at Guy's Hospital and by Mr Gardiner at St George's Hospital.

evidence that a solvent action had been exerted; at the end of that time the cutting operation was performed and the stone was found to be eroded to a considerable extent, so that an incomplete layer of oxalate of lime was exposed, part of which was actually undermined. The proof of the efficacy of such treatment in the case of renal calculi is necessarily less complete. But there is a strong presumption in its favour.

The following case occurred to the author. A man came with a number of little uric acid calculi which he had been passing frequently. A vegetable salt of potass was prescribed, and a fortnight later he brought a single concretion, the only one he had passed, coated over with a white layer, which looked like phosphates, but which may have consisted of the biurate of potash. There seems to be little doubt that many other concretions must have been dissolved, for all the renal symptoms which had been troubling him disappeared, and he voided no more calculi.

The great drawback to this solvent treatment is that it is unfortunately altogether ineffectual when a stone consists of oxalate of lime, and that it fails even in the case of mixed calculi as soon as a complete layer of the oxalate is reached. It has been supposed that by rendering the urine alkaline one runs a risk of bringing about a deposition of phosphates, and so of actually augmenting the size of a calculus. But Roberts has shown that so long as the alkalinity is due to fixed alkali there is no danger of this result; and as a matter of experience, he has found that after the treatment has been continuously carried out for three months, an oxalate of lime calculus in the bladder has remained entirely free from phosphatic incrustation. On the other hand, in the experiments already referred to, in which uric acid calculi were exposed outside the body to a slow stream of urine rendered alkaline by fixed alkali, and in which the calculi underwent solution, it was ascertained that as soon as ammoniacal decomposition of the urine occurred, a layer of mixed phosphates was deposited, and all further solvent action ceased. It is therefore useless to attempt Roberts's plan of treatment unless the urine is acid. But even when putrefaction of the urine within the urinary passages has begun, the administration of benzoate or salicylate of soda may sometimes succeed in arresting this change, and in restoring the natural acid state of the fluid, so as to bring the case again within the scope of solvent remedies.

Unfortunately, among adults of middle age, the relative frequency of oxalate of lime calculi compared with those of uric acid is very much greater than would appear from the statements usually made in books (see p. 658). Perhaps these statements are based upon museum specimens, or upon statistics of cases of vesical calculi that have undergone surgical treatment. At any rate, in patients complaining of renal symptoms, examples of oxalate of lime calculi are more numerous than of those of any other kind. When the presence of such a calculus is suspected, the only prospect of cure, apart from surgical operation, lies in the possibility that it may either pass down the ureter and be voided, or else become fixed in the renal pelvis or "encysted," so as to cause no further symptoms. The possibility of the latter occurrence was especially insisted on by Dr Rees in the Croonian Lectures for 1856.

Operative treatment.—In all cases of protracted and severe pyelitis the question of surgical interference has to be taken into serious consideration, and it should not be delayed too long; for the chances of recovery are much greater at an early period of the disease than when it is far advanced. If there is an abscess in the loin there can of course be no question as to the advisability

of thoroughly exploring it, and of searching for and removing any calculi that may be present; and even when there is no evidence of anything more than pyonephrosis it is almost always advisable to cut down upon the kidney in the loin, or to lay open the suppurating cavity, so as to allow of its thorough drainage. This operation is termed *nephrotomy*, and how successful it may sometimes be is well shown by a case related by Rosenberger, of Wurzburg ('Trans. Internat. Congress, 1881'). The patient, a medical man, had during the previous year had an incision made behind the anterior superior spine of the ilium, with discharge of several pints of offensive pus. He was reduced to a skeleton when the lumbar operation was performed. Yet, the cavity having been washed out with carbolic acid and a drainage-tube inserted, he gradually regained his healthy appearance and resumed his practice, a small sinus alone remaining. When, however, a large branched calculus is found occupying the renal pelvis, the attempt to extract it may be as dangerous a procedure as the complete removal of the diseased organ. A case of this kind, which occurred to Mr Morrant Baker ('Trans. Internat. Congress, 1881'), proved quickly fatal by shock and by hæmorrhage from the walls of the dilated renal pelvis.

Nephrectomy itself is a most formidable operation in such circumstances. When there is a large pyonephric sac, a lumbar incision may fail to give room for its extirpation—as has been pointed out by Mr Howard Marsh ('Trans. Clin. Soc.,' 1882). Another difficulty is illustrated by two cases of Mr Barker's ('Med.-Chir. Trans.,' lxiv); in each of them the kidney was found to be surrounded by a mass of dense vascular tissue, which could not be removed, and which in one instance matted together the structures at the hilus in such a way that they could not be isolated. Lastly, there is the difficulty of making sure that the opposite kidney is capable of maintaining by itself a due secretion of urine. Cases have already been recorded in which fatal suppression of urine has occurred; for the organ affected with calculous pyelitis, of such severity as to justify its extirpation, was nevertheless the only functionally active kidney which the patient possessed. To obviate this risk Czerny has proposed to make two stages of the operation, first producing a urinary fistula, and after an interval proceeding to nephrectomy. Another suggestion made by Simon is that in female patients, after dilating the urethra, it may be possible to catheterise each ureter separately; and Teichmann, who for several years carried on elaborate investigations in the *post-mortem* room at Guy's Hospital, believes that even in the male subject he can, with an instrument introduced along the urethra, nip up the mouth of each ureter in turn, and so withdraw from the bladder the secretion of each kidney separately.

Nephrolithotomy.—When no lumbar abscess or even pyelitis is present, cutting down upon a kidney with the object of removing a calculus—a procedure which was condemned by Sir Benjamin Brodie as dangerous and absurd—is now proved to be feasible. The first successful operation was by Mr Henry Morris at the Middlesex Hospital ('Clin. Trans.,' xiv) in 1880. The patient, a girl aged nineteen, was admitted under the care of Dr Coupland. She had for several years been liable to severe paroxysmal pain in the right lumbar region, which made her life as a domestic servant unendurable, and for at least two years her urine had often contained blood. Mr Morris cut down upon the kidney, and with his forefinger almost at once detected "something rounded, about the size of the uncut end of a pencil, causing a slight irregularity of the surface of the

organ at a spot just a little behind the hilus." With a bistoury he incised the kidney at this spot, and succeeded in removing a calculus, which weighed thirty-one grains and consisted of oxalate of lime. The patient made a good recovery. In vol. xv of the 'Clinical Society's Transactions' two similar cases are recorded, each of which was no less successful than that of Mr Morris. One, by Mr Marcus Beck, is that of a young man aged nineteen, who had suffered for twelve years from symptoms of stone in the left kidney, some pain in the loin increased by movement, hæmaturia, and great irritability of bladder. Mr Beck exposed the kidney, and on thrusting a darning-needle into the organ, a stone was at once felt. An incision was then made which was followed by an alarming jet of blood, but the stone was extracted after the incision had been enlarged. It consisted of alternating layers of uric acid and of phosphates. Rapid recovery took place, there being no escape of urine from the wound except for a brief period of four days. In the other case, Mr Butlin's, the symptoms were almost exclusively those of neuralgia of the right testicle, which had continued for ten or twelve years. There was, indeed, some pain in the loin, but the urine never contained either pus or blood, though crystals of oxalate of lime were almost always present, and often a trace of albumen. The kidney having been exposed, a hard body was felt with the finger, and removed; it proved to be an oxalate of lime calculus. The wound quickly healed, but for some weeks after the operation the wound contained pus, apparently as the result of pyelitis. In volume xvi of the 'Clinical Society's Transactions' there is a fourth case of successful nephrolithotomy (the stone weighing 473 grains) by Mr May, of Birmingham, and a fifth by Mr Howse (see also the same 'Transactions' for 1884, 1885 and 1887).

The chief difficulty is that of diagnosing with sufficient certainty that the stone is present, and that it is too large to pass down the ureter into the bladder. Mr Morris cites seven instances, in each of which an incision down to the kidney has been made without any stone being detected. See cases of this operation and of nephrectomy by Prof. Czerny, Mr Baker, Mr Lucas, Mr Barwell ('Internat. Cong., 1881,' pp. 242—279), and Thos. Jones, of Manchester ('Brit. Med. Journ.,' June 2nd, 1883), also Sir Spencer Wells, 'Abdominal Tumours,' p. 199, and Mr Henry Morris's monograph on the subject.

TUBERCLE, CANCER, PARASITES AND ABNORMALITIES OF THE KIDNEYS

TUBERCULAR PYELITIS.—*Tubercular nature—Symptoms, causes, and treatment.*
MALIGNANT DISEASE.—*Sarcoma—Carcinoma—Symptoms—Nephrectomy.*
Hydatids—Chyluria and Filaria sanguinis—Endemic hæmaturia and bilharzia—Renal malformations—Floating kidney.

THE present chapter completes the subject of renal diseases by dealing with degenerations and new growths, parasitic affections, and displacements of the kidney.

TUBERCULOUS DISEASE OF THE KIDNEY. NEPHRO-PHTHISIS.—In cases of acute tuberculosis, miliary tubercles are found after death in the kidneys, as in other organs, but they are generally few in number, so far as the naked eye can judge. Even when numerous they are not known to produce any symptoms, or to affect the course of the disease. Ebstein, however, says that the epithelial cells of the kidneys are generally in a state of advanced granular degeneration, a point of interest in connection with the occurrence of catarrhal changes in the lungs and in the testicles in acute tuberculosis. The urine is free from albumen, and there are no symptoms which point to the kidneys during life.

The affection now to be described is of a different nature. It consists in the gradual destruction of the kidney, generally of one side only, by the formation of cavities with cheesy walls, which may fairly be called *vomicæ*, from their resemblance to vomicæ in the lungs. Precisely the same differences of opinion have of late years prevailed about this "nephro-phthisis" as about the corresponding pulmonary disease. Some observers have refused to recognise it as tuberculous, regarding it as a mere "caseous inflammation," and declare that if tubercles are found they are secondary and accidental formations. But there was always strong evidence, from the close likeness which exists between this renal affection and those in the lungs and in many other organs, that they all possess specific characters; and now, since the discovery of the tubercle bacillus, the truth of this view has been established not only as concerns the so-called "caseous" and "catarrhal" forms of pulmonary phthisis, but also for the corresponding morbid conditions of the lymph-glands, the testes, and the kidney. Moreover, the tubercle bacillus has been repeatedly found in the urine.

Anatomy.—There have been some differences of opinion as to whether tuberculous disease of the kidney has its starting-point in the cortex or in the medulla of a pyramid. The following observations of the author's show that it may begin in either situation, and also that, as a rule, tubercles are present from the first, and that even when this is not obvious, the character of the morbid process is yet unmistakeable.

(1) In 1874 a woman aged twenty-six died in Guy's Hospital of

phthisis; one kidney had in its cortex a cluster of yellow tubercles, from which a linear yellow streak extended down to the corresponding pyramid. (2) In 1873 a girl aged six died of tubercular peritonitis; in one kidney, near the apex of a pyramid, was a round tubercle just beginning to soften; the mucous membrane of the pelvis of the other kidney was covered with tubercles, as was also the lining membrane of the bladder. (3) In the same year another girl aged four and a half years died of acute tuberculosis, the bronchial glands being caseous; in a single pyramid of one kidney not quite reaching either its free surface or its base, there was a well-marked vomica, with an indurated cheesy border. (4) In the same year a man aged forty died of phthisis, with caries of the spine; in one kidney there was early tuberculous disease, ulcerating so as to form a conical cavity; in the adjacent part of the cortex there were cheesy grains and nodules up to the size of swan-shot, some extending to the surface of the organ. (5) In 1863 a boy aged fifteen, who had been admitted for vesical symptoms, died of tubercular meningitis; both kidneys, but especially the right one, contained "softening tuberculous matter, as well as distinct tubercles, regularly arranged in the cortex;" in the mucous membrane of the pelvis of the right kidney there were well-marked isolated tubercles, and also in that of the corresponding ureter and of the bladder near its neck. (6) In the same year a man aged twenty-two died of acute general tuberculosis and tubercular meningitis; the kidneys were stuffed with soft yellow tubercles, in some places collected into groups, and apparently about to soften into abscesses; the pelvis of the right kidney was lined with a layer of granular lymph, and this extended down the ureter into the bladder, which itself was affected with tuberculous ulceration; all round the opening of the right ureter into the bladder its mucous membrane was covered with isolated tubercles of various sizes. (7) In 1876 a man aged thirty-four died of bronchitis and emphysema, the lungs being quite free from tuberculous lesions; in one kidney a single pyramid was eaten away at the tip, and the rest of it was changed into a gelatinous material of sulphur-yellow colour. (8) In 1875 a man aged twenty-three died of pleurisy and of tubercular disease of the lung; in the substance of a single pyramid of one kidney there was an early patch of caseous infiltration. (9) In 1878 a woman aged twenty-eight died of phthisis: one kidney contained a circumscribed cheesy mass of the size of a damson, and two of its pyramids were also affected with early tuberculous lesions. (10) In 1879 a youth aged nineteen died of spinal disease with psoas abscess; one kidney showed several early tubercular masses excavating the cortex and forming vomicæ with cheesy walls; on the mucous membrane of the pelvis there were also scattered grey tubercles and caseating patches. (11) In 1880 a woman aged twenty-five died of bronchitis; in one kidney there were two typical vomicæ with cheesy walls. (12) In the same year a man died of lardaceous disease of the viscera, the result of caries of the spine; in one kidney there were two vomicæ, one in the cortex, the other in a pyramid, with opaque caseating tubercles round them. (13) Another man, also in 1880, died of phthisis; in one kidney a pyramid was eroded by a single tuberculous ulcer, and beyond this, in the cortex, there were opaque white tubercles.

These cases, while they show that the characters of tuberculous disease of the kidney at its commencement vary within certain limits, yet appear also to prove that the morbid process is always fundamentally the same.

They also indicate clearly how close is the relationship between this affection and the tuberculous diseases of other organs, though it is to be observed that in two instances the cause of death was merely bronchitis. When the renal mischief has time to run its course, and to prove directly fatal, it is obviously far less likely to have been preceded hy tuberculous lesions elsewhere; but even in such cases they often develop themselves sooner or later, not only in other parts of the urogenital apparatus, but also in the most remote organs.

In a single case, inspected in 1874, an ulcer which excavated a single pyramid of one kidney had a hard calcareous wall, which seemed to indicate that the affection was arrested in its progress, and might have remained stationary had the patient lived. Otherwise, all the author's observations tend to confirm the usual opinion that when once tuberculous disease has begun, it goes on and destroys the whole substance of the organ. The vomicæ, which correspond more or less accurately with the affected pyramids and the portions of cortex belonging to them, keep increasing in size, their cheesy walls spreading further and further into the renal tissue, until they lie close beneath the capsule and touch one another on all sides, or communicate by lateral openings. At the same time the capsule undergoes great thickening and may become almost as hard as cartilage. The mucous membrane of the pelvis, from an early period, is converted into a thick whitish-yellow layer. Any parts of the cortex that escape removal by ulceration are converted into a tough, white, fibrous material that shows no trace of a tubular structure, as was well illustrated by a specimen which was shown by Mr Lucas to the Pathological Society in 1875. Almost always there is enough of this material to provide septa by which the excavated organ is permanently divided into a series of sacculi which may be more or less completely shut off from one another; and these may at length lose their caseous walls and become bounded by a smooth lining membrane. Their contents vary in consistency in different cases; sometimes they are full of a cheesy semifluid pulp, sometimes of a substance exactly like putty or mortar, and highly impregnated with lime salts; occasionally some of them ultimately come to contain a transparent yellowish fluid in which there may be seen floating crystals of cholesterine. It is obvious that such matters could hardly accumulate in large quantity were there a way freely open for their escape. But the fact is that from an early period in the course of the disease the *ureter* is commonly blocked and impervious. Its mucous membrane undergoes the same change as that which affects the renal pelvis; its other coats are indurated, and it becomes converted into a hard cord, which may be as thick as a pencil, or even as one's finger, and which has a very narrow lumen left in its centre.

The *bladder*, in its turn, shares in the morbid process. Sometimes an excavated ulcer forms round the orifice of the ureter. Sometimes the whole of the vesical cavity becomes lined with patches of cheesy material, and more or less extensively ulcerated. In some cases, however, the vesical affection begins at the same time with, or even before, that of the kidney.

Not infrequently the morbid process extends into the *urethra*. In three cases, it reached as far as the external meatus, where the rough greyish-yellow appearance of the mucous membrane might easily have been seen during the patient's life had attention been directed to it. In one of these cases the canal was extraordinarily widened, so much so that a No. 16 catheter was required to fill it. When the affection was advancing along

the urethra up to the time of death, the part most recently affected sometimes shows very obvious grey tubercles, apparently situated upon the surface of the mucous membrane.

In many cases the genital organs—the prostate, the vesiculæ seminales, the vasa deferentia, and the testicles, some or all—take part in the disease. For example, apart altogether from cases in which the patient complains of anything that the surgeon would ordinarily recognise as "strumous disease of the testicle," one or more hard nodules can often be felt in the epididymis if careful search is made for them. As a rule, the vas deferens retains its natural size when affected with tuberculous mischief, but in one case the whole spermatic cord appeared obviously indurated during life. In the prostate the general result of tuberculous disease is to cause moderate enlargement, with the formation of vomicæ having caseous walls; and the same may be said also of the vesiculæ seminales, except that the cavities which are seen in them are formed out of those that are naturally present. In female patients it is not unusual for the internal genitalia to become affected with tuberculous lesions as a complication of a like affection of the kidneys; but in only two of our cases were such lesions found in the Fallopian tubes.

Altogether, among some thirty-four cases of tuberculous disease of the kidney from the *post-mortem* reports at Guy's Hospital, in which the renal affection was so far advanced as to have a more or less important share in the result, hardly a single instance occurred in which the other parts of the urogenital apparatus were free from tubercular lesions. Of the kidneys themselves one alone is very often affected, while the other shows no trace of tuberculous lesions. Among the thirty-four cases this occurred in twenty-two, while in the other twelve the disease was bilateral, but always much older and more advanced on one side than the other. It has been stated that the right kidney is less liable to become tuberculous than the left; and in the thirty-four cases the relative frequency of the disease on the two sides is as three to four.

Symptoms.—These are remarkably few. There may be pain in the loins, occasionally paroxysmal in character; and in some few cases there is tenderness on pressure. The diseased organ very rarely forms a tumour that can be felt by manipulation of the abdomen. In Mr Lucas's case, already referred to, which occurred in a little girl of seven, a circumscribed tumour was detected in the right hypochondriac and lumbar regions; after death the kidney was found to be six inches in length, and eleven in circumference. In another case of ours a tumour is said to have been felt, but at the *post-mortem* examination the kidney only weighed eighteen ounces. In another case there was a swelling which for a time led to the suspicion that the disease was malignant; but this ultimately proved to be an abscess behind the kidney, without any considerable enlargement of the organ itself. As a rule, indeed, a tuberculous kidney is but little above the natural size.

The *urine* is sometimes normal, the reason probably being that the ureter is blocked, as already described. It is therefore possible for tuberculous disease to go on to complete destruction of a kidney without any discharge from it reaching the bladder. But in most cases the urine is seldom free from either pus or blood, or both. Hæmaturia is not constant; among eighteen fatal cases with notes of the symptoms during life, which occurred at Guy's Hospital, in only ten is blood said to have been at any

time observed in the urine; and in most of these cases the bladder was likewise affected with tuberculous disease, so that the exact source of the hæmorrhage was after all doubtful. The most striking case is one of a man who a year before his death was stated to have one day passed a pint of blood by the urethra, after straining his back in lifting a heavy weight.

Pyuria is a far more conspicuous and important symptom. The quantity of pus in the urine is often so considerable as to form a thick deposit. Under the microscope a large proportion of the pus-cells are often found to be undergoing disintegration. The sediment may also contain granular amorphous masses insoluble in acetic acid, and even shreds of connective tissue, the presence of which is very significant. The bacillus of tubercle is frequently to be recognised in the pus-cells when successfully stained. In most cases the urine retains its acid reaction throughout the whole course of the patient's illness, but sometimes—probably always, especially when the bladder is also affected—it becomes ammoniacal and fœtid. There is then severe dysuria. Other cases differ widely among themselves as regards the presence or absence of vesical symptoms; sometimes pain in micturition and strangury have been conspicuous features of a case during life, but on *post-mortem* examination the bladder has been found apparently healthy.

Pyrexia is generally present if there are other marked symptoms, and it may assume a hectic type. It is attended with loss of appetite and often with nausea and diarrhœa, and emaciation may set in and bring the disease to a fatal termination.

Course and event.—The duration of these cases from the time when the patient is first discovered to be ill is commonly from six months to two or three years. But it would be a great mistake to suppose that this fairly represents the course of the affection. On the contrary there are many instances in which its progress is so slow that the opposite kidney has time to become hypertrophied, and probably carries on its function with perfect efficiency. Ultimately this healthy kidney may in its turn suffer from the effects of pressure upon its pyramids, as the result of tuberculous disease of the bladder; and an ascending suppurative nephritis may set in and rapidly bring the case to an end; or the hypertrophied organ may become affected with Bright's disease, as described at p. 673; or the drain of pus from the tuberculous kidney or from other parts of the urogenital apparatus may lead to the development of lardaceous changes, both in the opposite kidney and in the viscera generally. Under such conditions the urine may be albuminous and may contain tube-casts, even though, from obstruction of the tuberculous ureter, no pus is for a time being discharged. In one case at Guy's Hospital the immediate cause of death was the extension of ulceration from the tubercular kidney into the peritoneal cavity. A few cases have ended fatally as the result of the formation of perinephric abscesses, which have pointed in the loin, or have burrowed down in the sheath of the psoas muscle, until they made their way into the hip-joint or appeared in Scarpa's triangle. Lastly, the disease often terminates by the supervention of phthisis, or tubercular peritonitis or meningitis, or general miliary tuberculosis.

Ætiology.—With regard to the causes of tubercular disease of the kidney, apart from those that are concerned in the production of tubercular affections in general, very little can be said. It is much more common in men than

in women, the proportion being about three to one. Among twenty-nine cases at Guy's Hospital, taking only those in which the renal disease was the principal cause of death, there were three in which death occurred between the ages of ten and twenty, twelve between twenty-one and thirty, eight between thirty-one and forty, five between forty-one and fifty, and one between fifty-one and sixty. Roberts has given somewhat different figures, the proportion of cases during the later periods of life being much larger.

Treatment.—There is probably not much to be done beyond placing the patient under favourable conditions and giving cod-liver oil and tincture of steel. At an early stage, if the diagnosis could be made with certainty before the bladder had become affected, it might probably be advisable to run the risk of performing nephrectomy. Mr Morrant Baker ('Trans. Internat. Congr., 1881,' vol. ii, p. 262) performed this operation in the case of a girl aged seven, with a result so far successful that five months afterwards the child had greatly improved in health, being able to play all day long, and going out of doors frequently for a walk. Her illness had begun with hæmaturia about twenty-two months before the kidney was excised. In vol. xv of the 'Clinical Society's Transactions,' Dr Goodhart and Mr Golding Bird have recorded a case of nephrectomy for tuberculous disease which proved fatal about four hours after the completion of the operation. The patient had been seriously ill for about eight weeks, but had complained of pain in the back for eighteen months. At the autopsy the ureter was found to be diseased in its whole length, and the mucous membrane of the bladder was thickened and opaque. In the prostate there was some cretaceous material. It is the liability of the disease to affect other parts of the urogenital apparatus that renders nephrectomy a doubtful expedient.

Sarcoma and Carcinoma of the Kidneys.—When a malignant growth, in whatever part of the body it may be situated, gives rise to numerous secondary nodules in distant organs by infection of the blood-current, it not infrequently happens that some of them are seated in the kidneys. But in such cases the renal affection is seldom of clinical significance, except sometimes by causing more or less hæmaturia, or (if the primary growth is melanotic) by giving to the urine a brown or black colour.

Primary malignant tumours of the kidney are decidedly rare. Statistics from various sources are cited by Ebstein (in 'Ziemssen's Handbuch') in proof of this fact, and they are fully borne out by the reports of *post-mortem* examinations at Guy's Hospital, where only fifteen cases occurred during a period of twenty-two years.

Anatomy.—Two distinct affections were formerly included under the name of "malignant disease," or "cancer" of the kidney. One of them occurs chiefly in infants and young children up to the age of eight or ten years, though we have had two cases in boys aged respectively eleven and seventeen. This, although Ebstein speaks of it as carcinoma, is, as a rule, sarcoma, like most of the malignant growths of other parts that are met with in children. It forms a smooth rounded mass, which sometimes reaches an enormous size, weighing ten, twenty, or even thirty pounds, so that at one autopsy it was said that instead of the tumour being removed from the child's body, the body was removed from the tumour. Such a growth is commonly very soft and elastic, and may even appear to fluctuate; it is therefore likely to be punctured by the surgeon, a procedure which is gene-

rally harmless, but which may be followed by a sharp attack of peritonitis. Sometimes, beside blood, a small piece of sarcomatous tissue is brought away in the orifice of the trocar. These sarcomata of the kidney are very vascular, and hæmorrhage often takes place into their substance, causing a sudden increase in their size.* They grow, too, with great rapidity, destroying life sometimes within a few weeks, and almost always in less than a year from the time when they are first discovered. They commonly affect one kidney only, but Dr Abercrombie showed to the Pathological Society (in 1880) three cases, occurring in young children, in each of which both kidneys were invaded at the hilus by sarcoma.

A sarcomatous tumour in the position of the kidney is not always seated in that organ. Some years ago Dr Dickinson brought before the Pathological Society (vol. xxi, p. 397) a specimen in which the growth occupied the lumbar glands, and merely pushed the kidney before it; and in a case of Dr Day's it affected the capsule of an otherwise healthy kidney.

The other form of primary malignant disease of the kidney is true *carcinoma*. It is seen chiefly in persons past middle age; in almost every instance that has occurred at Guy's Hospital within the last twenty years the patient has been more than forty-five years old. It is much more common in men than in women. It does not generally grow very large, perhaps not exceeding the size of a cocoa-nut; but it may sometimes occupy all one side of the abdomen, or even appear to fill the whole cavity, like a great ovarian tumour in a woman. In such cases, however, a good part of its bulk is commonly made up of hollow spaces containing a blood-stained fluid; or there may be a large accumulation of a similar fluid in the dilated renal pelvis, which has been shut off from the ureter.

Carcinomata of the kidney are often spoken of as scirrhous, but they seldom deserve that name. They appear to be usually of the glandiform type, and of only moderate firmness. In very rare instances they are found to have undergone colloid degeneration. In 1876 the author showed to the Pathological Society (vol. xxvii, p. 204) a "carcinoma lipomatosum" of the kidney—a growth which looked like adipose tissue, but which had extensively invaded the renal veins, as well as the substance of the organ, and which had a typical alveolar structure, the alveoli being filled with large cells loaded with oil-drops. The specimen had no clinical interest, for the patient died of another disease.

Sometimes carcinoma of the kidney causes enlargement of the whole organ, the distinction between cortex and pyramids being still traceable in the tumour; sometimes only certain parts are affected, rounded or irregular masses of growth being separated by tracts of healthy tissue. Following Waldeyer, most pathologists now believe that the renal epithelium is the starting-point of the growth.

Ætiology.—With regard to the causes of malignant disease of the kidney very little is known. In a few cases it has appeared to be the result of a blow or of a kick in the loin; but one may doubt whether it was not really present before, and whether the injury did more than bring on symptoms, such as hæmaturia, by which attention was first drawn to it. In the following two cases the disease appeared to be added to another affection of older date. One occurred in a man of forty-five, who was said

* For an account of the histology see Dr Paul's paper with figures in the 'Pathological Transactions,' vol. xxxvii.

to have been troubled by passing a gelatinous substance in his water for twenty or thirty years. The right kidney was found to have its calyces dilated into a number of chambers, and in the pelvis lay a large, irregularly branched calculus, "like a knotted branch of a tree." Growing from the upper part of the organ was a cancerous mass, which also extended upwards behind the liver, and penetrated through the diaphragm into the lung. The other case was that of a man aged sixty-six, who had an attack of hæmaturia twenty years previously, and who came under treatment for a recurrence of this symptom ten months before his death. The lower part of the kidney showed the ordinary appearances of hydronephrosis, with "sacculation" from distension of the calyces; but into many of the sacculi soft masses of carcinoma were projecting; and the upper part of the organ formed a solid tumour. Even apart from the *post-mortem* appearances it is almost inconceivable that the cancer should in this patient have dated back as far as the earliest attack of hæmaturia, although Ebstein speaks of a case of renal cancer which was believed to have lasted for eighteen years. The occurrence of a calculus in the kidney as a complication of malignant growths is mentioned as not very rare; it may probably be the primary source of irritation, as with cancer of the gall-bladder (pp. 504, 541); but sometimes perhaps a phosphatic stone may be of later development than the tumour, especially if pyelitis happen to be present.

The ordinary duration of cases of carcinoma of the kidney in adults is probably from six months to two years after the first appearance of symptoms.

The symptoms of a malignant growth in the kidney, whether sarcomatous or carcinomatous, are mainly three: the presence of an abdominal tumour, hæmaturia, and pain.

1. The *tumour* has the usual characters of tumours seated in the kidney. It occupies one side of the abdomen, having its centre opposite the lumbar region, between the lower ribs and the iliac crest. It often bulges into the loin, and one can move it slightly forwards by pressing the loin with one hand, while the other is placed over the front of the abdomen. It does not descend during inspiration, and the fingers can be inserted between it and the rib cartilages, showing that it is not seated in either the liver (if on the right side) or the spleen (if on the left). It is sometimes perfectly smooth and uniform, sometimes more or less uneven and lobulated. Overlying it in front there is commonly a part of the colon (the hepatic or the splenic flexure, according to the side affected) which either may be felt as a ridge, or may be traced by its tympanitic percussion-sound. The dulness obtained on percussion over the tumour is continued into that of the lumbar region behind, whereas a splenic, ovarian, or other tumour, not growing from the loins, would be bounded externally by the resonance of the ascending or the descending colon. Sir Spencer Wells has proposed, in doubtful cases, to inflate the rectum with air, so as to render the position of the bowel more conspicuous. In one case the author could feel several coils of small intestine in front of the growth beside the colon; they were freely moveable, and slipped away from the finger under manipulation. Roberts relates a remarkable instance in which not only was the stomach made out during life to lie in front of a cancerous left kidney, but the spleen could be distinctly felt as a separate mass in the iliac fossa, lying over the lower and inner part of the tumour. Mr Holmes recorded, in vol. xxiv of the 'Pathological Transactions,' a case in which a malignant

growth of the kidney pulsated, and was attended with a bruit, so that aneurysm was suspected. There is often considerable distension of the superficial abdominal veins, which probably may be due to compression of the inferior vena cava by the tumour, or by enlarged glands; but in many instances the growth fungates into the renal vein, and it may even protrude into the cava so as to narrow its calibre. A further result of such conditions is that the feet and legs become œdematous; and Rindfleisch speaks of embolism of the pulmonary artery as being sometimes caused by the detachment of portions of the cancerous thrombus. In a case that was observed at Guy's Hospital in 1871 the disease made its way into one of the veins of the colon, and thence into the portal vein and its branches within the liver; ascites was the result.

2. *Hæmaturia* is by no means a constant symptom. Ebstein found it absent in twenty-eight out of fifty-two cases collected by him. Very often it is the earliest indication that anything is amiss with the patient. Sometimes it is directly brought on by a blow or fall. It may recur again and again at irregular intervals, for a considerable time before any tumour can be detected. It is then apt to be set down to a renal calculus, but one distinction is that the hæmaturia is not generally attended with a marked increase or aggravation of pain. In many cases the bleeding comes from portions of the growth that protrude into the pelvis of the kidney. But occasionally its source is from a tumour within the cortex, and if such is the case, tube-casts containing blood-corpuscles may doubtless be found in the urine, as stated by Ebstein.

3. The *pain* produced by a malignant tumour of the kidney is very variable in degree; sometimes it is altogether absent. Its usual seat is in one lumbar or hypochondriac region, but it may radiate widely over the lower part of the chest to the front of the abdomen, or to the crista ilii, and even down the thigh. It may be either constant, dull and aching, or paroxysmal, sharp and cutting in character. Sometimes there is much tenderness to pressure. The pain is seldom, if ever, attended with retraction of the testicle, in which respect it differs from the pain due to calculus. If, however, clots of blood formed in the pelvis of the kidney should become impacted in the ureter, the pain may assume a different character and become exactly like that which accompanies an attack of renal colic.

Other symptoms that may be present in cases of malignant growth in the kidney are anorexia, nausea, vomiting, and constipation or diarrhœa. In children, however, it is said that there is sometimes a voracious appetite with great thirst. The patient usually becomes rapidly wasted, anæmic, and cachectic. The temperature remains normal or subnormal, and the pulse may be unduly slow. Death is usually attributable to exhaustion, and is sometimes preceded for a few days by stupor or insensibility. In a case recorded by Bright in the first volume of the 'Guy's Hospital Reports,' the tumour gave way into the abdominal cavity, causing a large extravasation of blood. Some years ago a woman was admitted into hospital for paraplegia, which had been coming on during two months, but she was said to have had hæmaturia four months before and to have been ill for a year; at the autopsy it was found that there was a primary cancer of the left kidney, and that the growth had extended into the spinal canal. A like case has been observed by Cornil. In a patient who died in Guy's Hospital in 1870 all the symptoms were cerebral, and the immediate cause of death was the presence of secondary tumours in the brain.

Diagnosis.—It can easily be understood from the foregoing description that it is often difficult or impossible to recognise the presence of malignant disease of the kidney. A man aged thirty died in Guy's Hospital many years ago of wasting and weakness and anæmia, whose case excited great interest from there being no discoverable local symptoms. At the autopsy it was found that the right kidney was the seat of a primary growth which had destroyed nearly its whole substance, but which did not reach its pelvis. When a tumour in the lumbar region is accompanied by cachexia and emaciation in an elderly patient it is probably malignant, and if hæmaturia is present the diagnosis becomes nearly certain. But there are no anatomical signs to distinguish between a renal growth and one starting in the lumbar glands, the adrenals, or the vertebræ.

Treatment.—It is obviously improbable that any large measure of success will ever be attained by nephrectomy in cases of sarcoma or carcinoma of the kidney. But some few cases have already turned out much more favourably than could have been anticipated. In 1877 Mr Jessop, of Leeds, removed an encephaloid tumour of the kidney from a boy two and a half years old; rapid recovery took place, but about eight months afterwards the disease returned, probably in the lumbar glands, and the case ended fatally a few weeks later. In 1878, Martin, of Berlin, extirpated a sarcoma of the kidney, weighing twenty-eight ounces, in the case of a woman aged fifty-three; she was up on the eighth day, went home on the seventeenth; and Czerny speaks of her as being still well two years afterwards. In 1879, Lossen, of Heidelberg, performed nephrectomy in a woman aged thirty-seven, for an "angio-sarcoma" of the right kidney which, being moveable, was mistaken for an ovarian tumour; she recovered in six weeks, and she continued to be in good health eighteen months later. In 1881, Czerny removed a large vascular sarcoma from a man aged fifty-three, who at the time of the operation was very cachectic, and suffered greatly from vomiting; two months later he left the hospital in complete health, with the proportion of red discs in his blood twice as great as it had been previously. Against these successes however, must be set many cases in which extirpation of malignant growths in the kidneys has either been attempted ineffectually, or has proved quickly fatal by shock or by peritonitis. (See Czerny's tabular statement in the 'Trans. Internat. Congress, 1881,' vol. ii, p. 249.)

Apart from surgical interference, the only treatment is to relieve the symptoms as they arise.

Hydatid of the Kidney.—According to Davaine the kidney comes next to the lungs in the order of frequency among the organs liable to be infested with the echinococcus in its encysted state. In the records of *post-mortem* examinations at Guy's Hospital there are, however, only three or four instnces: in one the parasite was the size of a plum; in another of an orange; in a third it formed a bulging elastic swelling extending from the left hypochondrium into the loin, and containing two pints of fluid.

Clinically, the diagnosis must generally be based upon the recognition of an abdominal tumour, having the characters above given of tumours of the kidney, more or less tense and rounded in form, painless and possibly yielding fluctuation or palpable *fremitus*. The diseases for which it is most likely to be mistaken are hydronephrosis, soft sarcoma of the kidney, and (in the female) cystic disease of the ovary, as in a case in which Spiegelberg per-

formed an operation which he intended for ovariotomy. A point which may sometimes aid in the diagnosis is the discovery of a second hydatid in the liver; this was the case, for example, in a patient who died at Guy's Hospital with a hydatid in the kidney holding two pints of fluid.

In many instances an echinococcus in the kidney probably remains for years—perhaps from childhood or middle age throughout the entire life of the host—without affecting the health. Or it may die, and dry up into a pultaceous or cheesy mass, which henceforth has no power of doing damage. But in the majority of cases (if one may judge from hospital records) it sooner or later ruptures into the pelvis of the kidney, after which the daughter-cysts and scolices pass down the ureter and are expelled with the urine. Such cases present the symptoms of renal colic (see p. 667).

The presence of cysts or hooklets in the urine is not in itself proof of the existence of a hydatid in the kidney. Nearly, if not quite as frequent a seat of the parasite is the pelvic pouch of the peritoneum (cf. p. 558) and in such cases it sometimes ulcerates directly through the posterior wall of the bladder. In two cases of pelvic hydatids there was a tumour in the hypogastric region of the abdomen, having exactly the shape and the other characters of a distended bladder. In cases of renal hydatid, the passage of daughter-cysts down the ureter is often the first indication that anything is amiss with the patient; and when the parent cyst is small, no tumour may be discoverable in the loin. Not infrequently the rupture of the cyst is directly produced by a blow or by a fall; or the symptoms may appear to be brought on by riding or by driving, as in cases of renal calculus. Sometimes there is only a single discharge of daughter-cysts in the urine, and the patient afterwards remains perfectly well; sometimes the same thing recurs again and again, at intervals of months or years, during a period of ten, twenty, or even thirty years; sometimes suppuration within the capsule of the cyst occurs, and blood and pus may be voided in the urine. Roberts, however, says that the ultimate prognosis is generally favourable. Of sixty-three cases which he collected only nineteen were known to have ended fatally; and in nine of these the cause of death was some disease not directly connected with the renal affection. In some instances there has been ulceration through the diaphragm, with escape of daughter-cysts into a bronchial tube and expectoration through the air-passages; the prognosis is then very unfavourable.

The proper *treatment* of hydatid of the kidney, when a tumour can be detected, is puncture with a tubular needle, fitted to an aspirator. In two cases Roberts found that the withdrawal of only a drachm or two of fluid sufficed to destroy the life of the parasite, and caused it to pass very gradually into obsolescence and absorption.

Chyluria.—A very remarkable condition of the urine, first described by Prout, is one in which it looks white and milky when passed, and soon afterwards sets more or less completely into a soft jelly, like *blanc-mange*, which may take the shape of the vessel that contains it. Sometimes it solidifies within the bladder, and the result may be that there is pain and difficulty in micturition from obstruction of the urethra. The coagulum after a little while liquefies again; a material, somewhat like cream, collects upon the surface, and there falls to the bottom a deposit which is generally of a pinkish colour, from the presence of a small quantity of blood.

Prout recognised that the characters of the affection were exactly such as might be due to the admixture of chyle with the renal secretion. This view is confirmed by microscopical examination, which shows that the cause of the opacity is a finely granular material, not large fat-globules as in milk; and also by the application of chemical tests, for by ether a large quantity of fat may be extracted, and the urine also contains albumen in considerable amount. This peculiar state of the urine is often far more marked a few hours after a full meal than when the patient has been fasting for some time.

Until very recently, however, the pathology of chyluria has been a complete mystery, and there is scarcely anything more curious in the history of medicine than the way in which it has step by step been elucidated, until we now seem to know nearly all about it.

Prout noticed that a large number of those affected were born, or had lived for many years, in hot climates. Next, in 1866 Wucherer, in Brazil, detected in chylous urine certain minute living organisms, evidently the embryos of a nematode worm. Six years later, in 1872, the late Dr T. R. Lewis, in India, discovered similar embryos in the blood; and the parent worm (*Filaria sanguinis hominis*) has since been discovered, with the complete history of its development, which has been already set forth at length in the chapter on Entozoa (*supra*, p. 459).

The probable method by which the immature ova form obstructions in the lymphatic vessels has been also described (p. 462). The result is rupture of some of the lymphatics and extravasation of their contents. If a lacteal vessel thus bursts into the peritoneum, chylous ascites is produced (p. 484).

It now becomes an important question to determine how it is that the filaria produces such affections as chyluria, lymph-scrotum, and elephantiasis. The only hypothesis that can be said to account for such results is one that has been formulated by Dr Manson, in a paper in the 'Pathological Transactions' for 1882. His idea is that so long as the discharge of embryos goes on after the manner above described, the parasite is perfectly innocuous to its host. But from some cause or other, it happens in certain cases that, instead of the larval filariæ enclosed in their sheaths, ova in a much earlier stage of development, with unstretched shells, are extruded from the maternal vagina. Dr Manson has twice obtained such ova from the lymphatics; and probably they have been found in the urine also. Now, according to Dr Manson, they measure $\frac{1}{750}''$ in breadth by $\frac{1}{500}''$ in length; according to Dr Cobbold, $\frac{1}{1650}''$ by $\frac{1}{1000}''$. In either case their transverse diameter is far greater than that of the embryos; and nothing is more likely than that they should fail to pass along channels which the embryos would find no difficulty in traversing. Dr Manson supposes, for instance, that when they are carried by the lymph-stream to a gland, they become impacted in the smaller lymph channels in the cortex.

If the obstruction affects the abdominal or pelvic lymphatics the result will be more or less complete stasis of lymph, not only in the neighbourhood of the spot where the parent worm is situated, but also in the whole of one or both of the lower limbs, or in the scrotum. In the former case the chronic œdema with hypertrophy which is produced is called *elephantiasis (Arabum)*, or Barbadoes leg; in the latter it is known as lymph-scrotum. Dr Manson remarks that the ova may be conveyed across from one groin to

the other, and this explains why in one-sided elephantiasis the inguinal glands on both sides are often enlarged.

Elephantiasis will be considered under diseases of the skin.

Lymph-scrotum consists in the formation of vesicles, which are in fact dilated lymphatics, and which discharge fluid either clear or milky in appearance, according as it is derived from a peripheral vessel, or from one which has already passed through a lymph-gland. The tissues of the scrotum are thickened, but feel soft and spongy. Sometimes similar vesicles form on the inner side of the thigh. In the course of years the flow of lymph commonly ceases; and the scrotum passes into a state of elephantiasis. But the elephantiasis may be developed in the first instance without the formation of vesicles or escape of fluid.

When *chyluria* occurs, there can be no doubt that distended lymph-vessels open upon the surface of some part of the urinary mucous membrane; but whether this usually takes place in the bladder, or in the ureter, or in the renal pelvis, has not yet been determined.

One peculiarity of the affection is that it is often intermittent, the urine from time to time losing its abnormal characters for days or weeks together. In a case recorded by Ackermann ('Deutsch. Klin.,' 1863) the patient always passed normal urine after he had been lying on his right side for a time.

It is to be noted that chyle, in a strict sense of the term, does not exist in the urine in every case in which that fluid is more or less opaline in appearance; if the clot which forms is almost translucent, the cause of it may be merely the presence of lymph that has passed through one or more glands on its way upwards to the thoracic duct. But if the clot is opaque, like blanc-mange, and if the state of the urine varies in relation to the patient's meals, there can be no doubt that the obstruction involves lacteals coming from the intestines. The autopsy upon Dr Stephen Mackenzie's patient ('Path. Trans.,' 1882, pl. xxii, p. 394) showed a large mass of dilated lymph-sinuses and glands, extending from the bifurcation of the aorta below to the diaphragm above, and occupying the whole of the space between the kidneys. The lower part of the thoracic duct was sinuous and pouched, varying in diameter from three eighths to half an inch. About three inches above the diaphragm it became impervious, and was lost in a quantity of tough, dense material, apparently of inflammatory origin. In this case the communication between the lymph and the urine was probably in the kidneys.

These interesting observations as to the relations between the filaria and the chyluria of hot climates leave undetermined the pathology of the disease when it occurs in persons who have always resided in Europe. Four well-authenticated instances of this are cited by Roberts. In every case there is no doubt a definite fistulous communication between the lymphatics or thoracic duct and the urinary passages, whether caused by the filaria or any other agent.*

It is obvious, from what has been stated with regard to the life-history of the filaria, that the *prevention* of the diseases due to this parasite is quite possible. Dr Manson suggests that wells and water-jars should be covered with a netting sufficiently fine to prevent the entrance of mosquitoes, but it must surely be a better plan to drink no water which has

* See a case of chylous ascites admirably worked out and recorded by Dr Whitla, of Belfast, in the 'British Medical Journal,' May 30th, 1885.

not been boiled or filtered. Care must also be taken to have all raw vegetables thoroughly washed with boiled or filtered water before eating them.

In the *treatment* of chyluria, when it is once established, very little can be hoped for from medicines. But perhaps it is worth while to remember that Bence Jones thought that by giving gallic acid to the amount of two drachms daily he was sometimes successful in restoring the urine to a normal state, at least for periods of several months at a time. When the loss of chyle is considerable, it sometimes causes emaciation and debility, as well as a craving appetite and urgent thirst. In such cases the patient must be impressed with the importance of rest, for exercise is found to aggravate the complaint.

The duration of the disease is often very long. Roberts cites two instances, in one of which it continued for twenty-eight years, and in the other for more than fifty years. If death occurs during its course the cause is generally some intercurrent malady, such as phthisis or Bright's disease.

PARASITIC HÆMATURIA.—As back as 1812 Chanotin recorded the prevalence of an endemic form of hæmaturia in Mauritius; and subsequent writers afterwards noticed the occurrence of a similar affection in other hot climates. But nothing was made out with regard to its nature, until in 1851 Bilharz, being engaged with Griesinger in investigating the diseases of Egypt, discovered in certain of the veins of the abdominal viscera a trematode worm, to which he assigned the name of *Distoma hæmatobium*. It was found that this parasite gave rise, in some cases, to more or less severe urinary symptoms, and Griesinger, in the 'Arch. d. Heilkunde' for 1854, suggested that it might probably be the cause of the endemic hæmaturia of other countries. Afterwards it was shown to be generically distinct from the flukes, and Dr Cobbold proposed for it the name of *Bilharzia hæmatobia*, which is now generally adopted. In 1863 Dr John Harley detected the ova of the same entozoon in large numbers in the urine of a man who had become affected with hæmaturia at the Cape of Good Hope; and he has since shown (in papers in the 'Med.-Chir. Transactions') that the complaint prevails, not only in the Cape Colony, but also along the coast of Natal.

The Bilharzia is a soft, milk-white creature, which differs in shape in the two sexes. The male, half an inch in length, is flattened; but the hinder part of its body acquires a cylindrical appearance from its edges being thinned and folded inwards, so as to overlap one another, forming a hollow channel, within which the female lies during congress. The female, three quarters of an inch long, is slender and filiform. The ova are about $\frac{1}{170}''$ in length, and have a sharp projecting beak-like spine, placed usually at one end, but sometimes laterally. According to Dr Zancarol, of Alexandria ('Path. Trans.,' xxxiii), ova with lateral spines are found only when the seat of the parasite is in the veins of the intestine, whereas when it occupies the veins of the urinary tract the spines are terminal; and this statement seems to correspond with previous observations. (See the drawings by Dr Cavafy, p. 410.) It often happens that empty eggshells are found in the interior of the human body, so that there can be no doubt that the ova may be hatched while in the tissues. But Dr Cobbold believed that the ova in urine never give exit to the embryos which are often to be seen

within in a fully-developed condition, and quite ready to escape. Such ova, however, when placed in water, become ruptured in a few minutes. The embryos are covered all over with cilia, and swim actively about.

The further steps in the life-history of the Bilharzia have not yet been ascertained; but the presumption is that the embryo finds in some fresh-water mollusc an "intermediate host," and there develops a Cercaria-form. Leuckart seems to think that the most probable way in which human beings become infected is by their swallowing encysted cercariæ in the substance of snails or minute slugs eaten accidentally with raw vegetables. Griesinger was of opinion that in Egypt the chief danger lay in the use of fish as food, though he also mentioned the drinking of Nile water as possibly affording the parasite a mode of entrance into the human body, in which case one must suppose that it is capable of developing directly from the free cercaria into the sexually mature condition. Dr Harley inclines to think that bathing in the rivers of Natal may bring the host into contact with the parasite, which may either make its way through the skin, or even pass straight into the bladder through the orifice of the urethra. Males are much more liable to suffer from the Bilharzia than females. The resulting hæmaturia commonly appears during boyhood, but not under five or six years old. It may, however, occur at any age, for one of Dr Harley's patients was a man of seventy-six.

Endemic hæmaturia appears to be mainly vesical in its origin. At the end of micturition, the urine having been quite clear, the patient voids a small quantity—perhaps a teaspoonful or less—of dark blood. Or instead of the blood there may be soft shreds or filaments of mucus, by which the urethra is sometimes blocked for a few minutes; in these shreds the ova of the Bilharzia are found in large numbers. Sometimes a little pain is experienced in the loins or in the perinæum, especially after active exercise, which also increases the amount of blood. The health, as a rule, remains good, though a feeling of lassitude is sometimes complained of, and a more or less marked degree of anæmia may be developed. Boys in Natal appear to take no notice of the complaint, and about the age of puberty it commonly ceases. Long after this, however, Dr Harley has found that ova are still present in the urine, although the patient may imagine that he is quite free from his disorder. And what is very remarkable is that during early adult life he often begins to pass small *calculi*, in the centre of which the remains of ova may be detected. Indeed, in Egypt the affection commonly leads to the formation of large stones, which have to be removed by surgical operation.

Our knowledge of the lesions produced in the viscera by the Bilharzia appears still to be based almost entirely upon the investigations made by Bilharz and Griesinger in Egypt; they found it present in no fewer than 117 out of 363 autopsies. However, it is fair to assume that the state of the bladder in persons who (like the boys of Natal) suffer comparatively little from this parasite, corresponds with only the slightest cases examined by the two German observers. They give a much more serious account of the ordinary symptoms of Bilharzia, which they describe as chronic cystitis. The earliest morbid change in the bladder was the formation of swollen, hyperæmic, ecchymosed patches, varying in size up to that of a shilling, and generally coated with tough mucus or with a layer of soft greyish-yellow exudation; they were often limited to the posterior wall of the organ. But in many cases there were also thick deposits of a buttery

or soft and granular material upon the surface of the mucous membrane; generally encrusted with urinary salts. Sometimes there were raised warty vegetations or fungous-like excrescences, due to a swollen infiltrated condition of the submucous tissue. Ova and empty shells of the Bilharzia were present in large numbers throughout the substance of the diseased tissues, and also in the mucous and other exudations upon the surface of the lining membrane of the bladder. Deeper down lay the parasites themselves, in smooth-walled spaces, which communicated with the veins and evidently were nothing else than altered blood-vessels. The alimentary canal of the worms was always full of blood-corpuscles, which no doubt served them for food.

How the ova effect their escape from the spaces in which the parent worms lie, and how they manage to pass through the substance of the mucous membrane, does not seem to have been made out. Is it possible that the sharp spine with which each of them is provided enables them to work their way gradually outwards, under the influence of the pressure to which they are subjected by the contraction of the muscular wall of the bladder? Cobbold supposed that the spine may act as a "holdfast," giving purchase to the embryo, and so aiding it in the violent efforts which it sometimes has to make before it can get out of its shell.

In a great many cases the ureters were affected as well as the bladder, and sometimes they suffered when it escaped; in exceptional instances the morbid process extended even to the pelvis of the kidney. The lesions in the ureter were identical with those in the bladder, but were far more serious in their effects, inasmuch as they obstructed its channel, leading to hydronephrosis or pyelitis, and at length to complete destruction of the renal cortex. The natural result of such an affection would be the death of the patient by gradual exhaustion, and this was often observed. But in most of the instances the direct cause of the fatal issue was either pneumonia or dysentery. In regard to the latter disease it was at first a question whether it might not also be dependent on the presence of the parasite. For, beside the veins of the urinary apparatus, the only other vessels in which the Bilharzia was found were the portal vein and its tributaries, including the radicles of the intestinal veins. Ova seemed, in fact, to be scattered through the coats of the bowel exactly as they were through those of the bladder; and the mucous membrane of the bowel showed similar patches of inflammation. But although it is not unlikely that symptoms identical with those of dysentery may sometimes be produced by the Bilharzia, Griesinger soon satisfied himself that the common endemic dysentery of Egypt could not be attributed to this cause. On the other hand, he was inclined to think that in some cases the morbid process set up by the Bilharzia in the urinary apparatus gave rise to acute and rapidly fatal "typhoid" symptoms. If this is the case they are due perhaps to septicæmia. That the eggs may be carried to distant parts of the blood-stream is shown by a case in which a few empty shells were found in the interior of the heart; and they may act as mechanical or septic-emboli.

In the *treatment* of endemic hæmaturia, Dr Harley thinks that he has obtained good results from daily injections into the bladder of from twenty to thirty grains of iodide of potassium dissolved in five ounces of warm water. Dr Guillemard, however, has recorded a case in which even a weaker solution than this set up acute cystitis, and Dr Cobbold had previously expressed a strong opinion that such procedures are more likely to

do harm than good. Dr Harley has also prescribed, with some apparent advantage, draughts each containing ℥xv of oil of male fern and the same quantity of oil of turpentine. When there appears a tendency to the formation of uric acid calculi it should of course be counteracted by the administration of alkalies or of salts of the vegetable acids.

Malformation and Fixed Malposition of the Kidneys.—In some persons the two kidneys have their lower ends united in front of the spine so as to form a body of the shape of a horseshoe. The chief clinical importance of this condition lies in the fact that if the abdomen is thin and flaccid it may easily lead to the supposition that a morbid growth is present in the lumbar glands, or that the aorta is affected with aneurysm. The occurrence of hydronephrosis or of pyonephrosis in a "horseshoe kidney" would also be very likely to be misinterpreted during life.

Occasionally one kidney—generally the left one—lies at a lower level in the abdomen than natural, over the sacro-iliac synchondrosis, or even within the pelvic cavity. The misplaced organ may be mistaken for a tumour, as in a case recorded by Mr Durham in the 'Guy's Hospital Reports' for 1860. Or, in the female, it may interfere with parturition.

Moveable and Floating Kidney.—In some persons, instead of the kidneys being fixed deeply in the loins, one or both of them may become loose, so as to move in various directions, downwards, forwards, or inwards, to be readily felt by the hand through the abdominal wall, and to be almost as readily pushed back for the time into the natural position. At the same time, the lumbar region may be felt to be flattened or even hollowed, and percussion may yield an abnormally tympanitic note there. This condition occurs much more often on the right side than on the left. Among ninety-one cases collected by Ebstein, there were sixty-five in which the right kidney was moveable, fourteen in which the left was moveable, and twelve in which both were moveable.* As a rule, the place in which the kidney is felt is in the iliac fossa, or somewhere between this and its natural seat. But it is said sometimes to "float" and to come into contact with the front wall of the abdomen, or it may be in front of the spine. One cannot conceive of a kidney as *floating* without its having peritoneum on both sides of it; and there appears to be evidence that it does actually make for itself such a covering by pushing forward the membrane that should naturally be in contact only with its anterior surface. In vol. xxvii of the 'Pathological Transactions' may be found a case observed by Dr Goodhart, in which the right kidney while lying on the spine and over the psoas muscle, had become completely turned over, so that its anterior surface looked backwards; both surfaces were covered by peritoneum. As a rule, however, a moveable kidney glides about behind the serous membrane, merely dragging this with it to a slight extent.

The *causes* of the affection appear to be complex. Sometimes, perhaps, the kidney is pushed downwards by an enlarged liver; in one case, in the *post-mortem* room of Guy's Hospital, the liver was cancerous. Cruveilhier thought that displacement of the right kidney was often an indirect result of tight lacing, through its altering the position and shape of the liver. But although a mobile kidney is much more often seen in the

* In Landau's 173 cases (many of which, however, were identical with Ebstein's) 152 were right, only 12 left, and 9 double.

female sex than in the male (the proportion being as eighty-two to fourteen) it is believed to occur chiefly in women of the labouring classes, who are not so likely to wear tight stays; and it seems often to be due mainly to a relaxed state of the various structures forming the walls of the abdomen in consequence of frequent child-bearing. Sometimes, perhaps, an injury plays a part in determining the displacement. Thus, Ebstein cites a case from von Dusch, in which a woman who had borne eleven children, and whose abdomen was very loose, fell downstairs and struck her right side; soon afterwards she felt a tumour in the right hypochondrium. Becquet has maintained that a principal cause of mobility of the kidney is congestion and swelling of the organ, recurring at the menstrual periods. The objection to this view is that under normal circumstances such congestion is not known to take place. Roberts, however, in his article in 'Reynolds' System,' alludes to two cases in which a displaced kidney seemed to become larger and more sensitive to the touch each time that the catamenia appeared.

Patients in whom the kidney is moveable are often between the ages of twenty-five and forty, but Stoffen has observed this condition in children not more than nine or even six years old. In 122 collected cases, 43 affected patients between thirty and forty, and 79 those between twenty and fifty (Landau). The same writer found in 97 cases 87 women and 10 men.

Symptoms are not always caused by mobility of the kidney. Walther, of Dresden, some years ago examined a number of persons to decide this point, and detected the affection in many cases in which there were no symptoms whatever. But in some persons it causes a sense of weight and pressure in the abdomen, a feeling of dragging, as though something were loose in its cavity, or more or less intense pain which may radiate in various directions, to the ribs, the shoulder, the epigastrium, or the external genitalia. Sometimes there is nausea or vomiting. Patients are liable to attacks of intense suffering, attended with faintness and collapse, during which the kidney becomes exceedingly tender. Active exercise, whether walking or riding, often brings on or aggravates the pain; some patients are prevented by it from standing upright, or from turning in bed, or lying on one side. There is often an apparently disproportionate degree of anxiety and of depression of spirits, amounting to hypochondriasis.

The *diagnosis* of a moveable kidney is sometimes easy, sometimes difficult and uncertain. The affection may have to be distinguished in different cases from fæcal accumulation, from an enlarged spleen, from a distended gall-bladder, from a mass of swollen glands, or from an ovarian cyst with a long pedicle. Ebstein mentions an instance in which a hydatid cyst in the mesentery was mistaken for it; and in a case which the author saw, there were several such cysts, some of them oval in shape, and of almost exactly the size of the kidney.

The *treatment* of this affection generally resolves itself into keeping the patient in bed when there is severe pain, and applying fomentations and poultices. Afterwards an elastic abdominal belt should be applied, having a concave pad so placed as to maintain the kidney in its proper position. It is often, however, very difficult so to adjust an apparatus as to effect this object, and not to do more harm than good.

A suggestion made by Czerny is to inject alcohol into the connective tissue around the organ, while the patient is maintained in a recumbent position.

Hahn proposed to cut down upon the kidney by a lumbar incision, and to fix it in its proper place by sutures. Several cases are recorded by Ceccherelli ('Rivista Clinica,' April, 1884), quoted by Sir Spencer Wells.

When a moveable kidney is attended with unbearable suffering, it is doubtless justifiable to extirpate the organ. According to Czerny ('Trans. Internat. Congress, 1881') this had then been done in twelve cases. Four of them ended fatally, three by peritonitis, one in consequence of the opposite kidney being diseased; the others were completely successful, the patients not only recovering from the operation, but being also cured of the symptoms on account of which it was undertaken. No fewer than seven of these twelve cases of nephrectomy occurred in the practice of a single surgeon, Dr Martin, of Berlin.*

* Besides the chapters in the systematic works of Roger, Ebstein, and Trousseau, the following monographs on the subject are important:—Rollett's 'Pathologie und Therapie der beweglichen Niere' (Erlangen, 1866), and Landau's 'Wanderniere der Frauen' (Berlin, 1881), translated for the New Sydenham Society by Dr Champneys. Valuable papers on the subject have been published in England by Dr Hare ('Med. Times and Gazette,' 1858), Mr Durham ('Guy's Hosp. Rep.,' 1860), by a Committee of the Pathological Society (vol. xxvii), and by Dr Newman ('Glasgow Medical Journal,' August, 1883), with a useful bibliography.

DIABETES

Definition—Detection of glycose in the urine: the cupric, potash, fermentation, and other tests—Quantitative estimate by volumetric method—The amount, specific gravity, and other characters of the urine—Symptoms of diabetes—Complications—Modes of death—Physiology of the formation of sugar in the body—Glycosuria without diabetes—Theories of diabetes:—(1) *escape of sugar—*(2) *over-formation of sugar—*(3) *diminished destruction of sugar—exalted glycogenetic function of the liver—Experimental glycosuria—General ætiology of diabetes—Prognosis—Treatment: by diet—by drugs.*

THIS remarkable disease was once regarded as an affection of the kidneys. There are still great differences of opinion as to many points in the pathology of diabetes.* We now know that its origin is not in the kidneys nor in the urine nor even in the blood, but in the process of elaboration of the nutritious material absorbed from the food which takes place chiefly in the liver; and the physiological basis from which our discussions must start is the glycogenic function of the liver.

It will be convenient, however, before entering upon the theory of diabetes, to indicate its symptoms and the mode of detecting it.

The most common clinical history of the disease is somewhat as follows: A man finds that his strength is failing him, he knows not why. He is the more surprised at this because his appetite is excellent, and, indeed, larger than ever; yet he loses flesh as well as muscular power. Then he notices that he passes an unusually large quantity of water, and that he is always thirsty. He goes to a medical man, who makes a chemical examination of the urine, and finds that it contains a considerable quantity of a substance, *sugar*, which is absent from healthy urine, or present only in an exceedingly minute proportion. This justifies the diagnosis that the patient is suffering from Diabetes—Saccharine Diabetes or Diabetes Mellitus, as it is still sometimes called.

Tests for glycosuria.—There are several chemical processes by which it is possible to detect the presence of sugar in the urine, but only a few of them are used in practice.

(1) The chief among them is the *copper test*, which may be applied in several ways; but they are all based upon the fact that grape sugar (dextrose or glycose—the form of sugar that is contained in the urine in diabetes) possesses the property of reducing the oxide of copper to a suboxide at the temperature of 212°. The oxide of copper is blue, and liquids containing it in solution have a deep blue colour. On the other hand, the suboxide is orange yellow, so that there is no difficulty in seeing whether reduction takes place or not.

* The term diabetes is now restricted to cases of polyuria (διαβήτης) combined with glycosuria, *i. e.* the presence of glycose in the urine—Diabetes mellitus, as it was named by Willis, D. anglicus (cf. p. 561, note).—*Fr.* La diabète sucrée.—*Ger.* Zuckerharnruhr.

Trommer's method consisted in adding a few drops of solution of cupric sulphate to the urine and then an excess of liquor potassæ. The precipitate of the protoxide first thrown down is redissolved if sugar is present, and forms a deep blue clear solution, from which on heating the red hydrated suboxide of copper is precipitated. Liquor sodæ may be used instead of liquor potassæ.

Barreswil and Fehling introduced the method of keeping the copper in solution in excess of potash by means of tartrate of potash or potassio-tartrate of soda (soda tartarata) so as to have the test liquid ready for use.

The following solution is Dr Pavy's modification of Fehling's liquid :— 640 grains of neural tartrate of potass and 1280 grains of caustic potass are dissolved in ten fluid ounces of distilled water, and 320 grains of sulphate of copper are dissolved in other fluid ten ounces ; the solution of sulphate of copper is then poured into that of the potass salt, and forms a clear liquid of a deep blue colour.

The way to use this test is as follows :—About a drachm of the liquid is placed in a test-tube, and heated until it begins to boil. A drop or two of the urine is then added, and if no change is observed, a further quantity of urine, until this equals that of the copper solution. The test-tube is then again heated until the liquid in it reaches the boiling point. After this it is allowed to cool, there being no advantage, but rather the contrary, in continuing ebullition. If the urine contain any sugar, the liquid, before it cools, will be found to have deposited a yellow or red sediment of the sub-oxide of copper.

There are several points which require comment in the rules for the application of the copper test.

In the first place, one reason for applying heat to the copper solution first, and not to the urine, is that when the solution has been kept for some time exposed to light it is sometimes found to undergo a slight reduction when boiled by itself. According to Dr Pavy, the addition of a fragment of caustic potass to the liquid, when it has become deteriorated by keeping, will render it again as fit for use as ever. Another reason for heating the liquid before adding the urine is that healthy urine, if it happens to contain a large proportion of solids, possesses when boiling the property of decolourising the copper solution. The mixed liquids have then an amber yellow colour, and may often contain flocculi of phosphates—precipitated by the alkali of the test. This change is often regarded as proof of the presence of sugar ; but there could be no greater mistake. Indeed, Sir William Roberts says that when the blue colour of the copper solution has been discharged by the other ingredients of the urine it is no longer possible for any sugar that may be present to precipitate the suboxide. This peculiar change would doubtless commonly render misleading the careless application of the test to diabetic urine, were it not for the fact that such urine generally contains too small a proportion of the urinary solids for them to be able to decolourise the copper solution.

The reasons for adding urine in the exact way prescribed are thus given by Roberts, who has devoted much pains to the elaboration of the details of the process. In the production of the deposit of the suboxide it is necessary that the sugar should not be in excess. Unaltered sugar has the property of dissolving the suboxide of copper. Hence, if urine containing a large proportion of sugar be added to the copper solution in considerable quantity, no precipitate will result, but only an opaque yellow solution.

But if only a drop or two of the saccharine urine is added, a deposit is produced, which is then of a characteristic orange-red colour.

On the other hand, when the urine contains only a small proportion of sugar, it must be added in larger quantity. Then, as soon as the boiling point is reached the liquid changes to an intense opaque yellowish green, and a bright yellow deposit is slowly formed. Roberts has determined the exact limits of the application of this test. He finds that one tenth of a grain per fluid ounce can with certainty be detected by it. According to Dr Pavy, the yellow precipitate sometimes fails to be produced when there is albumen in the urine; the latter must then be boiled and filtered before it is added to the copper solution.

It is well known that many other substances beside sugar are capable of reducing the oxide of copper; but few of these are present in the urine.

Chloroform is one of them, and hence reduction of the copper solution in the case of a patient who has recently undergone an operation under chloroform is no proof of glycosuria; it is, however, rapidly eliminated by the lungs as well as the kidneys. The same effect is produced in the urine of patients who are taking chloral hydrate.* Leucin acts in the same way, but it never occurs in the urine except in cases of acute yellow atrophy of the liver.

Of drugs, the only one which causes a reduction of Fehling's solution is salicylic acid and its compounds. This effect is not constant, but frequent enough to be worth remembering. The reducing agent is probably salicyluric acid, in which form salicyl compounds are excreted by the kidneys.

Uric acid alone is likely to occasion a fallacy in the detection of diabetes. It is true that urine containing an excess of uric acid does not generally give rise to a change in the cupric solution at all resembling that which would be produced by sugar; but in certain cases it occasions a slight deposit of the suboxide.

Dr Pavy has since devised an ammoniated cupric solution which we have found very satisfactory at Guy's Hospital. The precipitated suboxide is kept in solution by ammonia and the test becomes one of colour only.

(2) Although the copper test for sugar fulfils all practical requirements, in accuracy and delicacy, there is an advantage in having another method of detecting diabetes upon which to fall back for corroboration in a doubtful case. Such is that known as Moore's test. It consists in boiling one or two drachms of the urine in a test-tube with half its bulk of liquor potassæ. The ebullition must be kept up for some little time; and as it goes on, the liquid become darker, passing through a series of colours which are almost exactly like those of different kinds of sherry wine, until it at last becomes brown. This test is not a very delicate one; it does not succeed with urine containing a quantity of sugar less than a grain and a half or two grains in the ounce. Moreover, urine containing albumen, and generally all high-coloured urines, become somewhat darker when boiled with liquor potassæ, even though they contain no sugar; and if the potash solution has lead in it, as is often the case, albuminous urine sometimes gives with it a dark porter-brown colour, which has been mistaken for that which sugar produces. Moore's test is therefore chiefly valuable as a preliminary test. But when it yields a negative result, this may generally be depended upon.

(3) Another test for sugar, which was first applied by Dobson, of Liverpool (1779), is the *fermentation test.* A small quantity of yeast, which must

* Dr Sherwin ('Boston Medical Journal,' November, 1886), quoted by Johnson.

be first thoroughly washed, so as to remove any adhering starch or sugar, is added to the urine, and this is set aside in a warm place, with a control glass. When sugar is present, it is presently decomposed into alcohol and carbonic acid. The latter, if found in any quantity, is given off as a gas, and may be readily collected. For this purpose all that is needed is that the urine should be made to fill a test-tube, and that this should be then inverted in a saucer, and kept in position by a clamp. After some hours it will be found that the liquid has receded from the upper or closed end of the tube, in consequence of the accumulation of the carbonic acid gas.

(4) A new test has been introduced by Dr George Johnson. It consists in the addition of liquor potassæ and a solution of picric acid to the urine. Heat is then applied, and when the boiling point is reached, the picric acid is turned into the red picramic acid. Dr Johnson recommends half the quantity of solution of potash to be added to the urine (say a fluid drachm); forty minims of saturated solution of picric acid to be added to this, and the mixture to be made up to half a fluid ounce before boiling. The addition of potash to picric acid deepens the tint to an orange colour, but when heated together it becomes of a much deeper claret-red. The reaction is produced by creatinin as well as glycose; but its chief practical drawback is that the change of colour is not so unmistakeable as in the case of Moore's test, or of the cupric test in any of its modifications.*

Quantitative analysis.—By the employment of two of these tests, or even of one of them, it is easy to determine whether a patient is or is not suffering from glycosuria; but this is by no means sufficient. Both for prognosis and treatment it is necessary that an estimate should be obtained, not only of the proportionate amount of sugar which the urine contains, but also of the total amount of sugar which is excreted by the kidneys in the twenty-four hours. With this object in view, all the urine which the patient passes must be carefully collected and measured every day, for some days in succession; for the proportionate amount of sugar contained in the urine is by no means uniform at different periods of the twenty-four hours. It is therefore from the mixed urine collected in a single vessel that a sample must be taken for analysis.

There are two or three different methods by which the amount of sugar present in a certain quantity of urine can be accurately determined.

(1) One consists in ascertaining how many minims of urine are required to reduce the whole of the oxide of copper in 100 minims of Pavy's copper solution. The solution is first measured by a pipette into a porcelain capsule. Into it is then dropped a fragment of caustic potass, of about twice the size of a pea, this having the effect of causing the reduced oxide afterwards to fall in a dense form, so that the colour of the remaining liquid can be more readily observed. The capsule is next heated by a spirit lamp until it boils steadily; a pipette graduated to hold 100 minims, with subdivisions, is in the meanwhile charged with the urine, and this is now allowed to flow drop by drop into the boiling copper solution, which is kept constantly stirred with a glass rod. If sugar be

* Of other tests for glycose, that which depends on the reduction of bismuth by carbonate of soda and heat (Böttger's test) was formerly much used in Germany. The determination of the presence and the amount of grape sugar by the polariscope is scientifically interesting and exact; it depends on the well-known property which glycose possesses of rotating the polarised ray of light to the right, whence its name *dextrose*. The earliest chemical test was devised by Cruikshank, and published in Rollo's 'Treatise on Diabetes,' 1798. It consisted in converting the dextrose into oxalic acid by the action of nitric acid.

present, the yellow or red oxide of copper gradually appears in greater quantity, but as soon as it is formed it settles, leaving the liquid still blue. At length, however, the blue colour is entirely removed, being replaced by an orange or an orange-red. At the moment when this occurs the operation is suspended, and a glance at the pipette shows how much urine has been used. The copper solution is of such a strength that exactly half a grain of sugar is required to discolourise 100 minims of it. Thus, there is half a grain of sugar in the quantity of urine that has been dropped from the pipette. It is a matter of the simplest calculation to determine the amount of sugar that must be contained in each ounce of urine or in the whole amount excreted daily. In his book on diabetes Dr Pavy gives a table by which the trouble of making this calculation may be saved. The process takes a very short time, and after a few trials anyone can learn to do it with sufficient accuracy. If the urine be highly charged with sugar, it is advisable to dilute it with from two to four parts of water before employing it for analysis, of course making the necessary correction afterwards. The ammoniated solution above mentioned may also be used with great ease and accuracy for volumetric analysis ('Lancet,' March 4th, 1884).

(2) Another plan is to ferment the urine with a little yeast, and next day to take its specific gravity and to compare it with that of the same urine in its unfermented state. For each grain of sugar per fluid ounce, one degree of density is lost by the process of fermentation. Roberts says that this method yields very fairly accurate results, and its performance requires no technical skill; the only objection to it is the delay, the result being obtained only after the lapse of twenty-four hours.

(3) The picric acid test may also be used for a quantitative purpose by comparing the tint obtained with a standard solution made of a definite colour by mixing liquor ferri perchloridi with acetic acid and ammonia; and Dr Johnson has found it convenient as well as trustworthy in practice.

The amount of sugar contained in the urine in diabetes varies from the smallest trace up to forty-eight grains in the ounce. Dr Pavy believes that this proportion is never exceeded, and that when it has been reached, any further increase in the quantity of sugar requiring to be excreted by the kidneys leads at once to an augmented flow of urine. The total quantity of sugar excreted daily shows of course corresponding variations.

Quantity of urine.—A diabetic patient, instead of two or three pints of urine in the twenty-four hours, or less, often passes as much as fifteen pints; and according to Sir Thomas Watson, cases are recorded in which seventy pints have been passed. Dr Pavy has himself seen a case in which thirty-two pints were collected and measured in one day.

Specific gravity.—It will be readily understood that in diabetes the specific gravity of the urine is higher than normal. Instead of being between 1015 and 1025, it is from 1030 to 1040 or 1045. Some writers have said that it may reach 1060 or 1070. But according to Dr Pavy, the maximum is a little above 1050. From the specific gravity of the urine in a case of diabetes one can form a rough estimate of the proportionate quantity of sugar contained in it; but the relations between them are by no means absolutely constant.

Some other characters of the urine in diabetes remain to be mentioned. It is generally pale, and the more so the greater the quantity that is passed. When this is very large, the urine may look just like water. Again, it is almost always clear, and deposits no urates after it has cooled. This fact

has a practical value, because it sometimes enables us to form an opinion as to when the disease began. Dr Prout used to ask his patients how long a time had passed since the urine became thick on cooling, and if such turbidity of the urine had previously been frequently observed he would date the commencement of the diabetes from the time when it ceased to occur.

Urine containing sugar has a sweet taste, a fact which was first discovered by Willis about the year 1674, when chemical methods of detecting sugar were not known. It is said that flies and wasps are attracted to vessels containing diabetic urine. The secretion has a peculiar odour, which was compared by Dr Prout to that of sweet hay or milk, by Sir Thomas Watson to the faint smell of certain apples, or of an apple chamber. The *Torula cerevisiæ*, or yeast plant, forms in diabetic urine when it is left freely exposed to the air in a warm place; and the sporules of this fungus may be readily detected with the microscope. Formerly its development was supposed to be a proof that a specimen of urine contained sugar, but even in healthy urine sporules may be found, which are, indeed, said to be *Penicillium glaucum*, but which are undistinguishable from those of the torula.

When a patient suffering from diabetes is attacked by any intercurrent febrile disease, the urine often, but not always, becomes for the time free from sugar. This is a point of considerable theoretical interest, as tending to confirm the view that the disease depends upon a perversion of the glycogenic function of the liver; for it has been shown that when fever arises in healthy subjects glycogen disappears from the liver.

Glycosuria without diabetes.—Physicians who make it a rule to examine the urine of every patient for sugar as well as for albumen not infrequently find traces of the former in otherwise normal urine, and in persons free from other symptoms of diabetes. Minute quantities of glycose occur normally in blood-serum, and from its crystalline character and diffusibility one would expect it to appear in minute quantities in normal urine. Some authorities deny its presence even in traces, but Brücke and Bence Jones long ago asserted its presence, and Pavy has brought positive evidence to the same effect ('Croonian Lectures,' p. 10). Hence "physiological glycosuria" has been supposed to exist, like physiological albuminuria. It has been asserted that "dietetic glycosuria" can be produced by eating too much sugar, and "dyspeptic glycosuria" has often been described, chiefly in gouty persons at or beyond middle life. This was taught by Sir Henry Marsh and by Graves, by Trousseau, who called a form of occasional glycosuria *glycosurie alternante chez les goutteux*, and by many living physicians, including Dr George Johnson.

But at present it is safest to regard the presence in the urine of sugar (as of albumen) in quantities appreciable by the cupric test without concentration or extraction as pathological; and excluding the fallacies produced by uric acid and otherwise which are mentioned above, the conclusion is probably also scientifically accurate.

If small quantities of sugar sometimes appear and vanish again under dietetic treatment, it is not unlikely that these are incipient cases of what would develop into the slighter forms of diabetes so often met with in persons who are past middle life.

However this may be, there are no other diseases than diabetes which produce glycosuria as one of their symptoms, in the way that albuminuria is pro

duced by cardiac lesions, fevers, and the other morbid conditions enumerated in the chapter on Bright's disease. The only partial exceptions to this are (1) temporary glycosuria from inhalation of chloroform or ether—apart from the presence of chloroform itself as a reducing agent in the urine, (2) from paroxysms of whooping-cough, asthma or epilepsy, (3) during pregnancy, (4) after injuries to the head. The last group is of great physiological interest, as we shall presently find (p. 725), but has little or no clinical bearing.

In another class of cases the ordinary symptoms of diabetes are absent, although instead of being transitory the presence of sugar in the urine is persistent. But there is no definite boundary-line between such cases and those in which the most marked symptoms are present; indeed, the same patient may come in turn under the one and under the other category. The same treatment is beneficial in both kinds of cases, and the same name should be given to both; in fact, persistent glycosuria is diabetes.

Symptoms.—The early signs of diabetes have already been mentioned (p. 708). Foremost among these must be placed *muscular weakness*. This is often extreme. It is by no means to be regarded as a mere result of the wasting of the muscles which generally accompanies the disease. In a series of experiments which Dr Pavy made with various kinds of diet on a man affected with diabetes, he found that as soon as the patient was put upon food which caused him to pass an excessive quantity of sugar, he complained that he had no life or energy in him. Another patient when admitted was so weak that he could not stand alone; after about three weeks, under treatment, he had gained strength so that he ran to the end of the ward and back to show what he could do. Loss of virility is another frequent effect of the disease, and in women suppression of the catamenia. Natural vigour of character may also be replaced by feebleness, moral and intellectual. The knee-jerk is sometimes absent, and neuralgia is not an infrequent complaint.

Thirst is another of the earliest and most persistent symptoms of diabetes. The patient generally drinks from eight to twelve pints a day, but sometimes as much as twenty-five or more. Yet even this does not satisfy the craving. The mouth and fauces are also the seat of a sensation of dryness, which causes great discomfort. Dr Pavy says that the way in which the patient keeps rolling the tongue about in the mouth, and the sound which it produces by sticking to the palate from time to time, may be recognised as signs of the disease. The tongue is often intensely red and unnaturally clean, and sometimes it is fissured. Occasionally a sensation is experienced of a sweet taste in the mouth.

Increased *appetite* for solid food is by no means so constant a symptom as thirst. Sometimes the appetite is enormous, but in the later stages of diabetes there may be a loathing of all kinds of food. The teeth generally become carious, and the gums swollen, loose, and inclined to bleed. Very often the patient complains of a peculiar and distressing sensation of hollowness at the pit of the stomach. Occasionally vomiting and symptoms of dyspepsia are present, but, as a rule, the digestive powers of persons suffering from diabetes are remarkably good: the stomach and liver are capable of dealing efficiently with the large amount of food taken.

Accordingly *urea* is formed in abundance. It was formerly supposed, indeed, that much less urea is contained in the urine of diabetic patients than in that of healthy persons. But it has been shown that this was a

mistake, and that the total amount of urea voided in the twenty-four hours is as great as, and often greater than, the normal quantity. We shall presently see that, at least in some cases of diabetes, a part of the sugar is formed from the nitrogenous elements of the food, or rather from the peptones into which they pass before they are absorbed from the alimentary canal; and it is probable that these peptones, under such circumstances, split up so as to produce two series of substances, of which the one has its final term in sugar and the other in urea. We should of course not expect that this proportion would show itself exactly in the urine, since a part of the urea excreted must be derived, not from food, but from the nitrogenous tissues, as in health. Dr Dickinson has made calculations, allowing a certain proportion for tissue-urea, and he found that in at least one patient although the actual daily quantities of urea and sugar varied greatly, yet the proportion of surplus urea to sugar was almost constantly as 1 to 6·1. On the other hand, Dr Ringer some years ago arrived at the conclusion that during abstinence, or under a non-nitrogenous diet, the *total* amount of urea and that of sugar excreted, whether by different patients or by the same patient at different times, had a constant ratio of about 1 to 2. But his results are difficult of acceptance, because they involve the supposition that even the disintegration of the nitrogenous tissues gives rise in diabetes to the formation of sugar as well as of urea.

A further question concerns the amount of *uric acid* which is excreted by diabetic patients. This also was supposed at one time to be much less than normal; but Dr Dickinson thinks that the deficiency is probably apparent rather than real. He has seen a copious deposit of crystals of lithic acid in urine of which more than fourteen pints were passed daily. Still, in comparison with urea, it appears to be clear that but little uric acid is found in diabetes.

In this connection one of Dr Ringer's results may be noted, namely, that in diabetes the ingestion of *non*-nitrogenous food was followed by a marked increase of urea, as well as of sugar, in the urine. For this may fairly be correlated with the fact pointed out by Murchison that lithæmia, a state in which nitrogenous principles fail to be fully oxidised, is especially apt to be induced by the ingestion of non-nitrogenous matters in excess, or in such quantities that the digestive organs become overloaded.

Nor are other facts wanting that appear to show an antagonism between lithæmia and diabetes. Sir Charles Scudamore long ago showed that whereas gout was less common in Scotland than in England, the relative liability of the two populations to diabetes was reversed. One of Dr Pavy's patients, who had before been a martyr to dyspepsia, said that his gastric troubles entirely ended as soon as diabetes appeared. It has also been noticed that gout has ceased to return in persons who have become diabetic; but this is less conclusive, because it might be merely from increased flow of urine preventing accumulation of uric acid in the blood.

These facts, indicating that lithæmia and diabetes are inversely correlated, are interesting not only in themselves but also because they afford further evidence of the correctness of the views which regard both these diseases as disorders of the hepatic functions.

The *bowels* are usually constipated in diabetes, the fæces being dry and hard. But diarrhœa sometimes occurs, and it may lead to a state of prostration which is the immediate cause of death.

Diabetic patients often complain of chilliness; the *temperature* of the body

is as a rule lowered, bearing some proportion to the severity of the disease. In one very severe case recorded by Dr Dickinson, it varied from 93·6° to 94·8°, and when fatal pneumonia set in, the thermometer only rose to 97·8°. In another case the same disease was attended with a temperature of 103·2°. But we shall presently see that in many cases the approach of death is preceded by a fall of temperature.

The *skin* is usually dry and harsh. The cuticle of the palms is stiff, and the furrows have a peculiar white mealy appearance. Generally speaking there is no sensible perspiration through the whole course of the disease, but sometimes profuse sweats occur. This is difficult of comprehension, and so, perhaps, is the fact that the subcutaneous tissue frequently becomes œdematous. This, however, is probably the result of anæmia. When it occurs, the presence of albumen in the urine should be sought for, as Bright's disease is a not infrequent complication of diabetes. But there is often œdema of the ankles, even when the kidneys are healthy.

One of the most marked symptoms of diabetes is *emaciation*. The features acquire a peculiar drawn, pinched look, by which the disease may often be recognised. Sometimes, however, the patient remains well nourished. Roberts says that one of his patients weighed more than fifteen stone, when he had been passing twelve pints of highly saccharine urine for some months; and that one of Prout's patients weighed twenty-three stone.

Another symptom of diabetes is that the patient's breath has a peculiar *sweet smell*, like that of the urine but less distinct. Dr Dickinson says that this is connected with a constipated state of the bowels, and he appears to take it as an indication of the near approach of the coma which is often the immediate cause of death. It can be detected by some people even at a little distance: it is said that the late Dr Guy Babington, when he came down to Guy's Hospital to take in patients on a Wednesday morning, could tell at once whether there was a case of diabetes among the applicants.

Complications.—In some diabetic patients the ordinary symptoms are absent, but it is rare for sugar to be discovered in the urine of persons who believe themselves to be perfectly well. Of this Bence Jones gave an instance. A gentleman noticed some little white bodies in his urine, and consequently had it tested. They proved to consist of epithelium from the bladder, but there was sugar in the urine, and this continued to be the case whenever it was examined afterwards. He was a stout man, and remained in good health. Another case, mentioned by Griesinger, is that of a medical student, whose urine was saccharine during the whole of one winter, while he was residing in a moist and foggy locality in Switzerland. He never had a single symptom of diabetes, and both before and afterwards the urine was often tested and found normal.

Instances such as these, however, appear to be of most exceptional occurrence. In the immense majority of cases in which sugar is found in the urine of an individual who does not present the ordinary signs of diabetes, he nevertheless is far from being well, and complains of more or less definite symptoms, which may indeed occur in patients in whom the urine is normal, but which nevertheless are so often accompanied by glycosuria that no cautious practitioner ever meets with them without ascertaining whether this is present or not. The affections in question are perhaps best described as "complications" rather than as ordinary symptoms of diabetes; but recognition of the saccharine condition of the urine is of the utmost

importance in all such cases, because it is very often the key to their successful treatment.

One of these affections is *pruritus vulvæ* in women, often attended with a lichenous or eczematous eruption. That such complaints are often due to diabetes is mentioned by most writers. A lady aged fifty-two consulted the author on account of an eruption affecting the vulva, and attended with severe pruritus. The parts were reddened, but dry; the disease in fact resembled a chronic case of lichen. Medicine failed; but next time her urine was found to contain ten grains of sugar to the ounce. In a few weeks, under proper treatment, her troublesome complaint was removed; and this although the urine still contained a small quantity of sugar. Most writers say that this pruriginous dermatitis is set up by the local irritation of the sugar in the urine; and they remark that the orifice of the male urethra and the glans penis are sometimes excoriated under the same circumstances. But pruritus of the vulva is comparatively rare in ordinary cases of diabetes.

Again, *carbuncles* and *boils* are apt to arise in patients whose urine is saccharine. Prout stated that in his experience diabetes always accompanied carbuncles and malignant boils or abscesses. But it has since been shown that this is too sweeping a statement. The importance of remembering the liability of diabetic patients to carbuncular affections is well shown by a case of Sir William Gull's, which is related by Dr Pavy. A medical man was suffering from cerebral symptoms, for which it was suggested that he should apply a blister to the nape of the neck. His urine contained sugar, and on this account he was cautioned against doing so. However, the blister was employed, and a large carbuncle soon developed itself, which proved fatal.

Prout also spoke of glycosuria as a temporary accompaniment of affections of this kind. A patient of his, middle-aged, told him that for a long period he had been subject, at intervals of a year or two, to boils and carbuncles, and that during such attacks he always passed a quantity of saccharine urine, whereas at other times the secretion was natural. Later writers also have given cases in which patients have had sugar in the urine only while they suffered from carbuncles or boils.

Gangrene of one of the lower limbs, resembling senile gangrene, is also frequently associated with a saccharine state of the urine. This is a fact which has been especially insisted upon by the surgeons of Dublin; and several cases of the kind have occurred in Guy's Hospital.

Defective accommodation is another symptom which is common in diabetic patients, due to impairment of the power of the ciliary muscle; in such cases Dr Pavy has found that the application of Calabar bean to the conjunctiva is very beneficial. Sometimes, however, sight is affected by the formation of *cataract*. Many years ago Mr France published several cases of this kind in the 'Ophthalmic' and the 'Guy's Hospital Reports,' 1859 and 1860.* Diabetic cataract has acquired special interest from experiments made by Dr Weir Mitchell, in which frogs were immersed in a saccharine solution, with the result that the crystalline lens became opaque. Other causes of loss of sight in diabetes, are atrophy of the optic discs, or retinitis, or opaque patches in the vitreous.

Phthisis.—The most important complication is a destructive disease of

* Lécorché's paper on the same subject appeared in the 'Archives générales de médecine,' in May, 1861, but cases had been noticed before by von Gräfe.

one or both lungs, which is very like the more acute forms of phthisis. This is often the immediate cause of the patient's death. The cases in which diabetes has led to a fatal termination in Guy's Hospital during the last twenty years (1860–80) were 40 in number, and in 17 the immediate cause of death was phthisis. Dr Addison taught, and Dr Wilks has since maintained the same opinion, that the pulmonary disease in cases of this kind is not tubercular phthisis, but rather a form of chronic pneumonia. Some writers have hesitated to adopt this opinion. The author therefore searched the records of Guy's Hospital to see how far they support Addison's view, and found that in twelve among the seventeen cases there was nothing that could fairly be identified as tubercle in the lungs, and in all of these it was either expressly stated that the larynx and intestines presented no tubercular ulceration, or, at least, no mention is made of these organs. On the other hand, it is said that in four cases the lungs contained grey or miliary tubercles; and in two of them, as well as in the other case, the intestines showed tuberculous ulcers. Now, referring the reader to a previous chapter for a discussion of the relation of "pneumonic" phthisis to the ordinary "tubercular" disease (pp. 201, 203), it is clear that such a proportion of cases without tubercles in the larynx and intestines is very different from what occurs in any form of phthisis, apart from diabetes; hence it supports the opinion that the pulmonary affection in this disease is not of tubercular origin. It might, indeed, be urged that the development of mischief in the lungs is so rapid that there is no time for the formation of tubercles in other parts of the body; for in several cases the earliest pulmonary symptoms appeared only from two to five months before the patient's death. But that this is not the only reason why the intestines and larynx escape, is evident from one case in which the symptoms of disease of the lungs preceded those of diabetes, and began fourteen months before death; for even in this case the intestines were free from tubercle.

The pulmonary affection generally spreads through the lungs from apex to base, like ordinary phthisis; it very rapidly leads to the formation of one or more large cavities, by which the whole of an upper lobe may be excavated, and which have usually very thin ragged walls. It is generally much more advanced in one lung than in the other, but sometimes attacks both organs pretty equally. A further proof of the rapidity with which it advances to a fatal termination is afforded by the fact that in only one of the twenty-eight cases in which death was not the direct result of this form of pulmonary disease did the lungs present any trace of it. If it were ordinary phthisis, it would surely be often found at an early stage in those cases which are fatal from other causes.

Another frequent cause of death in diabetes is ordinary lobar or "croupous" *pneumonia*. This was present in ten out of the forty cases collected by the author. In four of them, the hepatised parts were more or less distinctly passing into a gangrenous state. The onset of pulmonary symptoms in these cases was generally well marked, and occurred two or three days before death.*

Diabetic coma.—Of the remaining fatal cases there were 6 in which the immediate cause of death was the supervention of *cerebral symptoms*. These generally began with drowsiness, and in a few hours passed into coma. Once

* At the Editor's request, Mr Kelbe has analysed the anatomical reports of seventeen subsequent cases of diabetes which proved fatal in Guy's Hospital by inflammation of the lungs. The disease affected one or both apices in ten cases; with vomicæ but no sign of

or twice there was more or less well-marked delirium, or even convulsions; the pulse was often very feeble, and the temperature low. Indeed, in the cases of this kind the state of the patient is often one of collapse quite as much as of coma. In the 'Guy's Hospital Reports' for 1873–74 the author recorded one such instance, in which the pulse was scarcely perceptible, and the body and limbs were cold. In this case twenty-six ounces of a solution of phosphate of soda and chloride of sodium, of sp. gr. 1020, were injected into the right cephalic vein, with the effect of restoring the patient to consciousness for a time. He sat up, answered questions, took nourishment well, and even asked for it; his pulse was 80. Thirty-two hours later, however, he again became drowsy and died. This patient's condition, before the solution was injected into his veins, was strikingly like that of a man in the collapse of cholera: only a few drops of thick dark blood escaped from the wound in the arm. A few months afterwards Dr Frederick Taylor had a similar case, in which he employed the same treatment, but with scarcely any good result (*ibid.*, vol. xix, p. 521).

This "diabetic coma," has been ascribed to the presence of *acetone* in the blood. But acetonæmia is not always present in diabetes, nor is it always, when present, accompanied by cerebral symptoms.*

Dr Dreschfeld, in his Bradshaw Lecture before the College of Physicians ('Lancet,' August, 1886), states that during diabetic coma the following abnormal constituents have been discovered in the patient's urine:

(1) Acetone (C_3H_6O dimethyl-ketone);
(2) Aceto-acetic acid, which yields acetone and carbonic dioxide;
(3) Crotonic acid ($C_2H_6O_2$); and
(4) Oxybutyric acid ($C_4H_8O_3$).

The acetone which gives the odour to the breath, and which gives a bright crimson colour to the urine when treated with perchloride of iron, is the product of aceto-acetic acid, which may be extracted from the urine by ether after acidulation with dilute sulphuric acid.

Dr Dreschfeld recognises three types of diabetic coma:

1. Diabetic collapse, with coldness, lividity, and subnormal temperature. The pulse is rapid, and there is little dyspnœa. The patients are usually elderly, stout, and long diabetic. The attack comes on after fatigue (as in Prout's cases), and proves fatal rapidly—within twenty-four hours as a rule. The heart is often found fatty, but no acetone or other abnormal constituent but glycose is found in the urine.

2. A rare form, closely resembling drunkenness, with staggering gait, incoherent speech, and disturbed mental faculties. Acetone is often present in the urine and the breath, and sometimes alcohol.

3. The most frequent form, with muscular weakness, drowsiness, rapid breathing, and at last coma. There is the acetone smell in the breath and urine, and not only aceto-acetic but also crotonic and oxybutyric acids can be demonstrated in the urine.

As described by Prout and by Pavy, and afterwards by Kussmaul, the condition of coma (as distinct from collapse) agrees closely with the third of

tubercle in four, with miliary tubercles of the lungs or other organs in the other six. In two cases there was acute hepatisation of the base without any sign of phthisis. In the remaining five cases there was less acute pneumonic consolidation not affecting the apices, described as caseous in one case and necrotic in three, in one of which it was caused by the presence of particles of food.

* See an excellent account of Acetonæmia and Lipæmia in Diabetes, in Dr Gamgee's 'Physiological Chemistry,' vol. i, pp. 168—172.

these varieties. "Breathlessness without dyspnœa," occasional convulsions, and a subnormal temperature are its leading features. It occurs mostly in young patients, and early in the disease. It, like collapse, often follows fatigue.

In addition to the six cases mentioned, in which death was preceded by more or less marked cerebral symptoms, there were two in which it was quite sudden, and probably these should be placed in the same category with the others. There was this peculiarity with regard to a large proportion of these eight cases, namely, that the fatal symptoms developed themselves very shortly after the admission of the patient into the hospital. In five of them death took place within five days from the date of admission, and in three of them it occurred either on the day of admission itself or on the following day. The cause of the sudden fatal termination was, no doubt, the fatigue and excitement which the patients underwent in coming to the hospital. Exactly the same thing was noticed long ago by Prout, who says that four of his private patients sank almost immediately after coming to London from the country to consult him, and one of them was very near dying in Dr Prout's own house.

Dr Pavy has observed that those cases of diabetes in which the disease has been kept under control by treatment are particularly apt to end at last in convulsions, collapse, or coma; whereas when the disease is allowed to run on unchecked, the chances are in favour of the supervention of pulmonary disease.

Experience in the *post-mortem* room has shown that great caution is necessary in assigning the cause of death in diabetes, unless an autopsy is made. In two cases of diabetes that have terminated fatally in Guy's Hospital the patient died with cerebral symptoms, but in each of them death was found to be due to local inflammation. In one the pelves of both kidneys were dilated and inflamed, and the tissue of one presented numerous points of suppuration. In the other case there was extensive pneumonic consolidation of the base of the left lung.

Morbid anatomy.—The inflammation of the kidneys in the former of these two cases was so exactly like what occurs in cases of stricture and other diseases of the urethra or bladder that these parts were very carefully examined. The urethra was perfectly healthy; the *bladder*, on the other hand, was greatly hypertrophied. This led the author to consider whether the increased thickness of the coats of this viscus could be due to the augmented work it had had to perform in consequence of the over-secretion of urine. In the second case, therefore, we looked at the bladder with much interest, and found that it also was markedly hypertrophied, and that its mucous coat protruded between the muscular fasciculi, so as to form numerous sacculi. Probably hypertrophy of the bladder may be found to be frequently present in diabetes.

With regard to the other appearances found on *post-mortem* examination of those who have died of diabetes there is very little to be said. The viscera have a decidedly sweet smell, resembling that observable during life in the urine and breath; and it may be noted that in one case Dr Wilks found that this odour was still observable, although the patient died of typhus, and his urine (which also retained the sweet smell) had been free from sugar some days before death. The *kidneys* are not infrequently large, soft, and fatty; and they are occasionally affected with chronic tubal nephritis or some other form of morbus Brightii. The liver is sometimes large, and Wilks describes it as having a uniform fleshy appearance.

In some cases of diabetes the blood has been found creamy from the

presence of fatty molecules, and fat-embolism has been found in the lungs (Sanders and Hamilton, 'Edin. Med. Journ.,' July, 1879). But *lipæmia* (as this condition of the blood has been called) is far from constant and is probably very rare. It was first noticed by Rollo, in 1778, and afterwards by Dr Babington the younger. Dr Pavy regards it as a physiological effect of the abundant food that is taken.

In a series of autopsies on diabetic patients collected by Dr Windle ('Dublin Medical Journal' Sept., 1883) the *liver* was reported normal in 84 cases, enlarged in 57, congested in 40, fatty in 15, and tubercular in 2.

The *kidneys* were normal in 115, enlarged in 88, fatty in 35, affected with tubal nephritis in 6, cirrhotic in 10, and lardaceous in only a single case. The *bladder* was normal in 20, and hypertrophied in 13 cases.

The *lungs* were normal in 81 cases, and congested or œdematous in 37 more, making 118 in which they were free from organic disease. In 136 they were phthisical ("tubercular" in 109, "pneumonic" in 27), to which 17 cases with cheesy masses, 12 with vomicæ, 3 with caseous bronchial glands, and 8 with miliary tubercle may be added, making a total of 178. In 24 cases there was acute lobar or catarrhal pneumonia, and in 3 gangrene, while in another 3 fat-emboli were discovered. The *heart* was normal in 70, large in 4, small in 9. The *brain* was normal in 91, and perivascular changes were discovered in 19. The *cord* was normal in 37, and perivascular changes were found in 11. The sympathetic ganglia were normal in 10 cases, and "cirrhotic" in 5.

In 44 cases at Guy's Hospital, abstracted by Mr H. J. Campbell ('Guy's Hosp. Reports,' vol. xliv, p. 207), the *kidneys* were usually large, sometimes "coarse" in aspect, and occasionally fatty. The presence of glycogen in the looped tubes, as asserted by Frerichs and Ehrlich, was not clearly demonstrable. Sections stained with iodine, logwood and eosine, silver or osmic acid, showed very often the necrotic degeneration of the epithelium described by Ziegler. The *bladder* was hypertrophied in 13 cases.

The *lungs* were affected in 23 cases: in 18 there was ordinary phthisis of the apex, in 1 gangrene, and in 4 acute œdema or pneumonia.

The *brain* was firm and healthy: in 2 cases the perivascular spaces described by Dr Dickinson were seen. That these cribriform spaces are not characteristic of diabetes, and are probably not present during life, seems proved by the observations of Drs Taylor and Goodhart ('Path. Trans.,' vol. xxxiv, and the report, p. 377 of same vol.). Dr Hale White (*ib.*, vol. xxxvi) has confirmed this conclusion, and has also shown that the pigmentation, induration, and other changes of the *semilunar ganglia* reported in diabetes are present in other cases ('Journ. of Phys.,' vol. viii, p. 70).

Physiology of glycose.—The point from which all discussions as to the pathology of diabetes must start is the glycogenic function of the liver.

In 1848 Claude Bernard published the startling discovery that sugar, which had before been supposed to be always a vegetable product, was to be found in abundance in the liver in all animals, even when they had been kept for some time on a diet containing neither sugar nor any of those amylaceous substances that were known to be capable of undergoing conversion into it. He found also that the blood taken from the hepatic vein contained sugar in much greater quantity than that from the portal vein or from the vessels in other parts of the body. From these observations he inferred that sugar was constantly being poured by the liver into

the blood, and that it there underwent oxidation, probably with the object of keeping up the animal heat.

Bernard further showed that when the liver had been removed from the body of an animal great quantities of sugar were formed within it. The organ was deprived of all the sugar it contained by the injection of a stream of water through its vessels, and it again became loaded with sugar after being left to itself for twenty-four hours.

It was therefore evident that there is in the liver some substance which, not itself sugar, is capable of becoming sugar, and in 1857 this substance was isolated in the form of a white powder by Bernard and called glycogen.

In repeating Bernard's experiments, Dr Pavy found that the conclusions drawn from them were inaccurate in one important respect, namely, as regards the presence of sugar in considerable quantity in the liver and in the blood of the hepatic vein *during life*. He showed that when certain precautions are taken, by which the tendency of the glycogen to undergo conversion into sugar is arrested at the moment of death, no sugar can be detected in the liver after removal from the body; and by catheterising the right ventricle during life he obtained blood which, although it had come straight from the hepatic veins, was approximately free from sugar.

These results are what might have been predicted from a knowledge of the relative properties of sugar and glycogen. The former is a highly diffusible substance, while the latter possesses scarcely any diffusibility. This is why, as in Bernard's original experiment, the sugar can be removed from the liver by a stream of water, while the glycogen remains behind. During life the liver is constantly traversed by a current of blood which at once carries away any sugar that is formed in the organ. It would not be possible for the liver, and still less for the swiftly flowing blood of the hepatic vein, to contain during life the quantity of sugar which Bernard found in them after death.

On the other hand, it is doubtful whether Dr Pavy's experiments, multiplied and varied as they have been, have established the conclusion which he drew from them, namely, that glycogen is not converted into sugar during life, and therefore that some other destination must be discovered for it. The quantity of glycogen present in the liver appears to be always changing; it increases after each meal, and diminishes during fasting. It must therefore be constantly passing into some other substance, or in some other way undergoing removal from the organ. And if we take into consideration the readiness with which it is converted into sugar under the influence of different ferments, we shall certainly require very strong evidence to convince us that this is not its natural destination.

Were it not stored as glycogen in the liver, the sugar absorbed from the stomach and intestines would circulate in the blood in excessive quantity after every meal. It probably could not be at once made use of by the tissues, and some of it would certainly be excreted by the kidneys. The liver takes from the portal blood its sugar, and converts it into the non-diffusible glycogen. This is stored up for a time in the organ, and afterwards during the interval between each meal and the one which succeeds it, the glycogen is gradually reconverted into sugar. Its diffusibility causes it to enter the blood of the hepatic vein, and thence it is carried to all parts of the body.

There may, indeed, be a question whether part of the glycogen does not enter the circulation as such. The white corpuscles of the blood contain

this principle; and it is possible that they may act as carriers of glycogen, and so diminish the quantity of this substance contained in the liver during fasting. But however this may be, there is every reason to believe that sooner or later the glycogen undergoes conversion into sugar.

Pathology of diabetes.—We must now consider what can be learnt of the nature of diabetes from these physiological facts and theories.

It is evident that there are three principal ways in which it is conceivable that the natural processes might become perverted so as to lead to the excretion of sugar by the kidneys. These are:—1. The entrance of sugar into the general circulation, without its having been arrested in the liver and converted into glycogen; 2. An excessive flow of sugar from the liver into the blood; and 3. A diminished destruction of sugar in the tissues. Dr Lauder Brunton ('Reynolds' System,' vol. v, p. 399) admits all these as causes of diabetes, and divides them again into minor heads. It, however, appears improbable that this disease should arise in each of all the different ways by which it can possibly be produced; and there are reasons for believing that it is always due to the second of the three causes enumerated, viz. to an excessive flow of sugar from the liver into the blood.

Let us, as the *first* theory, consider whether diabetes can ever be fairly attributed to a mere failure in the glycogenic function of the liver, allowing glycose to enter the general circulation. It has been proved experimentally that unless it has first passed through the liver the sugar absorbed from the food is incapable of being utilised in the system, and that it is excreted unchanged by the kidneys; and we can easily conceive that the same thing may occur as the result of disease. But it is to be observed at once that such a theory can be applied to very few instances of diabetes, as we see is clinically. For in all severe cases of this disease the urine remains saccharine, even when the patient's diet is restricted, so that it contains neither sugar nor any amylaceous principles which are convertible into sugar. Under such circumstances there can be no doubt that the sugar is formed by the liver, just as in healthy carnivorous animals it is formed by that organ out of the other elements of the food. But there are other cases in which it certainly appears at first sight to be possible and even likely that the disease may be due to a defective glycogenesis on the part of the liver, namely, those in which the urine ceases to contain sugar as soon as the patient confines himself to a properly restricted diet. Nay, since we have seen that in other cases the liver not only can, but does, form sugar out of food containing neither sugar nor starch, it might be thought that for these cases such a theory is not only tenable but alone admissible. Dr Bence Jones declared that in half the cases of diabetes that came under his observation the disease consisted simply in an arrest of change in the sugar, brought to the liver through the portal vein; and Dr Pavy has expressed a similar view with regard to the cases now under consideration.

But a closer examination of the question cannot fail to suggest doubts as to the validity of this explanation. If such a patient, whose urine is kept free from sugar by a restricted diet, one day breaks through the dietetic rules laid down for his guidance and eats an apple or drinks a glass of sweet beer, it will generally be found that the secretion again becomes saccharine. This is no more than might be expected; but now comes a circumstance which is remarkable, and for which some further explanation is evidently necessary. The quantity of sugar that is voided is altogether disproportionate to the amount contained in the apple or the beer; and

sugar often continues to be excreted for a very long time afterwards. Dr Pavy mentions the case of a patient in whom the disease had been kept under control by strict dieting, and who drank about a pint of cider. His urine thereupon became loaded with sugar, and remained so for a period of two months, before it again became normal. Now, the only hypothesis which seems capable of explaining such facts as these is that the saccharine or amylaceous article of food exerts some directly injurious influence, so as to cause the blood to contain an excess of sugar for long afterwards; and if we believe that the liver is the organ principally concerned in supplying the blood with sugar we can hardly help inferring that it is the liver on which this injurious influence is exerted. It seems as if saccharine food were a *poison* to a patient who is affected in this way. It is evident that this very hypothesis affords an adequate explanation of the fact with which we started, that in certain cases of diabetes the urine contains sugar only so long as the patient takes saccharine or starchy matters in his food.*

We may next consider whether there is reason to believe that diabetes is ever due to the *third* of the causes enumerated above, namely, deficient destruction of the sugar in the systemic capillaries, or rather in the tissues.

Now, Dr Owen Rees formerly showed that the sugar which is found in the urine in diabetes does not possess exactly the same properties as the sugar which is formed from hepatic glycogen. It is much less ready to undergo destruction in the presence of blood or other animal matters. It is therefore possible to suppose that, even in the most extreme case of diabetes, the total amount of sugar formed during the twenty-four hours might not really be excessive, since its escape in such large quantity in the urine might be due to its failing to become used up by the muscles and other tissues. It must be borne in mind that we have no knowledge as to the absolute quantity of glycogen which is naturally formed by the liver each day and subsequently converted into sugar.

But if the physiological theory above given is correct, there is a test which may be applied without difficulty to any case which is supposed to be due to this cause. We have seen that the object of the glycogenic function of the liver is to equalise the flow of sugar into the blood, instead of its entering the circulation in large quantity after each meal and being absent during fasting. We have therefore reason for expecting that in any case of diabetes due to imperfect destruction of sugar, while the liver is performing its functions naturally, the excretion of sugar would be uniform throughout the day, and that it should be entirely uninfluenced by the meals. Now, it appears that this is never the case. This is illustrated by a case which Dr Pavy studied very carefully, and of which he has published a detailed account in his work. A man, whose name was Joseph North, was placed in succession upon different kinds of food, and his urine was collected and analysed every four hours. As a very general rule, the

* When there is extreme obstruction to the portal circulation in the liver, the blood must needs go in great part by other channels, and we found evidence of the fact (*supra*, p. 526). Under such circumstances glycose absorbed from the stomach and intestines would not be transformed into glycogen, but would be excreted by the kidneys, and thus glycosuria would result. Bernard himself tied the portal vein in a dog, and thus produced artificial *glycosurie alimentaire*. Dr Lépine (following Couturier) observed the same result in three patients with cirrhosis of the liver (verified after death) who were fed on starchy and saccharine diet, but he failed to obtain it in cases of cancer of the liver and of fatty liver in phthisis. The result, however, is far from constant; it failed when tried several years ago by the editor at Guy's Hospital, and sugar ought far more frequently to appear in the urine in cases of cirrhosis, adhesive pylephlebitis, and the like affections.

amount of sugar excreted in the urine was considerably greater between 5 and 9 p.m. than at any other part of the day; while, on the other hand, it was commonly at its lowest point during the hours of the night and early morning. These variations were evidently due to the influence of the food taken during the day. A striking illustration of this is afforded by one occasion, when the man departed from the instructions given to him, and at 4 p.m. drank some cocoa sweetened with sugar. Between 5 and 9 p.m. of that day his urine contained 1311 grains of sugar, whereas in the twenty-four hours previously the quantity in equal periods of time had ranged between 166 and 468 grains; and from 9 to 1 a.m., after the cocoa had been taken, it was again only 483 grains.

For such a case as this, any theory which refers the glycosuria to deficient destruction of sugar would be altogether untenable; and there is no reason for doubting that similar variations in the amount of sugar excreted at different periods of the day occur in all cases of diabetes.

We may therefore suppose for the present that diabetes (or persistent glycosuria) is due to the *second* of the three causes enumerated on p. 723 namely, to an excessive flow of sugar from the liver into the blood.

Cause of increased glycogenesis.—It remains to determine how this excessive influx of glycose is brought about. The well-known discovery of Claude Bernard was that in the lower animals the operation of puncturing a spot in the floor of the fourth ventricle is followed by glycosuria, or (as it is called) by artificial diabetes. No fact in experimental pathology is more firmly established than this; and the explanation at present accepted is that the puncture inhibits the vaso-motor centre of the nerves distributed to the hepatic blood-vessels. The vaso-motor nerves being thus paralysed, the hepatic artery dilates. The flow of blood through that vessel is thereby augmented, and the result is an increased conversion of glycogen into sugar, and a flow of sugar into the blood in excess of the requirements of the system.* Whenever the liver of an animal happens to contain no glycogen, the operation fails to cause an excretion of sugar in the urine.

This explanation substitutes for the *excessive formation of sugar* which has been shown to be the cause of ordinary diabetes a mere *increased rapidity in the discharge of sugar* into the blood. Thus, although it may account for glycosuria in Bernard's experiments, it will not explain diabetes. For, according to what has been stated, it is clear that after the puncture, in proportion as the amount of glycogen in the liver becomes reduced by the excessive demands made upon it, the glycosuria must pass off; and, in fact, after a few hours sugar can no longer be detected in the urine of animals on which this experiment has been performed. It is true that Schiff found in rats that by dividing certain columns of the cord he could make the animals diabetic, and that this condition would last for two or three weeks. But even these results scarcely warrant our attributing ordinary persistent diabetes in the human subject to paralysis of the vaso-motor nerves. It seems to be impossible that the excretion of such large quantities of sugar as are passed by patients affected with this disease, at all hours of the day, and in amounts influenced only partially by the meals, can be due to a mere increased rapidity of the conversion into sugar of the glycogen naturally formed by the liver.

It is true that this difficulty might be removed by introducing, as an

* Dr Pavy has shown that one can bring about the same result experimentally by dividing the sympathetic, or by causing arterial blood to flow through the portal vein.

auxiliary cause, Dr Rees' observation, that diabetic sugar possesses greater power of resisting oxidation than the sugar which is normally formed by the liver. As already shown by North's case, this cannot be the sole cause of diabetes; but the line of reasoning there adopted would be no bar to the acceptance of this as a second factor in the development of the disease, if it were also assumed that, in consequence of the vaso-motor paralysis, the flow of sugar into the blood from the liver is accelerated after each meal, instead of being equable, as it is in health.

But it appears much more probable that the really important second factor in the causation of diabetes (in addition to an increased rapidity in the conversion of glycogen into sugar and its discharge into the blood) is an *excessive production of glycogen* itself by the liver.

Diabetes, then, would seem to be the result of an excessive activity of the glycogenic function of the liver. In the first place an undue quantity of glycogen is formed from the materials brought to the organ. In the second place, this glycogen is converted into sugar more rapidly than under normal conditions. Perhaps also the sugar which is formed in diabetes has a greater power of resisting oxidation in the tissues than the sugar which is produced in the healthy liver.

Nervous origin.—These considerations render it improbable that true diabetes is caused by disease of the nervous centres acting upon the liver through the vaso-motor nerves, as in the affection artificially induced by Bernard and others. Nevertheless some interesting cases have been recorded in which the urine has been found to contain sugar after the brain has been injured, or when it was diseased.* As far back as 1854 Dr Goolden published a series of instances of glycosuria following blows or falls upon the head; but in most of them the presence of sugar in the urine was transitory. Dr Pavy, however, mentions the case of a cadet at Woolwich who was attacked with strongly-marked diabetes a few days after being stunned by a violent blow on the head. He also records two instances of diabetes after an attack of hemiplegia, and he has frequently seen it in ataxic patients. The most striking case in which disease of the fourth ventricle has led to a saccharine state of the urine in man is one quoted by Trousseau, in which there was a tumour in the floor of this cavity. Similar cases have been since recorded; but interesting as they are from a physiological point of view, they lend small support to a nervous theory of diabetes.

Ætiology.—Even the predisposing causes of diabetes are as yet very imperfectly understood. Sometimes diabetes appears to be a result of mental anxiety. Dr Hermann Weber met with the case of a gentleman who became diabetic on two separate occasions, at an interval of nine years, under the pressure of intense anxiety from impending commercial ruin. In other cases it has followed a violent fit of grief or of anger.

Whatever we may think of these facts, an *inherited tendency* is certainly sometimes a factor in causing diabetes. Pavy gives numerous instances of this: in one family two sisters and two brothers; in another a son, his father, and an aunt; in a third a father and his two daughters; in a fourth a father and a son; in a fifth two brothers; in a sixth three brothers; in a

* Many years ago (about 1867) a little girl was brought into Guy's Hospital dead from fracture of the skull and other injuries in being run over. A patch of hæmorrhage was found in the floor of the fourth ventricle. Some urine was then collected from the bladder and gave decided evidence of sugar.

seventh a brother and a sister; and in an eighth the father, mother, and three daughters; and (to mention only one other case) a mother, grandmother, and four out of five children. Roberts speaks of a family of eight children, all of whom became diabetic, although the parents were healthy.

One hundred and twenty-two cases have been collected by Mr J. E. Nevins from the records of Guy's Hospital, and in eight of these there was a history of diabetes in one or more members of the patient's family. Adding cases obtained by the courtesy of the registrars of the three other large hospitals, St Bartholomew's, St Thomas's, and the London, and those recorded by Schmitz and Griesinger, he finds thirty-five hereditary cases in a total of 537. In one remarkable case the mother and seven of the mother's brothers had died of diabetes.*

Distribution.—Diabetes is said to be rare in Northern Europe, more common in Ireland and Scotland than in England, comparatively rare in Berlin, but common in Saxony and Thüringen, and more common in Ceylon and parts of India than in other tropical countries. Jews are said to be particularly liable to it, and negroes to be almost exempt.

Dr Pavy finds that it is more frequent in private than in hospital practice, and that on the average private patients are older than those in hospital.

Age and sex.—Diabetes rarely attacks children under five years of age. According to Roberts women become less liable to it after forty-five years of age; whereas in men there is no decrease in the mortality from diabetes until the age of sixty-five years has been passed. Among 1360 private patients who came to Dr Pavy, diabetes began under ten years old in 8, between ten and twenty in 57, between twenty and thirty in 97, between thirty and forty in 224, forty and fifty in 339, fifty and sixty in 418. Even between sixty and seventy the number was 182, very large considering the fewer number of persons living over sixty compared with those between twenty and forty. Above seventy there were 35 patients, one in whom the disease began when he was over eighty. In every decade (excepting the children under ten) the number of males was far greater than that of females, and the total was 966 to 394. The youngest patient he ever saw was an infant twelve months old ('Brit. Med. Journ.,' Dec. 5th, 1885).

It is far more common in men than in women: in cases at Guy's Hospital, above two to one; in Dr Pavy's private cases, 2·45 to one.

Prognosis.—In its well-marked forms diabetes always tends to the death of the patient, and that in a comparatively short time. It is true that one cannot often state definitely what is the whole duration of the disease, because its onset is generally insidious; but for practical purposes this is of no consequence; the important fact is that (for all but elderly people) when the urine has once been found to be persistently saccharine, the days of the patient are numbered, unless the progress of the malady can be arrested.

Cases are sometimes met with in which death occurs within a few weeks from the time when the first symptoms of the disease appear. Sir George Paget observed a case in which the patient, a Cambridge undergraduate

* It has been stated by Dr Maudsley that diabetic parents often have insane children, and Dr Dickinson found glycosuria in 17 per cent. of insane persons. But Dr Hale White found it in only 4 per cent. of the inmates of Bethlem, and in 2·6 at the Surrey County Asylum. Dr Savage also believes that insanity and diabetes are unconnected ('Brit. Med. Journ.,' 1885, vol. ii, p. 1054).

supposed to be in perfect health, took part in athletic sports and came in second in a race, within twelve days of his death. More frequently, however, the duration of diabetes is from one to three years. Prout said in 1848 that, among nearly seven hundred patients whom he had seen within thirty years, he then knew of but two in whom the disease had been perfectly developed ten years before. Dr Dickinson, however, mentions the case of one patient in whom the urine was constantly saccharine for fifteen years, and Dr Pavy a similar case which lasted twenty-five years.

The *age* of the patient appears to have a greater influence than any other condition on the rate with which diabetes advances towards a fatal termination. Children who are attacked by it never live to grow up. On the other hand, old people sometimes pass saccharine urine for years without appearing to suffer much in health; but in such patients the urine is seldom excessive in quantity, nor are marked symptoms of diabetes generally present. At a middle period of life the prognosis of a case of diabetes, when it first comes under observation, must mainly be based upon the degree of severity of the symptoms. Thus all writers regard it as a favourable sign if the patient remains well nourished and florid. It is generally said that the detection of albumen in the urine in addition to the sugar is a serious indication, but Dr Pavy says that he has known a small quantity of albumen to be present for years without apparent harm.

But these considerations, which guide the physician in forming his first impression as to the probable issue of a case of diabetes, require to be combined with, and corrected by, the results of treatment, before a final judgment is pronounced. For medical science sometimes attains more striking results in this disease than in almost any other of equally dangerous tendencies, and the cases which do best under treatment are not always those which appeared the most promising.

Treatment by diet.—If the view above taken of the pathology of diabetes be correct, the rational treatment of the disease is clearly to limit as far as possible the ingestion of any substances which experience shows to be especially apt to stimulate the liver to the excessive formation of glycogen and sugar; and all starchy and saccharine matters have this property Thus, as Prout long ago laid down, "diet is the first and chief point to be attended to." Dr John Rollo, towards the end of the last century, appears to have been the first to make a suggestion of this kind; and he proposed to confine diabetic patients entirely to animal food. But although the inhabitants of arctic regions and the trappers of North America live for months together without the chance of obtaining anything else, experience shows that in civilised life, when all kinds of food are within reach, there is very great difficulty in keeping patients exclusively on meat; and the more so, since in diabetes the appetite is voracious, and the craving for forbidden food all the greater. Of late years, therefore, all those who have studied the subject have striven to include among permitted viands as many of vegetable origin as possible. The result has been the construction of a tolerably copious diet-table.

Almost all kinds of *animal food*, flesh, fowl, and fish, may be eaten by persons suffering from diabetes. But it is necessary to bear in mind that the ordinary methods of cooking such food often introduce deleterious matters. Thus, soups are thickened, jellies are sweetened, and even roasted

joints are basted with flour. There are few alimentary substances of animal origin which produce sugar; but such is the liver of calves or pigs, and such are also the edible molluscs, oysters, cockles, mussels. All of these undoubtedly contain much glycogen, and Roberts forbids them; but it may be noted that in the case of Joseph North, to whom Dr Pavy twice gave four dozen of oysters for two days running, they caused no decided increase in the amount of sugar excreted. *Honey* (containing dextrose and lævulose) is of course injurious; and so is also *milk*, but not in the degree that might have been inferred from the amount of lactose contained in it. Dr Pavy indeed found that in North's case the administration of three pints of milk daily not only caused a marked increase in the amount of sugar, but also brought back the uneasy sensations which the man had experienced when the disease had been uncontrolled by treatment. But Roberts mentions the case of a girl who (her diet being restricted in other respects) continued to gain strength and to improve in health when she was allowed to drink three pints of milk daily.

Cream and butter form important articles of diet in diabetes. Cod-liver oil is useful in a similar way. Though glycerine might be expected to be harmless, since its composition is so different from that of sugar, yet Dr Pavy found that it caused a great increase in the sugar excreted; but the daily quantity which he administered was from six to ten ounces, and perhaps it does not follow that smaller quantities would be injurious. A better substitute for sugar than glycerine is the lately introduced Saccharine (Benzoyl-sulphonic-imide). It sweetens far more powerfully than cane sugar, and of course does not yield glycose; but it renders the saliva unpleasantly sweet and possibly may have other drawbacks.

Dr Donkin's success in treating diabetes by restricting the patient entirely to a diet of *skimmed milk*, of which he allows six or eight pints daily, is difficult of explanation, if his results are compared with those of others. Roberts refers to the cases of several persons treated with skimmed milk, and says that few of them could tolerate it for more than a few days, and then were rapidly reduced, while one case was brought to a fatal termination in three months. Dr Greenhow, however, has recorded a case in which a patient took from four to six quarts of skimmed milk daily with the effect of removing his symptoms and of freeing his urine from sugar. Two months afterwards he remained quite well.

Most *vegetables* are injurious to diabetic patients. The most noxious of all are said to be potatoes; and carrots, parsnips, turnips, peas, beans, Brussels sprouts, cauliflowers, broccoli, asparagus, and sea-kale are also forbidden, as containing starch or sugar.

Greens, however, as well as spinach, lettuces, watercresses, and mustard and cress may be eaten; and even small quantities of radishes and celery. The general rule is that all white parts of vegetables, in which chlorophyll has not been developed by exposure to sunlight contain sugar, and are harmful. But by boiling in a large quantity of water, even the forbidden kinds of vegetables, if they contain sugar only and not starch, may be rendered much less injurious. According to Prout, there is a direct advantage in the use of such green vegetables as are harmless; for they are in great part incapable of being digested, and so tend to correct the constipation which is often so troublesome in diabetes.

All kinds of sweet fruits appear to be injurious, although it has been

said that currants and raspberries and other acid fruits may be eaten by diabetic patients. Nuts are harmless; but chestnuts abound in starch.

Wheaten flour, as well as that of other kinds of corn, is of course very injurious; and so are rice, arrowroot, sago, tapioca, macaroni and vermicelli, which chiefly consist of gluten. It is with regard to *bread* that the diabetic patient finds the greatest difficulty in carrying out the rules of diet laid down for him. Consequently, there have been many attempts to invent a substitute for bread, which forms so large a part of the food taken by most civilised races of man. The earliest of these attempts was perhaps that of Bouchardat, who, in 1841, suggested that a kind of bread should be made from the gluten of flour, after removal of all the starch by washing. But this gluten bread is by no means perfectly free from starch, for it at once turns blue if a little iodine be dropped on it. Patients also complain that it is very unpleasant to chew, feeling like india-rubber between the teeth. Dr Pavy says that gluten biscuits are much more palatable, but these will not keep more than ten days. Gluten is also prepared as a kind of flour, and in many other ways, to be used for cooking purposes, by which variety may be given to the meals put before a diabetic patient. A second substitute for ordinary bread is a sort of cake made from bran. Prout appears to have first suggested this, and Dr Camplin, who was himself affected with diabetes, greatly improved the method by which it is prepared, so that bran cakes are still made according to his directions. A third substance which may be used by diabetic patients instead of bread is the almond food which was introduced by Dr Pavy; its only fault is its high price.*

It is of course necessary to regulate the *drink* as well as the food of diabetic patients. The first question is whether the quantity of fluid taken into the stomach should be restricted. This, it appears, is attended with but little advantage, and it adds greatly to the patient's discomfort.

The beverages which are admissible are tea, coffee, cocoa from nibs, dry sherry, claret, burgundy, chablis, hock; also brandy and other unsweetened spirits, Burton bitter ale in moderation, and, of course, soda and mineral waters. Chocolate, sweet ales, porter and stout, cider, all sweet and sparkling wines are to be forbidden. It is not desirable that alcohol should be taken in any considerable quantity by diabetic patients.

It remains to be considered whether the restricted diet should be commenced gradually or suddenly. On the principle laid down above of the *poisonous* action upon diabetic patients of saccharine and starchy substances, the effects of which persist for a considerable time after their ingestion, the excretion of sugar in the urine cannot be expected to subside otherwise than gradually even when all such substances have been withdrawn from the food, and experience shows that this is the case. Dr Pavy says that the patient often at first complains that his food is not bulky enough to satisfy him; but after persevering for a few days he finds that his appetite becomes less. Johnson's dictum that it is easier to abstain than to be abstemious is very applicable to the diabetic.

Before a patient suffering from this disease adopts the restricted diet, and also while it is being carried out, the amount of urine passed each day and its specific gravity should be carefully registered, and the quantity of sugar should also be accurately determined at frequent intervals, if not every day. In no other way can the progress of the case be properly watched, or an opinion be formed as to the necessity for subsidiary treat-

* Dr Palmer, of Nayland, recommends the *boules de soupe*, little dumplings of gluten.

ment. Moreover, a daily analysis enables the physician to check the proceedings of his patient, and to tell with considerable certainty whether he is adhering strictly to the prescribed rules. Those who suffer from diabetes are often untruthful, and wilfully deceive their medical advisers. Prout speaks of a mental imbecility and want of fortitude as one of the effects of the disease. This appears to be the explanation of the assertions that have been made that diabetic patients sometimes pass a quantity of urine exceeding that of the fluids ingested. Niemeyer mentions a case, in which careful watching proved that the patient had really been drinking a larger quantity of fluid than that to which she had confessed. Patients are often detected in having surreptitiously eaten apples, bread, &c., by the discovery that the urine has on some one day been found to contain a larger quantity of sugar than had been present before. The advantage gained by daily analysis can therefore hardly be over-estimated.

The effect of dieting alone, without any medicinal treatment, varies greatly in different cases of diabetes. Sometimes, but very rarely, it cures the disease entirely. Roberts gives a case of this kind. A man aged thirty-nine was passing eight pints of urine daily, containing 5680 grains of sugar. Under a restricted diet the mean daily flow of urine went down within a week to 60 ounces; the sugar fell on the third day to 134 grains, at the end of a week to 48 grains, and after a fortnight only a trace of it could be detected. The patient lost all his symptoms, and gained flesh at the rate of three pounds a week. In about eight months the last trace of sugar disappeared. When seen nearly four years afterwards he was quite well.

But in the great majority of cases the results of dieting are much less decisive than this. The patient's symptoms may be greatly benefited, or altogether removed; the amount of urine which he passes may be much diminished, or reduced to the normal quantity; it may even cease to contain sugar, so that for the time all signs of the disease are absent; and yet, if he now attempts to return to his previous way of living, the urine soon again becomes saccharine, and one by one all the symptoms of diabetes reappear.

Indeed, even when the patient under strict diet loses his symptoms, and the quantity of urine which he passes falls to the normal amount, it by no means always ceases to contain sugar. Its specific gravity may be hardly, if at all, lowered; and the percentage of sugar (although not the absolute amount excreted) may be as great as before. Whenever dieting, or, indeed, any other treatment, does good, it appears always to bring the quantity of water down to normal, before seriously affecting the specific gravity or the proportionate amount of sugar contained in the secretion. But whenever dieting readily lowers the specific gravity, it may be assumed that the quantity of urine passed by the patient is not excessive.

Drugs.—Sometimes a restricted diet altogether fails to control diabetes. There are cases, especially those of very young subjects, in which all treatment appears to be useless. Other patients, again, are at first benefited by dieting; but after a time it ceases to be useful, and in certain cases the disease appears to advance less instead of more rapidly when all restrictions are withdrawn. Sometimes, again, the appetite fails entirely as soon as the patient is required to give up starch and sugar.

Whenever we have reached the limit of the good that can be effected by diet alone, the next step should be the administration of *opium*. The

value of this drug in the treatment of diabetes was known to Prout, and Sir Thomas Watson spoke of it as a treasure. It was also known that patients suffering from this disease could take much larger quantities of opiates than healthy persons. The earlier writers, however, recommended that its dose should as much as possible be kept within moderate limits. Dr Pavy advocates a very different way of administering opium. He gives it in increasing doses, until he finds either that the disease yields or that the patient is unable to take it. In what way opium acts in diabetes there is difference of opinion. Sir William Roberts—who says that, without any restriction as regards diet, daily doses of six to twenty grains always reduce the quantity of urine by about one half—declares that in patients who are dieted it is altogether useless, except to induce sleep and allay irritability. This, however, is inconsistent with Dr Pavy's accurate observations.

In some cases opium disagrees, either causing narcotic symptoms or, not infrequently, diarrhœa. When the former effects are produced, there is often great advantage in the employment of *codeia*, as was first recommended by Dr Pavy. Half a grain three times a day should be given at first, but the daily quantity has afterwards been gradually raised to as much as thirty grains. Often codeia may be given without any obvious effect beyond diminishing the sugar excreted. The tongue remains clean and the appetite good. Sometimes, however, codeia causes headache.

Ammonia and alkaline carbonates are sometimes of service. The waters of Vichy have great reputation in France, and those of Carlsbad in Germany.

Diabetic patients should be clothed in flannel. Bouchardat maintained that active exercise should be taken, and Trousseau recommends it in the strongest terms,* but few English physicians agree with the advice.

The intolerable thirst of diabetes may be relieved by dilute phosphoric acid, or solution of bitartrate of potash. Constipation should be treated with Ol. Ricini or Conf. Sennæ rather than with saline purgatives. The painful sensation of sinking at the epigastrium is said by Roberts to be sooner relieved by two or three grains of assafœtida than in any other way.

* "Un diabétique, qui chaque jour, fait à pied un exercise violent, peut, sans changer rien de son régime, retrouver temporairement la santé qu'il avait perdue," &c. ('Leçons,' tome ii, p. 764). It is curious to note that Celsus wrote:—"At, cum urina, super potionum modum etiam sine dolore profluens, maciem et periculum facit, si tenuis est (*i.e.* clear and pale) opus est exercitatione," &c. (lib. iv, cap. xx).

ADDISON'S DISEASE

WITH OTHER AFFECTIONS OF THE ADRENAL BODIES, AND REMAINING ABDOMINAL VISCERA

The anatomical changes in the adrenal bodies—The symptoms: melanodermia, asthenia, cardiac failure, gastric disturbance—Order of the symptoms—Course, mode of death, and duration of the disease—Controversial points—Origin—Relation to caries and to tubercle—Theory of the symptoms—Prognosis—Other morbid conditions of the adrenal capsules.
Diseases of the pancreas, the spleen, the testes, and the ovaries.

AMONG the most remarkable of all the diseases with which modern pathological anatomy has made us acquainted is one which has its seat in the suprarenal (or adrenal) bodies. It was first recognised by Thomas Addison (some years before the publication of his work on the subject in 1855), and consequently it is spoken of as "Addison's disease."* The symptoms which characterise it may be briefly enumerated as (1) gradually increasing and fatal weakness of the heart and other muscles; (2) a peculiar discolouration of the skin; (3) liability to nausea and vomiting.

Anatomy.—The exact characters of the affection of the suprarenal bodies which gives rise to these symptoms vary considerably in different cases, but only within such limits as may fairly be supposed to belong to the successive periods of a single morbid process. The earliest change seems to be the formation, within the medullary substance, of a firm grey or whitish mass, which is more or less nodulated at its growing edge and sometimes surrounded by clusters of what appear to be miliary tubercles. In this stage, however, the disease rarely becomes the subject of anatomical investigation. When death has occurred, the grey material is generally found to have already extended into and destroyed the cortical substance. The adrenal is then greatly enlarged, hard, and of irregular form; when a section is made the cut surface shows no trace of the natural structure; it may be of a more or less uniform grey colour, which, according to Dr Greenhow ('Croonian Lectures,' 1875), quickly acquires a pink hue when exposed to

* *Melasma suprarenale* was the name proposed by Addison in his original work on 'The Constitutional and Local Effects of Diseases of the Suprarenal Capsules,' 1885 (republished in Addison's works by the New Sydenham Society, 1868).

The most important later facts on the subject will be found in the ninth volume of the 'Pathological Transactions' (1858), in Dr Wilks's papers in the eighth and eleventh volumes of the 3rd series of 'Guy's Hospital Reports' (1862 and 1865), and in Dr Greenhow's Croonian Lectures "on Addison's disease" (1875).

Dr Wilks has drawn attention to two undoubted cases of the disease, described (but not recognised as dependent on the lesion of the adrenals) by Aran in 1846, and by Bright somewhat earlier, the diseased organs of the latter case being still preserved in our museum ('Guy's Hospital Reports,' 3rd series, xxii, p. 266).

the air. In most cases, however, certain parts of the grey substance have undergone caseation, forming rounded yellow nodules embedded in it; and, at a still later stage, the conversion is complete, so that there is only a single large yellow mass. Softening often takes place at this period, and a creamy liquid results, which used formerly to be mistaken for pus. Finally, a process of absorption begins, and the diseased organ, from being many times larger than natural, shrinks into a very small puckered mass, in which irregular nodules of calcareous matter are deposited.

During the early stages of this process, the fibrous envelope of the organ becomes swollen until it is from half a line to two lines in thickness. Adhesions to the neighbouring parts are also formed, principally to the diaphragm or stomach, liver, kidney, pancreas, or vena cava.

In some cases nothing is left but a hard puckered fibrous knot, sending out bands into the fatty tissue in which it lay embedded, and often containing irregular calcareous masses.

The morbid changes in Addison's disease are probably never limited to a single suprarenal body. One, however, is generally attacked earlier than the other, and on *post-mortem* examination may be found completely destroyed, while its fellow is still in a comparatively early stage of disease.

The new tissue which is formed at the commencement of the morbid process consists of small round cells of lymphoid character lying in the meshes of a delicate, wavy, fibrillated stroma. These cells may subsequently undergo development, and form well-marked fibrous tissue, for some parts, still grey and translucent, have been found to consist entirely of such tissue, with some elongated cells and oval nuclei interspersed between its fibres. It seems, from Dr Wilks's original description in the 'Guy's Hospital Reports' for 1862 and 1865, that the specimens that he examined were generally in this stage. He speaks of a material, which he says is "without structure, or sometimes slightly fibrillated, or containing a few abortive nuclei or cells." When caseation has taken place the microscopical appearances are the same as those of any other structure which has undergone this change.

Melasma.—The discolouration of the skin which occurs in Addison's disease varies considerably in different cases, not only in intensity, but also in tint. Thus it is variously described as being yellowish or greenish brown, dusky, smoky, or as if stained by walnut juice. The term "bronzed-skin" was at one time commonly used for it, but is not a very apt one. One can hardly give a better idea of the hue than by saying that it resembles that of one or other of the dark races of mankind. As in persons who have long resided in hot countries, the discolouration is deeper on the face and neck, and on the backs of the hands, than on the covered parts of the body generally; but it is very marked on the genital organs and about the pubes, and also in the axillæ, navel, nipples and areolæ. In extreme cases it may be universal, so that the patient looks almost like a negro; but it is never uniformly deep over all parts of the body. There is no sharp line of demarcation between the discoloured parts and those which remain pale; they shade off into one another.

In most cases, some parts of the surface also present a few small black spots, resembling minute pigmented moles; these have comparatively defined outlines.

Parts of the body which are habitually rubbed are apt to become the

seats of pigment. Thus a brown ring is often seen round each leg where the garters have pressed; or round the waist in women, where the petticoat strings have been tied. A case is quoted of a baker's lad, whose shoulders showed dark stripes corresponding with the bands by which his basket had been slung over his back. So, again, the application of a blister to any part of the skin is followed by the formation of a brown patch; and deep stains are left by eruptions attended with congestion of the skin.

The stains and patches just alluded to are but an exaggeration of pigmentation that occurs in healthy persons, particularly in those of dark complexion; and the diffused discolouration of the face, neck, and hands may be compared with what would naturally be observed in anyone exposed to the heat of a tropical sun. Possibly the pigment is really deposited under the influence of solar rays. Some years ago, Dr Pavy had under his care a woman who suffered from the characteristic constitutional symptoms; she had on her legs reticulated markings, such as are seen in persons who habitually sit before the fire, and which commonly receive the name of *ephelis ab igne*. A short time afterwards she died, and disease of the suprarenal bodies was found to be the cause of her death.

The discolouration of the genital organs, axillæ, and nipples may also be regarded as an intensification of physiological deposits of pigment; and the small black scattered spots are perhaps representatives of the moles which they resemble. The same may also be said of certain marks which are seen on mucous membranes. Thus each lip commonly shows a bluish-black streak along the line of contact with the other lip; and on the buccal mucous membrane irregular and ill-defined brownish stains may occasionally be seen. These last Dr Greenhow has traced to the irritation set up by the pressure of protruding teeth. But probably they also occur independently of any such cause, for similar stains are found in the lining of the cheeks of Lascars. Another part of the mucous membrane upon which Dr Greenhow has noticed stains in Addison's disease, is the side of the tongue; they are of a purplish blue, or inky hue, and are situated near the free margin.

The conjunctivæ always remain pearly white, contrasting with the dark colour of the surrounding parts of the face.

The microscopical appearances of the discoloured parts of the skin bear a close resemblance to those which may be observed in individuals belonging to the darker races of mankind. The pigment consists of yellowish-brown granules, and its chief seat is in the deepest layers of the rete mucosum, close to the papillæ.

Dr Greenhow, however, remarks that he has sometimes found traces of pigment in some of the more superficial scales of the epidermis, and likewise in the cutis. German observers also have detected pigmented connective-tissue cells within the papillary layer; but there is nothing unusual in the presence of colouring matter there, for it occurs in pigmentary moles.

It is still a question whether the internal organs are discoloured in cases of disease of the suprarenal bodies. Addison's work contains drawings of a mesentery, intestine, and omentum, over all of which numerous minute black spots were scattered. They were taken, however, from a doubtful case, and the pigmentation of the peritoneum may have been due to a former attack of peritonitis. The late Dr Carrington once observed the normal pigmentation of the pia mater covering the bulb much intensified in a case of Addison's disease.

Asthenia, &c.—Next to the discolouration of the skin, progressive weakness is the most striking and important of the symptoms of Addison's disease. The patient becomes more and more languid as it advances. He takes to his bed, and his prostration increases until he becomes unable even to sit up. Merely being raised into a sitting posture may be followed by faintness and giddiness. Yet there is often but little loss of flesh; and after death a certain amount of fat may still be found. Anæmia also, though usually present, is not extreme.

The *heart's action* is remarkably weak, and the *pulse* very small and compressible. Breathlessness, palpitation after any muscular effort, frequent sighing or yawning, and persistent hiccough, are other symptoms more or less constantly noticeable; and the patient may complain of pain in the loins or in the epigastrium. The temperature is unaffected except by phthisis or other complications.

Nausea, with retching and vomiting, is very rarely wanting, and sometimes the gastric symptoms are the most prominent of all.*

Course.—The development of Addison's disease is gradual; and the order of the symptoms variable. Sometimes the skin becomes dark a long while before the general health begins to fail. Thus, a young lady, whose case was recorded by Addison, had become "bronzed" for one year before her death, but appeared ill during only about four months; and a man who was under the care of Mr Harris, of Hackney, had been noticed by his wife to be getting darker for two years, but mentioned no other symptoms as present for more than six months. In the great majority of cases, however, the patient suffers from progressive asthenia for a considerable period before pigmentation of the skin becomes noticeable, and if the former should develop itself rapidly in a severe form, death may occur at a time when the latter is still entirely absent.

In a series of 228 cases, collected by Dr Greenhow, there were twenty-nine in which, when the patients died, the skin was either not bronzed at all, or to a scarcely appreciable extent. But in eighteen of these some other disease was likewise present, which probably was immediately concerned in bringing about the fatal issue. In each of the remaining eleven cases in which there was no such complication, the patient's illness had been of comparatively short duration; in one only had it lasted eight months, and in another four months. However, at Guy's Hospital one case has occurred in which there was no discolouration, but in which the patient had been ailing for twelve months before his death. Probably the most precise statement which we can make is that the skin is always dark when the other symptoms of Addison's disease have lasted more than a year.

Although the course of this remarkable malady is progressive, its rate of progress is by no means uniform. Dr Greenhow has pointed out that alternate exacerbations and remissions occur, which can only partially be traced to changes in the conditions under which the patient is placed. It is said that the depth of discolouration of the skin may alternately diminish and increase.

Death sometimes takes place very gradually. The mind may be clear to the last, or the patient may lie in a drowsy and semi-comatose state, from which, however, he may be roused to give pertinent though slow answers. In such cases the temperature falls considerably below normal. Or death may be preceded by muttering delirium or coma. One of Dr Greenhow's

* On the urine in Addison's disease see Rosenstirn's paper ('Virch. Arch.,' lxi p. 27).

patients had a convulsive fit, and lay for hours with closed jaws and rigid limbs; whenever he was touched, convulsive twitchings took place. In some cases, again, the fatal termination occurs unexpectedly, the patient having been apparently pretty well a few hours before. Thus a young man some years ago was under the care of Dr Wilks for eczema which was getting better, when he was attacked with diarrhœa and vomiting; he became exceedingly prostrate, and died in less than twenty-four hours. Disease of the suprarenal bodies had never been suspected, but was found to be the cause of his death. A patient of the editor's who was in the hospital with Addison's disease but was so much relieved that he was about to leave, became suddenly worse and died in a few hours.

The *duration* of Addison's disease appears to be very variable. At least there are great differences in the length of time which elapses between the commencement of the symptoms and the death of the patient; but it must be admitted that the period at which the affection of the suprarenal bodies begins to develop itself cannot be determined. Dr Wilks some years ago stated that the average duration of the cases which he had collected was eighteen months. One of the most rapidly fatal instances is that of a girl who came under Dr Greenhow's care, and who, although weak and supposed to be sunburnt, attended school until about a week before she died. The longest case was one on which the author made a *post-mortem* examination in 1865. The disease had been diagnosed by Dr Gull at the time when the patient was first admitted into the hospital in 1860; and he then said that his skin had already been dark for two years. In this instance, therefore, the disease lasted for at least seven years.

Spurious and aberrant cases.—In describing the morbid anatomy of Addison's disease, we have implied that the affection is always of one kind. This, however, was not the opinion of Addison himself, who, in his original work, included four cases of cancer of the suprarenal capsules, and who at that time thought that any affection completely destroying the organs would be capable of giving rise to the characteristic symptoms. It was Dr Wilks who first pointed out that in all genuine instances one particular morbid change in the capsules is found. He showed that in Addison's cancerous cases the proper symptoms were not really present. He also first defined the characters of the discolouration of the skin. The errors into which Addison fell in regard to these points are much to be regretted, for they have led to much misunderstanding, and even to doubts as to the existence of the disease, which have not yet been completely dispelled. Its reality, however, is surely proved, beyond the possibility of dispute, by the mere number of cases that have been reported, many of which have been diagnosed during life in the most positive manner. In his lectures delivered before the Royal College of Physicians in 1875, Dr Greenhow was able to cite 183 recorded cases, in every one of which the special form of discolouration of the skin, and some at least of the constitutional symptoms, were present.

The cases which have been supposed to lead to conclusions adverse to those maintained by Dr Wilks and other modern supporters of Addison's views fall into two groups.

In the first group come those in which the capsules have been diseased *without giving rise to the characteristic symptoms.* We have seen that in some uncomplicated cases of Addison's disease death has occurred before the time at which bronzing of the skin usually developes; and in others a rapidly fatal issue has been due to some other malady, such as

phthisis or Bright's disease, by which the constitutional symptoms of Addison's disease were masked. Apart from such cases, Dr Greenhow has been able to state that in every recorded instance of suprarenal disease without symptoms, the affection was really different from that which alone is known to be capable of causing characteristic effects. In most instances it was cancer. The adrenals, indeed, are liable to be the seat of primary malignant growths, as well as to certain secondary nodules of various kinds. In one of our cases each of them was three or four times as big as the kidney, so that there was a large tumour observable during the life of the patient. It may seem strange that such an affection should not cause the symptoms of Addison's disease. But, as Dr Moxon remarked, there is an analogous circumstance in the fact that the most extensive cancerous growths in the liver often fail to produce jaundice. Indeed, there is reason to believe that the cause of the symptoms in question is the extension of morbid changes beyond the suprarenal bodies to the semilunar ganglia and sympathetic nerves; and it may be that these structures are not affected in the same way by even the largest malignant tumours.

Cases of Addison's disease have, however, been recorded by excellent observers, in which, instead of the usual changes, simple *atrophy of the adrenals* has been found: by Dr Wickham Legg ('St Barth. Hosp. Rep.,' 1874), Mr Davy ('Path. Trans.,' xxxiii, p. 360), Dr Goodhart (*ibid.*, p. 340), and Dr B. Fenwick (*ibid.*, p. 354, with table of cases). See also the same 'Transactions' for 1885, and the 'Clinical Transactions' for 1886.

The second group of cases which have been supposed to justify doubts as to Addison's discovery consists of those in which bronzing of the skin has been said to exist *without the characteristic suprarenal affection.* But it must be borne in mind that discolourations of the skin are from time to time met with, which may be mistaken for that of Addison's disease by an unpractised eye, although they are really altogether different. In one of the most curious of these the skin becomes, as it were, "piebald." Some parts are much darker than natural; others are entirely devoid of pigment, and the hairs upon them white. The white areæ always have very definite convex borders, and thus look as if they were encroaching upon the bronzed parts, which, on the other hand, shade off very gradually into the healthy skin. Thus the absence of pigment in some places is far more obvious than its excess in other places, and the affection is accordingly known by the name of "leucodermia." Addison, however, himself confounded it with what occurs when the suprarenal capsules are diseased; and in one of his cases, which appears to have been genuine, the skin did actually present an affection of this kind. All subsequent experience, however, has tended to show that leucodermia is altogether distinct from the discolouration which occurs in Addison's disease; and no other instance of it seems to have been associated with impairment of the general health. But not long ago a case of this kind was sent up to Guy's Hospital from a distant county as one of bronzed skin.

Again, as Dr Greenhow points out, elderly persons in indigent circumstances and of unclean habits, especially when infested with vermin, often have the skin of the back, chest, and abdomen deeply pigmented, so that the presence of disease of the adrenals might be suspected. The roughness of the cuticle in such cases affords a distinction. Dr Greenhow relates two or three cases of chronic phthisis in which pigmentation was present, some-

what resembling that of Addison's disease. The chloasma of women pregnant or affected with uterine disease, the discolouration of skin produced by malarious and tropical fevers, or by hepatic diseases, and even tinea versicolor, have each been mistaken for the bronzed skin of suprarenal disease. But the most extraordinary instance of perversity is perhaps that afforded ('Virchow's Archiv,' 1870) by a German observer, who met with a case of sclerodermia, in which large patches of the skin in succession became deeply pigmented, sometimes in a single night. The patient, an old woman, had Bright's disease, and died of pneumonia. Her suprarenal capsules were healthy; but her medical attendant, instead of seeing that his idea of the case was altogether wrong, proceeded to base upon it an entirely new theory with regard to Addison's disease, ascribing to it a functional disturbance of the cerebro-spinal system. The truth is that one cannot accept unreservedly the diagnosis of Addison's disease by a physician who has not already seen other cases, and so made himself thoroughly familiar with its characters. Even in a large hospital the disease is not common enough to come under the observation of every student.

Ætiology.—Addison's disease occurs much more often in *males* than in females. According to Dr Greenhow, the proportion is as 119 to 64.

The *age* of the patient is generally between twenty and fifty, but instances have been met with in children of five, eleven, and thirteen years of age.

Dr Greenhow thinks that men engaged in hard bodily labour are especially liable to this disease. He even thinks that its starting-point is sometimes a direct strain or a blow on the back. He mentions seven such cases: one is that of a woman, who constantly asserted that she had never recovered from a strain in the back which had occurred while she was turning a mangle some years before; another patient had always had ill-health from the time when she fell downstairs; a third dated his illness from a fall through a trapdoor.

In none of these instances was there any discoverable affection of the bones or ligaments of the spine. But in many other cases *vertebral disease* has been present. Dr Wilks first drew attention to this fact, and Dr Greenhow mentions eighteen instances. The lower dorsal or upper lumbar vertebræ have generally been the seat of the mischief, and there have always been abscesses in or near the psoæ muscles. Very often a sinus has led from the abscess to the neighbourhood of the adrenals; or it has at least been clear that the disease spread continuously from one structure to the other. In some cases the vertebral disease was directly traceable to injury, in others it appeared to be of tubercular origin. Indeed, even when its immediate cause was a blow or fall, a "scrofulous" tendency most probably combined in its causation.

Nature of the lesion.—This brings us to the important question whether the affection of the suprarenal body is inflammatory or tubercular. Addison spoke of it as "scrofulous," but formerly this name was applied to all caseous lesions. Wilks was inclined to deny that the disease was of a tubercular nature; he maintained that cases in which well-marked tubercles were discovered in other viscera were exceptional. Virchow and Rindfleisch, however, term Addison's disease a tuberculosis of the adrenals. They describe as the earliest stage of the affection one in which nothing but a cluster of grey nodules exists in the medullary substance. The author has observed the same thing, but only in cases of general or at least widely-diffused tubercular disease—in which bronzing of the skin was absent or but doubtfully present;

it would be very difficult to establish any connection between such cases and those of Addison's disease. Nevertheless, the affection is probably of a tuberculous nature. In the first volume of this work (p. 94) we pointed out that tuberculous diseases occur in at least four or five distinct forms, each more or less limited to one particular group of organs, but all of them in individual cases so frequently connected with one another as to show that their essential nature is the same. Now, in cases of Addison's disease, whatever morbid changes are found in the lungs or in other parts of the body bear unmistakeable marks of a tubercular origin. Schüpell's giant-cells are commonly present, and Koch's tubercle bacillus has been recognised by Guttman and Goldenbaum (quoted by Eichhorst).

Pathology.—What relation can be discovered between the morbid change and the symptoms that characterise the disease? The most plausible hypothesis is that the ganglia and branches of the sympathetic nerve, which are so intimately connected with the adrenal bodies, are involved in an inflammatory process, starting from them. Dr Habershon in 1863 dissected out the semilunar ganglia in a typical case, and found that they and their branches of nerve were surrounded on the side on which the suprarenal body was more severely affected by dense fibrous tissue. Similar observations have since been made by other pathologists both in England and abroad. The semilunar ganglia have been found enlarged and reddened; and under the microscope their cells have appeared opaque and granular, and remains of hæmorrhages into their substance have been discovered. The fibres of nerve-trunks embedded in adhesions have also been shown to be in a state of fatty degeneration (Tuckwell, 'Path. Trans.,' 1868). Again, one or two cases have been recorded to prove that the symptoms of Addison's disease may be caused by affections of the semilunar ganglia independently of any primary change in the suprarenal bodies. Sir William Jenner, when President of the Pathological Society, mentioned such an instance as having come under his observation (cf. *infra*, p. 764); and another is reported by Drs Barlow and Coupland ('Path. Trans.,' 1885).

What we know of collapse as a result of a sudden shock to the semilunar ganglia accords with the opinion that the extreme debility of Addison's disease may depend upon chronic changes in these important nervous centres; and the nausea and vomiting are still more readily accounted for.

No such obvious explanation suggests itself of the peculiar pigmentation of the skin. This has been attributed by Jaccoud to irritation of the vaso-motor nerves; but Risel, who has carefully discussed the question, concludes that some blood-change must also occur. The solution of this difficult problem may perhaps involve the principle that the "bronzed skin" of Addison's disease always corresponds with some form of pigmentation that may occur in health under the influence of stimuli, or in one of the dark races of mankind. It is probable that under normal conditions this, like other organic processes, is kept in check by inhibitory trophic nerves; and if in disease their controlling influence is withdrawn, we might expect pigment to be laid down in excess, or at least in quantity altogether disproportionate to the intensity of its exciting cause. Such an explanation involves more than one hypothesis, and it may appear far-fetched; but it is doubtful whether any simpler one will account for the facts.

Many attempts have been made to throw light on the disease by experiments on animals. Extirpation of the adrenals, performed many years ago

in rats by Dr Geo. Harley, failed to lead to death or definite morbid symptoms. But Nothnagel states that by setting up chronic irritation of the adrenals in rabbits he has succeeded in producing permanent pigmentation of the skin.

In an interesting paper published in the 'Lancet' for February 6th, 1886, Dr Churton, of Leeds, after relating a typical case of Addison's disease in a youth of nineteen, and five others of non-specific lesions of the adrenals without symptoms, discusses the possibility of senile atrophy of these organs producing more or less modified clinical effects. But the patient aged seventy-two, whose case he describes, may not impossibly have had the specific anatomical change of his adrenals; and, as he rightly states, these organs may be found perfectly healthy in old people.

Treatment and prognosis. — The treatment of Addison's disease may unfortunately be summed up in a very few words. The vomiting must be combated by appropriate medicines; but these are too often altogether ineffectual. For the debility and prostration tonics and stimulants are obviously indicated; but they likewise generally fail to do any good. The use of iodine (whether internally or as an application to the lumbar regions) might possibly lead to the subsidence of those inflammatory changes which have been shown to occur in the connective tissue round the semilunar ganglia and branches of sympathetic nerve; and if these changes are the cause of the symptoms, such treatment might do good.

Hitherto, no positive proof has been given that recovery from this disease ever takes place. But it is certain that many patients, after having been kept in a hospital for a long time, and perhaps after having been admitted over and over again, have been lost sight of. This might well have happened in the case already mentioned, of a young man in whom Addison's disease was diagnosed in Guy's Hospital five years before the time he died; for the morbid appearances which were found in the adrenals seemed to show that they had been destroyed for a long time.

Other affections of the adrenals.—These organs may be the seat of miliary tubercles in cases of general tuberculosis; or of hæmorrhage; or of cancer, either by extension from the kidney, lumbar glands or vertebræ, or as a secondary deposit;* or of lardaceous transformation; or of embolism; or of secondary pyæmic abscess. Lastly, they may apparently become atrophied without preceding disease. But none of these pathological states are of clinical significance. In a text-book of medicine, Addison's disease is the only lesion of the adrenals which demands a separate place.

Diseases of other abdominal viscera.—One of the greatest helps which morbid anatomy has given to practical medicine is the discovery that diseases do not affect organs and tissues indiscriminately. Acute idiopathic inflammation, suppuration, chronic fibroid inflammation with contraction, tubercle, primary and secondary cancer—each of these pathological processes is modified by the organ affected, and has a special predilection for certain of the tissues, with more or less complete incapacity of affecting others.

Of this rule we have had frequent examples in the study of diseases of the brain, the lungs, the liver, and the kidneys. In the remaining viscera,

* In a remarkable case which occurred in a child under the editor's care, there was primary sarcoma of each adrenal.

the pancreas, spleen, ovary and testis, there are no special diseases like pneumonia or Bright's disease, acute atrophy of the liver or morbus Addisonii, peculiar to the organs and associated with more or less constant clinical symptoms.

Diseases of the Pancreas.—Like the salivary glands which open into the mouth, the important abdominal gland (Bauch-speicheldrüse of the Germans) which so closely resembles them in structure and function, is very little liable to ordinary inflammation, and there is not known to be any zymotic affection of the pancreas answering to that of the parotid and submaxillary glands in mumps.

Wilks and Moxon describe suppuration of the pancreas in the later stages of enteric fever, which we may compare with the parotid buboes mentioned before as sequelæ of that disease (cf. p. 305).

The gland is indurated in cases of chronic heart disease, like the kidneys and the spleen.

Primary cancer of the pancreas exists, but is in reality a rare disease. In this want of adaptability for malignant growths the pancreas resembles other racemose glands, with the striking exception of the mamma (due perhaps, as Dr Creighton has suggested and partly proved, to its functional activity being often interrupted), and differs from the testis. What is usually described as "cancer of the head of the pancreas" begins in most cases in the epithelium of the pancreatic duct (or in that of the common bile-duct or duodenum) and gradually involves the glandular structure. Hence it is almost always accompanied by jaundice, to which enlargement of the liver from secondary growths is subsequent. So that clinically, when there is no reason to suppose the primary cancer to be in the stomach or rectum, malignant disease of the liver with early jaundice may be most probably ascribed to primary cancer of the head of the pancreas.

Often the structure of the new growth is cylindroma or duct-cancer, but sometimes it is of the ordinary glandiform type, as in one exceptional case lately in Guy's Hospital (1887), in which the autopsy showed primary cancer of the splenic end of the pancreas. Like other epithelial structures, the pancreas is not the seat of secondary cancer.

When *lymphoma* or lymphosarcoma affects the pancreas, as is occasionally seen, the new growth probably begins in the small lymph-glands, which are found in the pancreas as well as in the parotid. *Tubercle* appears never to affect the salivary glands or the pancreas.

Calculi have been found obstructing and dilating the duct of Wirsung, like those found in that of Stensen. In one instance numerous calculi were found, of which several are preserved in the museum of Guy's Hospital (1992[50]), and others were analysed by the late Dr Golding Bird and found to consist of phosphates of lime and magnesia, with oxalate of lime but without carbonate.

When from any cause obstruction has occurred to the passage of pancreatic secretion into the duodenum, we should expect, on physiological grounds, that the digestion of fatty matters would be suspended or at least impaired; and in several cases, notably that of a young woman, a patient of Bright's, whose symptoms during life were "emaciation, diarrhœa, and the passage of fatty stools," the pancreatic and biliary ducts were found after death to be occluded by a cancerous growth. But these cases are exceptional.

Certain cases of severe and acute symptoms, leading rapidly and sometimes suddenly to a fatal issue, have been recorded by Klebs and other observers, in which *hæmorrhage* into the pancreas has been the only symptom found after death. In some of these, hæmorrhage could be ascribed to chronic cardiac disease, in others to violence, and in others it was apparently spontaneous (see a case reported in the 'London Medical Record,' 1878, p. 69, from a paper by Dr Hiltz, in the 'Correspondenzblatt f. Schw. Aerzte,' November, 1877). It is supposed that the effused blood (or the injury which caused it) pressed on the solar plexus and produced fatal effects on the heart, as in Sir Astley Cooper's famous case, and in Goltz's 'Klopfversuch.'

As in the kidneys, the testes, and the mammary gland, retention-cysts are occasionally found in the pancreas. We have already seen how these may affect more than one organ at once, most often the liver and kidneys (*supra*, p. 650), sometimes the brain as well (*ibid.*, note), and in one case the editor met with multiple pancreatic cysts in a fatal case of simple cyst of the cerebellum ('Path. Trans.,' vol. xxxvi, p. 17). Their coincidence, however, was probably a mere accident.

DISEASES OF THE SPLEEN are remarkable in being all secondary to, or forming parts of, other morbid processes. It is very rarely the seat of acute suppuration, of chronic fibroid inflammation, or of parenchymatous diffuse splenitis.

Rare instances, however, occur of fatal illness, which is found at the autopsy to have been due to the formation in the spleen of a single abscess, for which no cause can be discovered, and which may reach a very considerable size. A case of this kind was recorded by Bright in the 'Guy's Hospital Reports' for 1838; about half of the substance of the spleen was involved, and there was an opening into the colon. He also gave another case, in which a sloughing abscess communicated with the stomach; but it is perhaps doubtful whether the original seat of the suppuration in this instance was the spleen itself. Dr Caton, of Liverpool, has recently recorded a case which ended favourably ('Brit. Med. Journ.,' 1888, i, 586).

The symptoms of splenic abscess are obscure, consisting chiefly of pain in the upper part of the abdomen, frequent vomiting, rigors, pyrexia, and emaciation. A more or less distinct fulness or induration may also be discovered in the left hypochondrium.

During life it would be impossible to distinguish cases of this kind from others in which suppuration occurs outside the spleen, but immediately in contact with it, and which are probably much more frequent. In the 'Guy's Hospital Reports' for 1874 nine or ten such instances were recorded by the author. In some of them there was a clear history of a blow or a fall having been the starting-point of the disease; in others a chronic ulcer of the stomach seemed to have set up inflammation outside that organ. One of the most remarkable among them was the case of a man aged thirty-seven, whose case has been already mentioned in the section on circumscribed abscess in the peritoneal cavity (*supra*, p. 469).

In 1870 the author was consulted by a gentleman aged fifty-six on account of a rounded, tender, and rather painful swelling, occupying the left side of a very protuberant abdomen. It seemed clearly to be an enlarged spleen, and this was confirmed by his pallid appearance, and by the fact that there was a decided excess of leucocytes in the blood. However, the skin

over the tumour gradually became reddened, hot, and indurated, and at the end of a month Mr Durham made a puncture with a small trocar, and drew off some pus. For some months afterwards there was a discharge of matter from the opening, but at last it closed, and the swelling disappeared. Still no doubt was entertained that the case had been one of abscess of the spleen. But ten months later a second large tumour formed in the right side of the abdomen. This also was opened, and a great quantity of very fœtid pus escaped. The patient was now exceedingly prostrate and ill, but he again recovered, and in 1876 was in excellent health. Probably the real seat of the abscesses was between the layers of muscles in the walls of the abdomen.

Another interesting case was that of a woman who was admitted into Guy's Hospital eleven days after having crossed the Channel on a very rough day: she had been very sea-sick, and had also been thrown out of her berth upon the cabin floor. A large tumour rapidly formed in the epigastrium, and in the left hypochondrium there was a second mass in the position of the spleen. It appeared not improbable that in the first instance blood was effused over the surface of the stomach, and that the process of suppuration was secondary. In three other cases a sub-diaphragmatic abscess perforated the pleura and set up fatal empyema.

In all fevers the spleen is the seat of congestion, and is found after death soft and more or less swollen. In typhus, acute pyæmia and tuberculosis, scarlatina and pneumonia, it is not obviously enlarged, but in enteric fever it is so, and can be often detected by palpation as well as percussion during life (cf. vol. i, p. 172).

The congested and swollen spleen of anthrax has been described in the first volume. As the result of intermittent fever it becomes much larger, perhaps because of the frequency of the attacks, and at last forms the characteristic ague-cake (vol. i, p. 345). Pigmentation of the spleen is part of the result of malaria, which was first observed by Dr Bright (*ibid.*, p. 346).

The passive congestion produced by general systemic venous stasis, particularly in cases of cardiac disease, leads to hardening but not to swelling of the spleen; but that which is due to portal congestion, and particularly to cirrhosis of the liver, leads to its decided enlargement, except when this is mechanically prevented by a capsule thickened from chronic perisplenitis.

Emboli produce the well-known wedge-shaped blocks (*infarctus*), first deep red, then white with a red border, then completely pale, and lastly shrivelling up into a deep scar. When the emboli, however, are septic, suppuration occurs in the spleen. Hence a swollen and tender spleen with pyrexia and a cardiac bruit is a certain sign of ulcerative endocarditis.

Infective abscesses from general pyæmia are also met with in the spleen, but less frequently than in the liver.

Miliary tubercles are extremely common, especially in cases of general acute tuberculosis in children; and caseous tubercles also occur, but they are probably always secondary.

Lardaceous disease is frequent, either as a uniform infiltration, or affecting the Malpighian bodies only, and producing the well-known "sago-spleen." No local effects follow, and no symptoms result.

Gummata are occasionally found, and have to be carefully distinguished from the fibrous scars of old emboli. Deposits of lymphatic new growths

are not infrequent, and will be described in the following chapter under Hodgkin's disease (*infra*, p. 761).

Primary malignant disease of the spleen is unknown, and even secondary nodules, whether of sarcoma or true carcinoma, are extremely rare.

Hydatids also are less common in this viscus than in the lung, kidney, or brain; when present, there have often been echinococci also in the liver.

In children the spleen is large and typically healthy. As age advances it atrophies like other lymphatic organs—following the fate of the thymus, the tonsils, Peyer's patches, and the lymph-glands themselves.

Hypertrophy of the spleen is the only condition of this organ which possesses clinical as distinct from pathological interest. It is almost always associated with anæmia, and often with leuchæmia, when it constitutes part of the remarkable disease to be described in the next chapter (p. 753).

The diseases of the abdominal viscera which remain, the testes and the ovaries, are by the conventional arrangements in use assigned to the departments of surgery and obstetrics respectively. The best account of their morbid anatomy is therefore found in treatises on pathology, and of the corresponding clinical disorders in those on practical surgery and diseases of women. But the student must not forget that the divisions of our art have no counterpart in nature, and that the physician must not neglect the diseases of any viscus.

Of the diseases of the OVARIES, the only ones which come under the scope of a treatise on general medicine are the cystic tumours of the ovarium and the parovarium, which have been already described, so far as is necessary for their diagnosis from encysted ascites and other abdominal tumours; and the functional disturbances of these organs which are sometimes associated with epilepsy, hysteria, and hystero-epilepsy.

The diseases of the TESTIS are acute inflammation from injury, "metastatic" or "sympathetic" swelling from mumps or from renal calculus, epididymitis from gonorrhœa, tubercular (or "strumous") orchitis, syphilitic orchitis with gummata and chronic fibroid atrophy, cystic disease, enchondroma, and medullary cancer.

It is important for the physician to remember that the testes are abdominal organs which have escaped from the general peritoneal cavity, and they should be examined in doubtful cases with the same care as the liver or spleen. The discovery of epididymitis or fungus testis will often throw light on the nature of hæmaturia and purulent pyelitis; while the presence of sarcocele may point to syphilis in other organs, or to malignant disease of the lumbar glands.

DISEASES OF THE BLOOD

CHARACTERISED BY ANÆMIA AND HÆMORRHAGE

ANÆMIA.—*Its general characters—Amount of blood—Number of red corpuscles—Amount of hæmoglobin—Changes in the blood-discs—Anæmic murmurs—Dyspnœa—Fatty degeneration of the heart—Other symptoms—Symptomatic or secondary and idiopathic or essential anæmia.*

LEUCHÆMIA.—*History—Anæmia splenica—Definition and characteristic symptoms—Physical signs of an enlarged spleen—The blood in leuchæmia—The other organs—Ætiology—Diagnosis—Pathology, course and treatment.*

HODGKIN'S DISEASE.—*Anæmia lymphatica—History—Ætiology—Morbid anatomy and pathology—Course, prognosis, and treatment—Primary tuberculosis of the lymph-glands.*

CHLOROSIS.—*Its relation to menstruation—to imperfect development of the heart and vessels—Symptoms—Prognosis—Treatment.*

IDIOPATHIC PERNICIOUS ANÆMIA.—*History—Name—Age and sex—Antecedents—Course and symptoms—Ætiology and diagnosis—Pathology—Prognosis—Treatment.*

SCURVY.—*History—Early symptoms: purpura, spongy gums, hæmorrhagic inflammations—Night-blindness—Course and prognosis—Ætiology—Prophylactic treatment—Pathology—Curative treatment—Diagnosis.*

HÆMOPHILIA.—*History—Inheritance usually in males through females—Course and symptoms—Prognosis—Diagnosis—Pathology—Treatment.*

PURPURA.—*Symptomatic and primary purpura—Ætiology—Characters, events, and treatment—Morbid anatomy—Pathology—Diagnosis.*

THE ancient humoral pathology assigned most diseases, at least in their origin, to dyscrasia or ill-tempering of the four humours of the body. When black bile was found not to exist, and pituita or phlegm was ascertained to be restricted to mucous surfaces, "overflow of the bile" and "impurities of the blood" were still the easy explanations of many obscure symptoms.

Even in recent times, fevers, syphilis, pyæmia, and cancer have been still called blood-diseases. But for this there is not adequate reason. There is no evidence that the blood of a person with malignant disease behaves differently to physical, microscopical, or clinical tests from that of one in health; at the utmost it is the chief channel of infection in cases of sarcoma, as the lymph is the chief channel in epithelial carcinoma. Nor are the specific diseases, pyæmia, syphilis and the infectious fevers, specially disorders of the blood. They affect the whole body. The poisons or microphytes which are their material basis circulate no doubt with the blood, but the circulation is only the carrier and not the primitive seat of the contagion. The spirillum of relapsing fever, the bacillus of anthrax, are the essence of the disease

and no doubt circulate in the blood; but they are found in the solid organs as well and ought to be compared to the mineral which is carried by the blood to the tissues in cases of arsenical poisoning, or to the filariæ which are transported by the same means to various parts of the body.

The only "diseases of the blood"—maladies, that is, of which we cannot trace the seat further back than to the blood—are the following.

(1) Those in which the anatomical elements—the red blood-discs—are diminished in number. Their diminution leads to the condition long recognised as *Anæmia.** Their shape, size, and colour may be also altered as we shall see, and in one remarkable form of disease their scarcity is accompanied by an absolute increase of the colourless corpuscles; the amount of hæmoglobin in each disc as well as in the total blood may also be diminished; and perhaps the amount of albumen and other constituents of the serum may be altered—but the small number of red corpuscles is the constant and demonstrable morbid condition.

Of *plethora*, the assumed opposite condition of too much instead of too little blood, we have no knowledge. Even in anæmia there is no proof that the mass of the blood is diminished; water is rapidly absorbed and makes thin blood, but the vascular system is as full as before. Nor is it ever over-full. If excess of blood is injected into the veins of animals the foreign corpuscles are rapidly destroyed, nor do we find the number of corpuscles or the amount of hæmoglobin increased in any form of disease. What used to be called plethora was only local congestion.

(2) *Hæmorrhagic diseases*—those in which the blood readily escapes from the vessels, without mechanical cause being demonstrable. Their true pathology is still obscure. There is almost always more or less anæmia antecedent as well as consequent to the hæmorrhage; but while in the former group of blood-diseases we shall see that anæmia leads to hæmorrhage as a frequent result, here hæmorrhage is the constant and anæmia the subordinate symptom.

General account of anæmia.—Under various circumstances the tissues of the human body, which normally have a pink or red hue, from the blood circulating through them, become pale as a result of deficiency of blood or of the colouring matter it contains. In this, as in other cases, there is great difficulty in fixing the boundary-line between health and disease. Some persons—nearly all the members of certain families—are naturally pale, and remain so, however favourable the conditions under which they live. Others lose their colour from time to time if they reside in large towns, work at sedentary occupations, or keep late hours; but a rosy complexion returns soon after they go back to the country, under the influence of sunlight, fresh air, and exercise. We may therefore conclude that among those who constantly exhibit pallor of countenance, but who are all their lives confined to dark workshops, or cellars, or mines, some, at least, would assume a very different appearance if they could spend their time in light, well-ventilated rooms, or in the open air. Again, there are others who owe their colourless, sallow appearance to habitual excesses, especially to the practice of onanism, or to premature sexual indulgence.

* Attempts have been made to improve upon this classical word, by substituting *oligæmia* or *spanæmia*. But it is a mere quibble to say that there is not complete absence of blood in anæmia. We say a person is bloodless in English, and the Greek privative is used like corresponding expressions in other languages.

Excessive smoking, and smoking at too early an age, are also important causes of anæmia; though whether it acts more by disturbing the digestive organs or the heart is uncertain. Again, the skin becomes pale as the result of diseases, whether acute or chronic, particularly enteric and other fevers, acute rheumatism, Bright's disease, and the cachexia of syphilis, ague, and plumbism. Another frequent cause is the occurrence of hæmorrhage to a very considerable extent, or repeated at short intervals. Anæmia is characterised by pallor, not only of the skin, but of all visible mucous membranes. The tint varies widely in different cases, and sometimes, instead of being white like marble, it is very decidedly yellowish, like wax. The hands, the fingers, the finger-nails, show the change as distinctly as the countenance, or even more so, for in those persons who have the smaller vessels of the cheeks dilated and varicose there may remain a crimson patch on each side of the face, contrasting strangely with the rest of the complexion. Moreover, the gums and mucous membrane of the lips do not change colour when the cheeks flush with excitement. The tongue, the fauces, the lachrymal carunculæ, are all more or less white and waxy looking. So, again, in the dead body, the amount of blood in the deeper tissues and in the various organs is found to be very deficient; the liver and the kidneys look like wax, while the heart and the great vessels contain only small loose shreds of coagulum and a little thin pink fluid.

Estimate of amount of blood.—The evidence derived from *post-mortem* examinations seems to show that the total volume of the blood is greatly diminished. There may be corresponding wasting of other tissues in the more severe cases of anæmia; but unfortunately it is difficult to obtain clinical proof of this fact, and almost impossible accurately to estimate the degree of deficiency. The only method seems to be that proposed by Quincke, of first counting (by a process presently to be described) the proportion of red discs contained in the patient's blood, then transfusing into his circulation a known quantity of healthy blood, and soon afterwards again counting the red discs in a fresh specimen taken when the new blood and the old may be supposed to have become thoroughly mixed together. A simple formula gives the total volume of blood which was present in the patient's body before the operation. In two cases of "pernicious anæmia" Quincke estimated by this method that the blood formed only 4 or 5 per cent. of the body weight instead of 7 or 8 per cent., which is supposed to be about the normal proportion. Such experiments, however, can rarely be carried out accurately enough to yield results that can be relied on.

Amount of corpuscles.—For practical purposes we may be content to remember that, whatever diminution there may be of the total volume of the blood, it probably corresponds with diminution of the red corpuscles, which can be estimated with comparative facility.

The "numeration of blood-corpuscles" is one of the most important advances in practical medicine that has been made within the last few years.* Originally suggested by Vierordt, it has been simplified by subsequent investigations, and especially by Malassez (1872), Potain, Hayem, and Gowers (1877), until it has now become a very easy matter. The principle is to dilute to a definite extent a measured quantity of blood, and then to count the number of red discs contained in a certain volume of the mixed liquid. The method of employing the *Hæmocytometer* of Gowers is as follows: (1) A small

* The most complete and elaborate series of measurements yet made have been published by Laage of Christiania ('Die Anämie,' 1883).

pipette, holding exactly 995 cubic millimetres, is filled with the diluting solution, which is then poured into a small glass jar or mixing vessel. A good solution, which leaves the corpuscles in a state favourable for observation, consists of sulphate of soda in distilled water, of sp. gr. 1025. (2) The patient's finger is pricked with a lancet, so that a drop of blood escapes without much pressure. Five cubic millimetres of blood are then taken up by a fine capillary tube, graduated for the purpose, and are blown into the diluting fluid in the vessel. If there is a difficulty in getting exactly the right quantity of blood into the tube, the best way is to take up a little more than enough, and then to let the excess escape into a soft cloth. (3) The contents of the mixing-jar are well stirred up with a glass rod. (4) A drop of the mixed liquid is placed in the centre of a cell excavated in a microscopic slide. This cell is exactly one fifth of a millimetre deep, and its floor is ruled in tenth of millimetre squares. The slide rests on a metal slip, to which two springs are attached. (5) A cover-glass is next laid over the cell, in contact with the liquid in it; the springs are brought over the edges of the cover-glass, and keep it in position with a pressure which is always uniform; the slide is placed horizontally on the stage of a microscope, and this is focussed upon the squares in the floor of the cell. (6) In a few minutes the red discs are found to have settled down upon the squares by gravitation. The number in the squares is now counted, and this, multiplied by 10,000, gives the number contained in one cubic millimetre of the blood. The average number in normal blood is believed to be 5,000,000 in males and 4,500,000 in females. It is usual, however, to state the "corpuscular richness" of blood as a decimal fraction of the normal richness, this being taken at 5,000,000 to the cubic millimetre. The decimal figure may be obtained by dividing by 5 the number of corpuscles contained in 10 squares. Thus if the number in 10 squares is 332, the "corpuscular richness" is ·66.

Amount of hæmoglobin.—It is now known that the deficiency of red discs in proportion to *liquor sanguinis* after all affords an incomplete measure of that which is believed to be the fundamental change in the composition of the blood in anæmia, namely, the diminution in the amount of hæmoglobin contained in it. For all practical purposes this may be estimated with sufficient accuracy by an apparatus called a *Hæmochromometer*, for which we are again indebted to Malassez ('Arch. de Phys.,' 1877), and Gowers ('Clinical Transactions,' 1879). It consists of two glass cylinders of equal diameter, which are placed side by side upon a small wooden stand. One of them is closed, having been filled with glycerine jelly, coloured by a mixture of carmine and picrocarmine of ammonia, so that its tint is that of blood diluted with water in the proportion of one part in a hundred. The other cylinder is graduated in such a manner that a space equal to two cubic centimetres has 100 divisions. It is open; and empty, except that a little distilled water is poured into it. Some of the blood, of which the hæmoglobin is to be estimated, is now taken up by a capillary pipette, marked for twenty cubic millimetres; this quantity is carefully measured off, and is ejected from the pipette into the open cylinder, which is quickly shaken so as to secure the admixture of the blood with the water previously contained in it, before coagulation has had time to occur. Distilled water is then added, drop by drop, by means of a pipette-stopper, until the tint of the diluted blood becomes the same as that of the standard in the closed cylinder. The degree of dilution, when this point is reached, indicates the percentage proportion of hæmoglobin in the blood under

examination, as compared with that of normal blood. The best way of observing the tint is to hold the apparatus up between the eye and a window, so that the light passes directly through the cylinder.

The value of hæmoglobin for each red disc may of course be obtained by combining the results yielded by the hæmocytometer and by the hæmochromometer. Thus the blood of an anæmic patient of Dr Gowers contained 60 per cent. of corpuscles, but only 30 per cent. of hæmoglobin; the average value of red discs was of course $\frac{30}{60}$ths, or one half of their normal value. Not unfrequently it falls as low as one third.

It is to be observed that the deficiency of colouring matter in the blood of an anæmic patient is often obvious to the naked eye when the finger is pricked to allow a drop to be taken for investigation. One sees at a glance that it is pale, thin, and watery, exactly as if it had been diluted.

Corpuscular changes.—More or less marked alterations in the microscopical appearance of the red discs of the blood may also be made out in many cases. Thus, the average diameter of a normal corpuscle being 7·5 μ (1 μ = 0·001 mm.), the average diameter in anæmia has been found by Hayem and by Eichhorst to be reduced to 7 μ, 6·5 μ, or even 6 μ.* Moreover, red discs are sometimes present which are far smaller than any that exist in health, their diameter being from 6 μ to 2 μ; these have been termed *microcytes*. Contrasting with them, however, there may be others which are larger than normal, having a diameter of 12 μ; they have been called *megalocytes*. Quincke and others have found red discs presenting curious irregular forms, being oval, elongated, curved, or drawn out into pointed processes. Another change that seems to have been first recognised by Drs Mackern and Davy, then students at Guy's Hospital, is that the hæmoglobin is sometimes separated from the substance of the corpuscle, forming a rounded body, which had been mistaken by previous observers for a nucleus. For these conditions the term *pœkilocytosis* has been needlessly coined. They were at first supposed to be peculiar to a special affection which will be described as idiopathic pernicious anæmia; but they have since been discovered in cases of anæmia secondary to phthisis, to cancer of the stomach, and to chronic Bright's disease.

The most essential character of the blood in anæmia is deficiency in hæmoglobin; and when the "corpuscular richness" of the blood is diminished, this may sometimes be due to the want of an adequate supply of hæmoglobin, rather than to failure of the process by which they are developed. Now it is obvious that there are two ways in which the amount of hæmoglobin may be reduced below the normal standard: (1) it may escape from the vessels by hæmorrhage, or be consumed within the body more rapidly than it can be reproduced; (2) its formation may be defective.

In those cases of anæmia resulting from hæmorrhage, in which the patient quickly regains his colour as soon as the bleeding is arrested, all the formative processes, both chemical and histological, are, we may suppose, in a perfectly normal condition. On the other hand, most pathologists have supposed that when anæmia seems to arise spontaneously, it is entirely due to a defect in these processes. But Quincke has recorded some observations which suggest the possibility that even in such cases there may be an undue destruction of red discs and of the hæmoglobin which they contain. He has

* The thousandth of a millimeter (μ) is often called a micromillimeter, but according to the usage of physical science it is a *micrometer*, and the *millionth* of a millimeter a micromillimeter.

found that in some instances the amount of iron in the liver is from ten to thirty times as great as under normal circumstances, and that there is also an increase of it, but to a less extent, in the kidneys and in the pancreas. The liver-cells present granules, which are supposed by him to consist of an albuminate of iron; they give to the organ a yellow-brown colour which he seems to think characteristic; the addition of sulphide of ammonium turns the tissue of a greenish-black colour; that of ferrocyanide of potassium turns it blue. However, the significance of this discovery remains doubtful. Quincke himself suggested that it may possibly have resulted from the medicinal administration of iron at some former period of the patient's life. A similar deposit of iron has also been found in diabetes and in enteric fever.

Murmurs.—Among the effects of anæmia, one which attracted great attention from the earlier auscultators was the production of abnormal bruits in the heart and in the great vessels. When the stethoscope is lightly laid upon the patient's neck, just above the clavicle, there is often heard a loud and harsh murmur, which is continuous, but may present variations of intensity, corresponding with the cardiac or with the respiratory movements. This venous humming sound was termed by Bouillaud "bruit de diable," the "diable" being a toy common in Paris in 1835, which made a similar noise. According to the theory which refers all murmurs to the formation of a "fluid vein," the *bruit de diable* may be accounted for, if we admit that in anæmia the volume of the blood is diminished. The jugular veins pass through dense cervical fascia. Consequently, when in an anæmic patient the veins in general shrink and adjust themselves to the small quantity of fluid circulating through them, this part remains unaltered in size, and forms a relatively wide space, within which the streams that enter it are thrown into vibration. The explanation is corroborated by the fact that in many healthy persons one can make a *bruit de diable* by pressure with a stethoscope in the neck.

An "anæmic murmur" of another kind is systolic in rhythm, and is heard over the heart and the main arteries. It is usually loudest at the base, and it often seems to be traceable along the pulmonary artery rather than along the aorta. This murmur can be accounted for in the same way as the other. The trunks of the two main arteries are supposed to be unable to retract, in correspondence with the diminished volume of the blood, to the same extent as the orifices through which the blood enters them. Whether an anæmic murmur is ever localised at the apex is uncertain. Writers on auscultation say that this may be the case; but the question is difficult to answer, on account of the doubt which prevails as to the origin and nature of apical bruits when they occur in persons who are florid.

It is important to notice that none of these murmurs are of significance, so far as the diagnosis of anæmia is concerned. But they are of considerable importance, because they would almost certainly be regarded as signs of organic disease of the heart or of the great vessels by anyone uninformed as to their characters. This is especially the case with the basic systolic murmur, which often has a rough harsh quality suggestive of anything rather than a functional origin. And when there are other reasons for suspecting an organic affection of the heart, as, for instance, when the patient has had rheumatic fever, it is often very difficult to determine whether such an affection may not be present, and whether the anæmia may not after all be merely one of its effects instead of being the primary disease.

Dyspnœa.—Considering that the red discs of the blood have the function of carrying oxygen to the tissues, one is not surprised to learn that a disturbance of the respiratory processes is among the most marked effects of anæmia. Dyspnœa is almost always present; even when the patient is at rest the breathing is unduly rapid without his being conscious of it; when he makes any effort he may be seized with the most distressing suffocative paroxysms and with palpitation of the heart. In some cases similar paroxysms come on without apparent cause. Probably the nerve-cells of the respiratory centre, which are known to be stimulated to excessive discharge by blood containing a normal quantity of hæmoglobin when this is imperfectly oxygenated, are affected in precisely the same manner by blood in which the hæmoglobin is greatly diminished in amount, notwithstanding that what there is of it may be saturated with oxygen. In either case the oxygen that reaches the nerve-cells is deficient.

Fatty degeneration.—The reduction in the amount of oxygen supplied to the tissues seems to be the cause of one of the most striking of the morbid appearances which are found in the bodies of those who have died in a state of extreme anæmia, namely, a granular or fatty degeneration of the muscular substance of the heart as well as of the lining membrane of the larger vessels, and of the secreting cells of the gastric glands, the liver, and the kidneys. At one time we supposed at Guy's Hospital that such changes were peculiar to the form of idiopathic anæmia which had been described by Addison, otherwise called pernicious anæmia. But in 1873 the characteristic appearance of the heart was observed in a woman who died of cancer of the breast; in 1874 in one after hæmatemesis from an ulcer of the stomach; in 1877 in a man who had suffered severely from hæmaturia, and also in a woman who had had a bleeding malignant tumour in the neck. Moreover, Perl has experimentally produced the same fatty degeneration in animals by repeated venæsection ('Virch. Arch.,' vol. lix).

The degeneration is not universally distributed, but specially affects the muscular fibres of the columns of the mitral valve and those which lie beneath the endocardium lining the septum and the ventricles generally. It gives rise to the formation of a series of parallel, opaque, yellowish or cream-coloured lines, which run across the direction of the fibres, the so-called "tabby-cat-striation" (vol. i, p. 940). With a microscope the opacity and pallor are seen to be due to the presence of closely aggregated fat-granules and globules, which look black by transmitted light. They are doubtless waste products that have accumulated in consequence of there not being oxygen enough to remove them. The reason why the muscles of the body generally show no similar change is probably that for a long time before death the patient has been resting in bed. It would, however, be interesting to know whether the diaphragm and the intercostal muscles are or are not affected like the heart.

Muscular weakness is one of the most marked effects of anæmia. The patient may be capable of exerting great power in a sudden effort, but he quickly becomes fatigued, and his strength is soon exhausted. He is also incapable of undergoing mental labour; but his nervous centres are often very excitable, being in a condition known as "irritable weakness." Thus there is sometimes exaggerated sensibility to sensory stimuli, such as a bright light or a loud noise. So, again, whereas the sexual appetite is, as a rule, diminished or suspended, it occasionally happens that a morbid erethism is developed, attended with frequent emissions and with inability

to complete the act of coitus. In the female severe anæmia is almost always attended with amenorrhœa, and generally with temporary or permanent sterility; but occasionally menstruation continues, and may even be profuse.

The *pulse* in anæmia is small, soft, and feeble, in proportion to the severity of the case; or it may be imperceptible at the wrist. But when the cause is a sudden loss of blood it generally has a loose, sharp, jerking character, which indicates that the arteries are imperfectly filled, not having yet adjusted their calibre to the diminished volume of the circulating fluid.

The *temperature* of the surface of the body is often low.

LEUCHÆMIA.*—In 1845 Dr Hughes Bennett, of Edinburgh, recorded a case of enlargement of the spleen in which, after death, the blood was found to be full of objects which he regarded as pus-cells and attributed to "suppuration of the blood." A month later Virchow published a similar case; but he perceived that the abnormal blood-elements were identical with the colourless corpuscles now called leucocytes, and proposed to call the affection "leukæmia." In 1846 Dr Fuller, at St George's Hospital, and Dr Walshe, at University College Hospital, demonstrated the same change in the blood of living patients, and the disease has ever since been universally recognised.

The fact that the blood contains a slight excess of leucocytes is not in itself sufficient to characterise a case as one of leuchæmia. Virchow long ago pointed out that they may be slightly increased in numbers under various conditions attended with irritation of lymphatic glands, as well as during pregnancy, and in fevers. He proposed to distinguish all such minor degrees of blood-change under the name of "leucocytosis;" and it has been suggested that the line should be drawn at the point where the proportion of white cells to red discs reaches one in twenty.

Again, an extreme degree of leuchæmia may occur without any appreciable lesion of the spleen. This was ascertained by Virchow as far back as 1847, and was fully recognised by Bennett in his work published somewhat later. In the first volume of his 'Archiv,' Virchow related an instance in which the principal morbid change was in the lymph-glands, which were enormously enlarged throughout the body. Subsequently he described two forms of leuchæmia—the one "splenic," the other "lymphatic"—distinguished

* *Synonym.*—Leucocythæmia.—*Fr.* Leucocythémie.—*Germ.* Leukämie.

It matters little whether, with Virchow, we call the blood "white" or "pale," or with Bennett call it "white-celled;" neither term is literally exact. But the former is shorter, is more generally accepted by pathologists, and has the right of priority. For Virchow gave it ("Weisses Blüt," 'Froriep's Notizen,' November, 1845; "Leukämie," 'Arch. f. Path. Anat.,' Bd. i, S. 563, 1847) some years before Bennett proposed to amend his first name, "suppuration of the blood," by the new one of "Leucocythæmia." ('Leucocythæmia, or white-cell blood, in relation to the physiology and pathology of the lymphatic glandular system,' Edin., 1852.)

Subsequent research discovered, as one would expect, many previously recorded but misunderstood cases. Thus Dr Craigie, after seeing Bennett's case in 1848, remembered a similar one years previously, in which the pathologist John Reid had recognised in the blood "globules of purulent matter and of lymph," and published it in the 'Edin. Med. Journ.,' October, 1845. Donné also, in 1839, had found what he called "globules muqueuses" in the blood of a patient of Barth. Velpeau, as far back as 1827, had recorded a case in which the spleen was enlarged and the blood looked as if it were mixed with pus. Other cases are quoted from Bichat and Andral., with descriptions of the blood as "*sanie grisâtre*," and "*sang comme sanieux*." Piorry had named the same condition "*Hémite*," by which he meant to denote "inflammation of the blood." On the history of the discovery see Virchow ('Gesammelte Abhandlungen,' pp. 149—218) and Bennett ('Prin. and Pract. of Medicine,' pp. 857—890).

by differences in the size of the leucocytes, these being comparatively large and having sometimes more than one nucleus in the splenic form, but in the lymphatic form being small, and having their scanty protoplasm in close contact with a solitary nucleus. This microscopical distinction is still admitted by Mosler and others. But subsequent experience has shown that to speak of a "lymphatic leuchæmia" as comparable with the splenic affection is attended with great practical inconvenience. Virchow himself soon became aware that the change in the blood was often absent when there was a general enlargement of the lymph-glands; and in the 'Krankhaften Geschwülste' he described under the heading of "lympho-sarcoma" cases of this kind, for which other German writers have used the singularly inappropriate name of "Pseudo-leukæmie." We shall presently see that it is impossible to draw a boundary-line between cases of anæmic lymphatica attended with leuchæmia and those in which the proportion of white cells to red discs is nearly or quite normal. Cases now and then occur which can only be described as presenting a combination of splenic leuchæmia with lymphatic anæmia or Hodgkin's disease. A marked example of this was recorded by Dr Frederick Taylor ('Path. Trans.,' vol. xxv, p. 246).

Apart from such exceptional instances, splenic leuchæmia is distinguished by the three following characters:—(1) Enlargement of the spleen is present from the commencement, it is enormous, and consists in a simple overgrowth of the splenic tissues, there being in the organ no scattered white nodules; (2) the excess of leucocytes in the blood is great; (3) an affection of lymphatic glands and of various other organs and tissues, if present, begins much later than that of the spleen, it is comparatively slight in degree, and has scarcely any tendency to assume the form of definite tumour.

Physical diagnosis of the enlarged spleen.—First it may be well to enter into some details with regard to the determination of the position and size of the spleen by percussion. It had been pointed out by von Luschka that the long diameter of the organ inclines downwards and forwards parallel with the lower ribs on the left side, and that its normal extent corresponds very closely with the space from the ninth to the eleventh rib. But about one third of its upper and hinder part is covered by the inferior border of the lung, and is therefore inaccessible to percussion. Consequently, as Weil and others have shown, the arch of splenic dulness, as it can be mapped out upon the surface of the chest, forms a figure which is rounded in front and below, whereas above it is flattened; behind it has no definite limit, and merges insensibly into the dulness produced by the kidney and other structures in the loin. Its length (in the line of the long diameter of the spleen) is usually about three inches; its breadth, in a direction at right angles with the length, is two or two and a half inches; the distance of the lower and anterior extremity of the organ from the edge of the costal cartilages is an inch and a half or two inches. The patient, while percussion is being practised, may either be sitting upright or lying towards the right side upon the right shoulder and the right hip, with the left side raised just sufficiently to make the lateral region of the chest accessible. It is also necessary to mention that the splenic dulness is always rather "superficial" or incomplete in consequence of the comparatively small thickness of the organ. Light percussion is therefore needed for its determination; and in some cases, especially when there is much subcutaneous fat, when the lung is emphysematous, or when the stomach and the colon are much distended, its extent cannot be accurately mapped out.

When the spleen becomes enlarged, its lower and anterior extremity projects more and more downwards and forwards, and soon can be felt below the costal cartilages. It then constitutes an "abdominal tumour." Guttmann states that he has seen three cases in which a spleen of normal size was displaced so as to lie far below its usual seat. In one instance, that of a man aged thirty, the organ was put back and kept in position by a bandage, whereupon the pain for which he had long been treated immediately disappeared. In another patient, a woman aged forty-eight, the dislocation of the spleen began as the result of a violent physical effort. In the third case the diagnosis was verified by extirpation of the organ; recovery after the operation took place in a fortnight.

(1) To return to the special symptoms of leuchæmia, in most cases the first thing noticed by the patient is that his abdomen is becoming larger, that there is a fulness or tumour in the left side, or that he has a dull aching pain there. On examination one generally finds that the spleen is already very large; even at this period its size is seldom less than that which would correspond with the most advanced stage, and with the most extreme degree, of any other disease. Sometimes, indeed, it does not reach below the umbilicus; but in many cases it descends to the level of the iliac crest. As time goes on, it occupies a position which could hardly have been anticipated (see the series of diagrams by the author in the 'Guy's Hospital Reports' for 1869). The vessels at its hilus seem to offer a resistance to its expansion in a straight line, and it therefore follows a curved course, its lower end sweeping across the brim of the pelvis, and even turning upwards when it has reached the right iliac fossa.* So completely may the organ fill the whole abdomen below the navel, that in women it has often been mistaken for an ovarian tumour. Its real nature may be easily distinguished by the sharp edge which crosses the abdomen obliquely from the left lower ribs downwards, and which presents one or more notches. Its surface is almost always smooth and firm. A friction-fremitus can sometimes be felt over it, and with the stethoscope not only a rub but occasionally a blowing systolic murmur is to be heard. At an advanced stage of the disease it may be separated from the parietes by a layer of ascitic fluid, through which the fingers dip before they reach it.

After death the spleen is often found to be fixed to the adjacent parts by numerous adhesions. Its capsule may present large white or yellowish-opaque patches of thickening. Its cut surface is generally smooth, shining, and homogeneous looking; but sometimes it is marked with whitish lines and striæ, due to thickenings of the trabeculæ. Its consistence is often very firm, and its colour may be brownish rather than purple. It not infrequently shows a number of wedge-shaped pale nodules, resembling the blocks produced by embolism. Histologically the only change discoverable is overgrowth of tissue elements exactly like those of which the organ consists in its normal state; but the stroma often has a peculiarly well-marked fibrous character.

The blood.—In splenic leuchæmia this is paler than natural, and may look turbid; indeed, when the excess of leucocytes is very great, it sometimes has a greyish-red colour, resembling a mixture of pus and blood. Charcot's crystals have sometimes been found (p. 297). After death the

* Probably the fold of peritoneum between the splenic flexure of the colon and the parietes, called *sustentaculum* or *trabecula lienis*, may, when well developed, help to direct the spleen forwards.

appearance of coagula in the heart, or in the great vessels, is often peculiar; they are grumous and opaque, and have been likened to solidified pus.

The proportion of leucocytes to red discs varies widely in different cases, and according to the stage of the disease. From the normal ratio, which is not higher than 1 to 300, it may be increased till it reaches 1 in 20, 1 to 10, 5 or 2. In extreme cases the leucocytes may even be the more numerous, as in a case of Sörensen's, in which they were counted, and found to be as 68 to 47 of red discs.

The number of red discs is always much diminished. But it is remarkable that this anæmia is sometimes unattended with obvious pallor of the countenance; as Wilks long ago pointed out, patients, even at an advanced stage of the disease, have often colour in their cheeks and lips, so that, seeing them in bed, one would hardly imagine them to be very ill. But sometimes the skin is yellowish white, like wax; it was so in the only infant we have had in Guy's Hospital with splenic leuchæmia.

Some years ago Mr Golding Bird examined blood from a case of leuchæmia, upon a warm stage; many of the leucocytes were found to be in active movement, but others remained motionless. In 1880 Dr Cavafy read a paper at the Royal Medical and Chirurgical Society upon a case, already far advanced, in which he repeatedly made observations of this kind: the proportion of leucocytes which showed even slight amœboid movements was at first only 12 per cent., at a later period only 6 per cent. He concluded that the greater number of these were dead or dying, and incapable not only of development, but even of emigrating through the walls of the vessels. Other observers have stated that in some cases of this disease many of the leucocytes are obviously in a state of fatty decay. In fact these two characters of immobility and granular degeneration go far to justify the old descriptions of "pus in the blood."

The altered condition of the blood is probably the cause of some of the symptoms of the disease which have yet to be mentioned. By diminishing the amount of oxygen which can be taken up, it helps in producing the *dyspnœa* which is sometimes the chief complaint of the patient. This may be present only when muscular efforts are being made; in extreme cases even the slightest movement is attended with the utmost distress. Perhaps, therefore, there is nothing surprising in an observation made by Pettenkofer and Voit,* according to which, during rest, the quantity of oxygen absorbed and that of carbonic acid given off seem the same as in health. Another cause of dyspnœa is displacement of the diaphragm upwards by the enlarged spleen.

Hæmorrhage.—The over-abundant leucocytes are probably apt to adhere in large numbers to the lining membrane of small vessels, and may accumulate so as to obstruct them after the manner of minute emboli. Moreover, if it be a fact that many of the cells no longer possess their vital properties and are undergoing disintegration, nothing is more likely than that they should set up a morbid change in the walls of the capillaries with which they come into contact, softening them, and rendering them liable to rupture. In this way may be explained a marked symptom of leuchæmia, namely, the tendency to hæmorrhage. Thus epistaxis is very common; it may recur every day, and it sometimes is the direct cause of death. Bleeding may also take place from the intestine, the stomach, the kidneys, the lungs, or the uterus. Moreover, the hæmorrhage after a wound or other

* 'Zeitschft. f. Biol.,' v, 319, 1869. The figures were: O, 790 to 832, CO_2, 265 to 249.

injury is apt to be excessive; even the extraction of a tooth has, in at least one case, led to a fatal result. The statement has been made that the amount of fibrin yielded by the blood in this disease is above the normal, but that instead of coagulating in long elastic filaments, when separated by stirring, it falls in granular fragments. Purpuric spots are frequently seen upon the skin; and after death the surface of the heart may be found ecchymosed. A large quantity of blood is sometimes extravasated among the muscles or behind the peritoneum. Lastly, hæmorrhage into the brain occasionally occurs, with the symptoms of apoplexy.

In the retinæ hæmorrhages are frequently observed, both during life and after death. Dr Gowers, in his article in 'Reynolds' System of Medicine,' says that they are usually small, and most abundant towards the periphery; they often form striæ, following the lines of the nerve-fibres. After a time the blood undergoes conversion into a brownish pigment. The hæmorrhagic patches often have white or yellowish-white centres, and similar spots may be observed without any accompanying extravasations. Dr Gowers has seen the retina affected with diffuse swelling, and its veins distended and tortuous: "leuchæmic retinitis." *

The lympharia.—Several of the organs and tissues are liable to changes in splenic leuchæmia, but few of these changes can be said to have any clinical significance. In about one of every three cases there is enlargement of *lymph-glands*, especially those of the abdomen and of the chest. The increase in their size is considerable; in our records it is often noted that they were twice as large as normal. They may be firm and fleshy, or soft and medullary in character. They appear not to become fused together, nor does a new growth start from them and penetrate into structures adjacent: characters which distinguish them from lympho sarcomata. The *follicles* at the base of the tongue and the *tonsils* may be greatly swollen, and here an inflammatory change may also be present, for in one of our cases the tonsil was found after death to be sloughing. There may also be diffused pharyngitis and stomatitis. The gums may become swollen, ulcerated, or gangrenous. The *intestinal follicles*, solitary and agminated, are sometimes greatly enlarged, and a lymphoid growth is said to extend beyond the limits of the glands and to infiltrate the submucous tissue. Even ulcers may form, which are said to have thickened edges, and to resemble tubercular ulcers.

Among the most remarkable of all the changes in the lymphoid structures of the body are those which occur in the *medulla* of the bones, and which were first described by Neumann. The cancellous tissue acquires a greenish-yellow appearance, exactly like that which is seen in osteomyelitis; and on pressure a puriform juice exudes. Moreover, the adipose tissue or yellow marrow of the shaft of long bones becomes changed into lymphatic (cytogenic or adenoid) tissue like the red marrow of the cancelli, but of the same pale unhealthy colour which is seen in the cancellous tissue itself.

Mosler met with a case in which this leuchæmic affection of the sternum was indicated during life by great tenderness on pressure.

It is a curious circumstance that in some instances wedge-shaped infarctus, exactly like those which are commonly seen in pyæmia, are found in the

* The changes in question resemble those which occur in Bright's disease. Have observers always been sufficiently careful to exclude the possibility of its presence as a complication?

lungs. This was the case in a patient whose spleen was excised by Mr Bryant in 1866, and who only survived the operation three hours. The patches which occupied the back parts of the lungs had gangrenous centres and red borders. Dr Gowers speaks of such infarctus as arising from plugging of the pulmonary capillaries by leucocytes, but even if this mechanical theory be adopted, the relation to pyæmia must be admitted to be very close. The lungs in leuchæmia appear not to exhibit definite nodules of a new growth, such as are seen in Hodgkin's disease. In many instances they are quite healthy.

Of the remaining organs, the *liver* is most frequently diseased in splenic leuchæmia. It not infrequently weighs as much as eight or ten pounds. There is not always any obvious change in the appearance of its cut surface, and Virchow has stated that the enlargement may be due simply to overgrowth of the hepatic cells. But in many instances there are masses of lymphoid growth scattered through the organ, especially in the neighbourhood of the vessels. Sometimes these are visible only with the aid of a lens, sometimes they are apparent as minute, greyish-white granules. Moreover, even within the acini, numerous leucocytes are seen between the hepatic cells; some appear to lie within the capillaries, others are outside them, being supported by a nucleated stroma of their own. It does not appear that these changes ever cause jaundice.

The *kidneys* are less often affected; in them the lesion assumes the form of scattered, greyish-white striæ, running through the cortex, and bearing a close resemblance to those which are seen in cases of ascending nephritis (p. 683). The surface is smooth and marked by ecchymoses. On microscopical examination, beside more or less numerous extravasations of red discs, vast numbers of leucocytes are seen between the tubules. The urine is not infrequently albuminous during life, but sometimes, though not always, this is to be accounted for by the presence of other changes in the kidneys, such as occur in ordinary Bright's disease.

Other symptoms.—Leuchæmic patients often complain of weakness, headache, giddiness, noises in the ears, palpitation of the heart. In many cases *pyrexia* is present from time to time, lasting a few days, and being followed by intervals during which the temperature is normal. Sometimes this pyrexia is accompanied with shivering and sweating, and may reach 103°, or higher still, but usually it is of a mild type, and would not be recognised without the thermometer.

Ætiology.—In the majority of cases no cause for splenic leuchæmia can be discovered, but in a certain number it appears to be a remote sequela of *ague*. Some of the writers who first studied the disease thought that it bore no relation to the marsh poison, because it often occurred in persons who had never been exposed to malaria, and because the affection of the spleen resulting from ague was then regarded as merely congestive. But whenever numerous cases of leuchæmia have been collected, some of the patients are found to have suffered from intermittent fever. Dr Gowers found it so in thirty among 150 cases which he collected, and at Guy's Hospital the proportion has been considerably higher. There is clearly something more than a mere coincidence in these results, but it is remarkable how long the interval has sometimes been, and how mild the attack of ague to which such serious consequences are traced. In nine out of twenty-one cases, a period of from ten to thirty years passed before leuchæmia showed itself by any symptoms. A patient in Guy's Hospital in 1880, a Pole,

had suffered from tertian ague in 1858, during four months in Warsaw, but first noticed an enlargement in the left hypochondrium in 1866.

In women, leuchæmia sometimes is discovered during pregnancy; sometimes it seems to arise out of the weakness resulting from parturition.

Of supposed exciting causes, such as over-fatigue, distress of mind, intestinal catarrh, and injuries, nothing positive is known.

Leuchæmia occurs about twice as often in males as in females. It affects persons of all ages, from infants to men above seventy, but it is most frequent in those who are between twenty and fifty.

Diagnosis.—This offers difficulties only in the early stages, with the exception of certain cases in which the characteristic symptoms fail to develop themselves fully, or which appear to be transitional between it and Hodgkin's disease. Mosler says that it is apt to be taken, in the first instance, for chlorosis. But if a large splenic tumour and a great excess of leucocytes in the blood are at any time present, well-marked indications of both these symptoms may always be discovered before there is pallor. It is otherwise with those cases in which the spleen is only moderately increased in size, and in which the proportion of leucocytes to red discs is but slightly augmented, although perhaps the anæmia is extreme.

Anœmia lienalis v. splenica.—There may be great enlargement of the spleen without leuchæmia. Dr Moxon cites two such instances, one of which occurred to Mr Spencer Wells, the spleen weighing 6 lbs., the other to Mr Squire, the spleen weighing 13 lbs. Griesinger terms this affection *splenic anœmia.* It is certainly rare.

Non-splenic leuchæmia.—Again, there may be an excess of white corpuscles in the blood, while the spleen remains of natural dimensions. If the excess is very great, as in a case related by Dr Goodhart, in the 'Clinical Society's Transactions' for 1877, although the spleen weighed only seventeen ounces after death, one may set down the disease as splenic leuchæmia. There is as yet no proof that a primary lesion of the spleen can lead to an excess of white corpuscles in the blood; but, on the other hand, it is certain that they may be moderately augmented in numbers under various conditions, including not only those enumerated by Virchow, for which he proposed the term "leucocytosis," but also the process of suppuration or development of malignant new growths. Thus Dr Gowers mentions that the blood of a patient recovering from perityphlitis showed as many as 150 leucocytes in a not overcrowded field a few days before a large abscess appeared in the back. In 1875 a patient died in Guy's Hospital of jaundice from a cancerous tumour in the head of the pancreas, attended with suppuration in the liver. The blood was repeatedly examined during the last fourteen days of his life, and was always found to contain a decided excess of leucocytes,—as many as 130 to 150 in a field,—which were in active movement.

Pathology.—The theory of splenic leuchæmia is still very imperfect. Many observers, basing their opinions on the admitted doctrine that some at least of the white corpuscles of the blood are normally derived from the spleen, have imagined that they could account for the phenomena of the disease by supposing that the organ, being enlarged, throws off an excessive number of white corpuscles. But this view ill accords with the fact that the total corpuscles of both kinds in the blood are actually fewer than normal, and that the red discs are greatly diminished in numbers. It seems impossible to escape the conclusion that there is a change in the leucocytes themselves, which prevents them from undergoing conversion into red discs,

as they naturally should. It is obvious that this in itself may be the starting-point in the whole morbid process. If so, one must suppose that the formation of red discs,—at least as much of it as undergoes interruption in splenic leuchæmia,—has its normal seat in the spleen. The lesions in the liver, the bones, and other organs and tissues of the body may, notwithstanding Dr Cavafy's observations, be plausibly ascribed to the infiltration of superfluous leucocytes into their interstices; for there is certainly as yet no proof that these bodies constantly, and from the very first, are so devoid of vitality as to be unable to penetrate the wall of a capillary.

The overgrowth of the spleen itself may be accounted for on the hypothesis that a large number of white corpuscles are retained in its texture; and the occurrence of splenic enlargement without leuchæmia may be attributed to retention in the organ of all the leucocytes that fail to undergo the normal transformation into red discs; while, conversely, the fact that the blood is sometimes loaded with them, the spleen remaining of normal size, may be supposed to depend on their all escaping.

Course and event.—Splenic leuchæmia, as a rule, advances slowly towards a fatal termination. Its duration is commonly from one to three years; in the case of a child, it is much shorter. The prognosis in an individual case must be based upon a careful study of the various symptoms; the extent to which the spleen is enlarged is of much less significance than the degree of anæmia and of breathlessness. As regards the state of the blood, it probably matters much less whether the number of leucocytes is greatly increased than whether that of red discs is greatly diminished.

Death often takes place unexpectedly by some complication, such as hæmorrhage, pleurisy with effusion, chronic peritonitis, phthisis or diarrhœa. Sometimes it is almost sudden, as in a case which occurred at Guy's in 1876, when œdema of the larynx was found at the autopsy.

Treatment.—There is reason to hope that at an early stage the disease may sometimes be arrested by treatment. Mosler relates the case of a boy aged ten, whose spleen was considerably enlarged, and whose blood contained leucocytes in the proportion of one to twenty red discs; he took a drachm and a half of sulphate of quinine in the course of four days, and then ten grains and afterwards six grains daily; he completely recovered. Dr Goodhart, in 1876, stated to the Clinical Society that in the previous two years he had seen six cases, all in children under two years old, with moderate increase of the spleen, and with about ten times the usual proportion of leucocytes in the blood, and that they all got better under treatment, the drugs used being either phosphorus or the iodide of iron or cod-liver oil. Other measures which have been recommended are a cold douche directed upon the left hypochondrium, and the application of a galvanic or even of a faradic current to that part of the body, the positive pole being placed over the tenth rib, the negative over the enlarged spleen; it is said that by either plan of treatment the organ may often be greatly reduced in size.

On the other hand, when a case is already far advanced, little or nothing can be done to check its progress. Quinine is given without any appreciable result, and iron is equally useless. The only medicine which has sometimes appeared to benefit the patient is arsenic. The transfusion of blood into his veins is hardly likely to be of any avail. Excision of the spleen should be rejected on account of the danger of hæmorrhage from rupture of adhesions, as well as of peritonitis.

Leuchæmia lymphatica.—Beside splenic leuchæmia, rare cases occur of the same condition of the blood, the same clinical features, and the same fatal event, without any enlargement of the spleen, but with hypertrophy of the lymph-glands, the marrow or other lymphatic organs. The first variety was described by Virchow, and may be named *Leuchæmic lymphatica;* it is extremely rare. The second (*Leuchæmia myelogenica*) was discovered by Neumann and Bizzozero, and is less infrequent alone; but it is far more often associated with the common (splenic) form of leuchæmia, or with Hodgkin's disease, or with Addison's anæmia (*v. infra*, p. 779). A third variety was observed in an isolated case by Béhier, and named *Leuchémie intestinale*, because it was associated with hypertrophy of Peyer's patches (1868). A similar case is quoted by Virchow as having been observed by Heschl.

HODGKIN'S DISEASE.*—In the chapter on tumours it was remarked, under the head of lymphoma (vol. i, p. 114), that at the bedside, and even in the *post-mortem* room, one is obliged to class together under a single name certain cases in which the lymph-glands, the spleen, and sometimes many other organs and tissues, become the seats of growths which in different instances vary; histologically they may be pure lymphomata, or they may be sarcomata, or they may occupy an intermediate position in the scale, so as to deserve the title of lympho-sarcomata. As a rule, the cases in question present at the bedside a common group of symptoms; namely, a more or less general and sometimes extreme enlargement of the lymphatic glands, a moderate increase in the size of the spleen, with the presence of scattered nodules in it, marked anæmia, and more or less subcutaneous œdema of the face, as well as of other parts, so that the patient's appearance is like that of a person suffering from acute Bright's disease. They want therefore a designation which shall leave their precise pathological anatomy an open question, indeterminable as it is during life, and unessential as it seems to be even after death. Such a designation, and one which is likely to meet with general acceptance, is that of Hodgkin's disease, proposed in 1865 by Dr Wilks, to whom we are mainly indebted for the recognition of the malady in this country, in place of the title "anæmia lymphatica," which he had given it in an earlier paper in the 'Guy's Hosp. Reports' for 1862. He found, in fact, that his own observations had been anticipated by a former lecturer on pathology at his own school, Dr Thomas Hodgkin. In a communication made by that accomplished physician to the Royal Medical and Chirurgical Society in 1832 there are recorded several instances in which the spleen and lymphatic glands were jointly affected; and two at least of them, but more probably four, are examples of the disease now to be described. In Germany, Virchow, Wunderlich, and many others have recognised the condition; and in France Trousseau described it in one of his graphic lectures under the name "Adénie."

Ætiology.—With regard to the causes of Hodgkin's disease but little is known. A very large proportion of those who are affected by it are children or young adults. Thus, of seventeen cases that have ended fatally in Guy's Hospital between 1856 and 1878 all but two have been in persons between eight years and thirty years old at the time of death; of the exceptions one was in a woman aged forty-five, the other was in a man aged fifty-six. A

* *Synonyms.*—Anæmia lymphatica—Multiple adeno-lymphoma, with anæmia or leucocytosis.—*Fr.* Adénie.—*Germ.* Pseudoleukämie.

series of cases collected by Dr Gowers for his article in 'Reynolds' System' also shows a marked immunity in those who are between thirty and fifty; but among them the number of patients between fifty and sixty was remarkably large. At Guy's Hospital there have been fourteen males to three females. Dr Gowers gives the proportion as three to one.

Trousseau thought that there was often a starting-point for the glandular affection in some acute or chronic irritation at the angle of the eye, or in the external ear; and we have had six cases in which there was some evidence of such a local origin. In one boy the swelling began in the neck, and was said to have followed a blow from a cricket ball; a girl had had suppuration of the ear, and indeed died of secondary meningitis; in a young man the development of the disease was preceded by abscess of the cervical glands; in two boys it followed measles, that exanthem having led in one instance to an abscess under the left side of the lower jaw, in the other to a swelling in a similar position which for a time subsided, but afterwards seemed to return. In a young man, in whom it began with enlargement of the glands in the left groin, there had been a chancre six months previously.

Symptoms.—As is implied in the preceding paragraph, there have been many cases in which an overgrowth of lymphatic glands in some particular region on one side of the body has been present for two or three years before there was any sign of extension of the disease to other parts. Dr Gowers alludes to the case of a boy whose axillary glands were excised by Mr C. Heath six years after they first became enlarged, and in whom, four years later, the cervical glands in the same side had alone become affected. And sometimes the morbid change has seemed to spread by continuity, as from the cervical glands to the thoracic, or from the inguinal to the lumbar. But in other instances it has apparently developed itself symmetrically on opposite sides of the body, as in both sets of axillary glands; or it may even have sprung up simultaneously in the most distant parts. One very striking character is that the affected glands, even when they have reached the size of pigeons' eggs, often remain isolated from, and freely moveable upon, one another, and unattached to the skin. They are commonly neither painful nor tender. But after a time they are apt to become fused together, either by a process of periadenitis, or more often by an extension of tumour-growth through their capsules, from one gland to another. They sometimes reach an enormous size; there may be several packets in different regions, each of the size of a child's head. They have been found after death to weigh as much as ten pounds. They are generally firm and elastic to the touch, but they may be very soft. A case of Bonfils' is cited in which an abundant quantity of lymph accumulated in one of the glands, and when a puncture was made, continued to run.

In other instances a general failure of the health precedes the development of external glandular swellings. One cannot help suspecting that the affection has then really begun in some of the deeper glands, which are beyond the reach of manipulation; and this idea seems to be confirmed by a case, recorded by Dr Wilks, of a man who died in Guy's Hospital in 1856 in an extremely debilitated and anæmic state, with an enlarged spleen, and in whom the autopsy showed that the mediastinal and the lumbar glands were very much enlarged, although the superficial glands were unaffected. In such cases a doubtful diagnosis might perhaps sometimes be cleared up by recognising signs of pressure; for example, spasmodic cough and

dyspnœa, distension of the veins, œdema of the arms or of the legs, or pain in the course of the lumbar or sacral nerves.

Dr Gowers some years ago made an autopsy in a case of Sir William Jenner's, in which, there being general glandular enlargement, a mass of growth extended from the abdominal glands, and involved the solar plexus and nerves going to the adrenal bodies, so as to produce a discolouration of the skin having the distribution of Addison's disease, notwithstanding that the adrenals themselves were healthy.

Even glands lying outside the great visceral cavities sometimes interfere seriously with adjacent structures when much enlarged. In the neck they may compress the trachea or the œsophagus, and cause suffocation or a complete inability to swallow food; or, it is said, they may give rise to severe cerebral symptoms by pressing on the great cervical vessels, or to irregularity of the heart's action by involving the pneumogastric nerves. Enlarged glands in the neck may hamper the movements of the lower jaw in mastication. In the armpit they may compress the axillary vessels and nerves, so as to cause much pain in, and swelling of, the arm.

Pallor almost always becomes, sooner or later, a conspicuous symptom in Hodgkin's disease. The cheeks and the lips appear bloodless and waxy looking; the more so, as there is generally considerable subcutaneous œdema, the eyelids being swollen, and the aspect that of a patient recovering from scarlet fever. It must be added, however, that albuminuria has sometimes been present as a complication, and that tubal nephritis has been found after death.

The state of *the blood* is commonly that of simple anæmia. When drawn, it is strikingly pale, and it has been compared to diluted claret; it coagulates slowly and imperfectly. Dr Gowers has found the red discs reduced to 60 per cent. of the normal proportion; in a case that occurred at Guy's Hospital in 1877 they were estimated at 76 per cent. The number of leucocytes is, in the majority of cases, perfectly normal; but sometimes there is a slight increase of them, and in rare instances the excess is considerable, rivalling that of all but the most extreme cases of splenic leuchæmia. At least it is difficult to know what other interpretation can be set upon the various observations cited by Dr Gowers in a footnote to his article; he adduces no fewer than eight observations of extreme leuchæmia with enlarged glands and a spleen of normal size; two in which there was overgrowth of splenic pulp, but in which the organ contained no conspicuous growths; and two in which even this last character was present, the proportion of leucocytes to red discs being, in all the last four cases, that of one to four red discs. It is possible that the occurrence of leuchæmia in these cases should be regarded rather as an accidental circumstance than as an essential feature of the disease; just as a similar state of the blood is sometimes observed as the result of the growth of round-celled sarcomata in various parts of the body. Moreover, splenic leuchæmia and Hodgkin's disease may be associated together in the same patient. Thus in the 'Pathological Transactions' for 1873, a case of Dr Frederick Taylor's is recorded in which the spleen weighed fifty-one ounces, and had quite the appearance usually seen in ordinary cases of leuchæmia, but there were also mediastinal and subpleural lymphomata of remarkable size.

The usual symptoms of severe anæmia accompany Hodgkin's disease. The respirations are accelerated, being often from 24 to 36 in the minute; there is dyspnœa, which on exertion may become extremely distressing.

Epistaxis and purpura sometimes appear, but much less frequently than in splenic leuchæmia. The temperature of the body often rises to 100° or higher; in one instance it reached 103·2°. According to Dr Gowers, the pyrexia is sometimes continuous with slight diurnal fluctuations; sometimes it lasts only for a few days at a time, the febrile periods being separated from one another by intervals during which the temperature is normal; sometimes it is especially characterised by morning remissions, the daily range amounting to 3° or even more. In 1860 a man died in Guy's Hospital nine days after his admission, in whose case no diagnosis was made during life; he lay with his eyes closed, and he was scarcely sensible; his skin was hot, and he had occasional rigors; his spleen could just be felt; there was a mass of enlarged glands in the left side of the neck, and the autopsy showed that it was a case of Hodgkin's disease. A persistent high temperature appears to be a sign which, more than any other, indicates that the patient's life will rapidly be brought to an end.*

The *course* taken by cases of Hodgkin's disease is very variable. Sometimes the patient is known to be ill during only a very short period before the occurrence of a fatal termination; and the lymphatic glands may then undergo a very rapid enlargement. Thus in 1867 a man aged thirty came under the author's care whose case was recorded in the 'Guy's Hospital Reports' for 1881. Except that a month previously he had a slight cough and hæmoptysis (which probably were due to tubercular disease of the lungs, since this was found to be present at the autopsy) he was supposed to be well, and remained at his occupation as a hawker of fish until three weeks before his death. He was then suddenly seized with a dull heavy pain at the chest, and six days later, on March 16th, he became covered with purpuric spots. On the 21st hæmaturia set in. He also expectorated a quantity of blood, which seemed to come from the mouth. He was admitted on March 23rd. The spleen was then much increased in size, its edge being felt about half an inch below the ribs; but no enlargement of lymphatic glands was discovered. During the next few days his temperature ranged from 99·4° to 99·9°. The diagnosis was "purpura hæmorrhagica." On the 28th he had epistaxis. On the morning of the 30th, at about 7 a.m., he noticed, for the first time, that the glands of his neck were enlarged and very tender. He pointed this condition out to the clinical clerk, and it was then found that all the other glands of the body were likewise swollen, although not so tender. Notwithstanding that he was perspiring freely, his temperature was 103°. Extreme dyspnœa set in two days later, on April 1st; and he died, suffocated by œdema of the larynx, at noon on that day. On *post-mortem* examination the cervical and the axillary glands were seen to be enlarged, so that some of them measured an inch in their long diameter; they were soft, of a pinkish cream colour, and spotted with ecchymoses. The tonsils presented a similar appearance, and were half an inch thick. The thymus formed a large pear-shaped mass. The spleen weighed twenty ounces; it was pale and soft. The kidneys were very pale and spotted all over with patches which looked as though they were suppurating. Dr Moxon found distinct evidence of leuchæmia: leucocytes were visible in large numbers in the liver between the hepatic cells, and as many as twenty-five were counted in a single short capillary

* In a case under Professor Bäumler's care, reported from Frieburg i. B., by Dr Brauneck, there was a temperature of 40·5 C. (105 Fahr.). 'Ueber einen Fall von multipler Lymphbildung' (Hodgkin'scher Krankheit), 1886.

vessel of the substance of the heart. Nevertheless, the blood was examined a day or two before the patient's death, without discovering any excess of white corpuscles. Thus the case rather tends to confirm the opinion expressed above that leuchæmia is no essential feature of the disease even when it is present.

Two cases somewhat similar were recorded by Dr Paterson in the 'Edinburgh Medical Journal' for 1870. The first was that of a young woman aged twenty, who having previously been plump and well-coloured, became towards the end of a first pregnancy very sallow, with hollow eyes, although she still said she felt well and in good spirits. Her confinement was followed by troublesome hæmorrhage, which, however, readily yielded to ergot. About the sixth day afterwards a marked change for the worse took place. The pulse became rapid, there was considerable heat of skin, the liver and the spleen were found to be enlarged, and the glands in the neck were also slightly increased in size. The blood was now examined and it proved to be highly leuchæmic. Death occurred only five days later from suffocation, the cervical glands having in the meantime reached a great size, and the dysphagia having been so extreme that she could not swallow even a teaspoonful of water. Dr Paterson's second case occurred in a policeman's wife, who had become languid, pale, and sallow during the latter part of her first pregnancy, and who had considerable hæmorrhage after delivery. Soon afterwards a slight increase in size of the cervical glands were detected, and it was also found that there was leuchæmia, and that the liver and the spleen were enlarged. The glands of the throat and neck and upper part of the chest underwent further increase, fever and restlessness set in, and she died of asphyxia only fourteen days after parturition. There was no autopsy in either case.

As a rule, however, the progress of the disease towards a fatal termination is slow. Dr Gowers gives a table showing the duration of fifty fatal cases, the length of which could be fixed with some degree of accuracy; thirty-three of them ended within two years. The most common mode of death is gradual exhaustion. But, as we have seen, suffocation sometimes occurs from pressure of the enlarged glands upon the trachea; and sometimes starvation, from pressure upon the œsophagus. Epistaxis has occasionally been directly fatal. Coma, delirium, and convulsions, without discoverable anatomical cause, have been observed in some cases by Dr Southey. Pneumonia, œdema of the lungs, and pleurisy are not infrequent complications, and may be the immediate causes of death. Diphtheria of the fauces seems to have occurred more often than can be accounted for on the supposition of a mere coincidence.

Anatomy.—The morbid anatomy of Hodgkin's disease varies in different cases. The affected glands usually appear whitish yellow, waxy, smooth, and firm, both on surface and on section; but sometimes they are opaque, white, soft, medullary, and perhaps spotted with hæmorrhages; and in one case they were of a uniform deep reddish-grey tint. They have remarkably little tendency to caseate.*

The *spleen* is only moderately enlarged, its weight in the cases that have occurred at Guy's Hospital having varied from eight to twenty-eight ounces. On section there are as a rule found scattered through its substance a number of firm whitish-yellow masses, of round or irregular shape, from the size of peas to that of hazel-nuts. Dr Wilks used to compare them to

* On this point see Mosler's paper ('Virch. Arch.,' lvi, p. 14).

masses of suet in a pudding, or to the almonds in "hardbake." Sometimes the nodules present one or more concentric rings. In one instance this affection of the spleen was unconnected with a diffused morbid change in the lymphatic glands. It was the case of a man aged sixty-seven who died of cerebral hæmorrhage, and in whom it was accidentally found at the autopsy, the glands being normal both in size and in appearance. A second example is recorded by the editor in the 'Path. Trans.' for 1870, p. 390, in a girl of seventeen; here there was also diphtheritic angina and colitis. Sometimes the spleen in Hodgkin's disease presents only an indefinite mottling, or its tissue may be uniformly red and homogeneous.

In other organs the growths are very variable in character. The *liver*, which may be greatly increased in size, sometimes contains distinct nodules, but more often it merely shows tracts of lymphoid tissue running along the portal canals, or minute nodules scattered between the lobules in such a way as to be distinctly recognised only with the microscope. Thus, the state of the liver is often undistinguishable from that which occurs in splenic leuchæmia. In a case under the editor's care in 1874, the liver weighed 88 and the spleen 83 oz., without leuchæmia or enlargement of lymph-glands ('Path. Trans.,' xxvi, p. 199).

The *kidneys* present similar combinations of diffuse interstitial growth with more or less sharply defined and rounded tumours.

The solitary *follicles* of the intestine and also Peyer's patches are sometimes greatly swollen and medullary-looking; and it is worthy of notice that the *tonsils* and the follicles at the root of the tongue may be affected in the same way, since their enlargement can be seen during life (see cases by Dr Moxon, 'Path Trans.,' xx, p. 369, and Dr Legg, 'St Barth. Hosp. Rep.,' vol. xi). The "lenticular" lymph-follicles of the stomach are also enlarged in some cases (Virchow, 'Krankh. Geschw.,' p. 509).

Another organ which is accessible to clinical investigation is the *testicle*. In Dr Taylor's case, already referred to, each epididymis was enlarged so as to be two or three times as big as the testicle itself; and a similar condition is mentioned as having been present in one of Hodgkin's original cases. Within the thorax there are sometimes enormous masses of growth. In Dr Taylor's case (where there was leuchæmia, with a very large spleen) the anterior mediastinum contained a flattened tumour, one inch thick, with the left innominate vein running through its centre. In that instance there were also in each parietal pleura large flat nodulated bands of lymphoid growth of red colour, running parallel with the ribs. In other cases the pericardium and the base of the heart have been invaded, or the disease has spread into the lungs from their roots.

Not infrequently the *thymus* has been greatly enlarged and infiltrated with a soft white growth.

The medullary tissue of the *bones* does not always escape. In a case recently examined, one tibia contained a soft rounded mass, as large as a nut, which, however, had not a lymphoid structure, but was made up of a glistening fibrillated matrix with nuclei embedded in it. Writers also describe a diffused change in the medulla, by which it is converted into a reddish-grey, semi-diffluent substance. The change is similar to or identical with that described above (p. 757).

In two cases at Guy's Hospital lardaceous degeneration was found in the viscera as well as in the enlarged glands, but in each case surgical operations had been performed which had led to suppuration.

Diagnosis.—The recognition of Hodgkin's disease is generally easy at an advanced stage; but at the commencement, when the only symptom is a mass of glands in the neck, in an armpit, or in one of the groins, it is impossible to deny that the case may turn out one of "simple lymphoma." And when the glands, instead of being freely moveable one over another, are matted together, one must bear in mind that the affection may perhaps be tuberculous, or syphilitic, or secondary to some deep-seated malignant tumour. On the other hand, the fact that degenerative changes, and even suppuration, should have occurred extensively in the packet of glands first affected, is not always a proof that the subsequent progress of the case may not be that of Hodgkin's disease rather than of a diffused tuberculosis. As regards syphilis, it is perhaps worth while to quote the case of a man who was admitted into Guy's Hospital in 1867, having in the left side of the neck, reaching from the occiput to the shoulder, a tumour which was believed by Dr Moxon to consist of a gummatous enlargement of the lymphatic glands; after death, however, it was found to have its seat in the other tissues of the neck, the glands being embedded in it, but being themselves unaltered. As an instance of secondary sarcoma of lymph-glands, the nature of which was unrecognised during life, may be cited the case of a girl aged ten whose body was examined in 1880, she having died immediately after an operation for the excision of a mass of glands in the left axilla and above the left clavicle. It turned out that there was a primary tumour in the left broad ligament, and that the lumbar glands were also sarcomatous, as well as one of the mediastinal glands, from which the new growth was extending into the right auricle of the heart.

Treatment.—In spite of such cases as these, it is probably well to have such glandular tumours excised as early as possible. Verneuil has recorded one striking case in which an immense mass was removed with perfect success, and the patient was still in good health seven years afterwards.

Of internal medicines, *arsenic* and *phosphorus* have been most recommended as likely to do good. The reputation of arsenic rests mainly upon a case of Billroth's, in which the disease had existed for ten months, the patient being a woman of forty; the cervical, the axillary, and the inguinal glands were greatly enlarged; within a fortnight after the commencement of the treatment they were already reduced in size, and after two months she was discharged with only a single gland of the size of a nut on each side of the neck. No similar success has been obtained by other observers. Injection of liquor arsenicalis into the enlarged glands by a subcutaneons syringe has also been practised at Vienna. The writer once tried it on a patient of his own, but it produced pain and inflammation without apparent benefit. Phosphorus was first given by Verneuil; Dr Gowers in one case found that its administration was followed by a remarkable diminution in the size of the glands, and by improvement of the leucocytosis, but the patient died of disease of the kidneys. It must be borne in mind that the glands have sometimes become much smaller shortly before death, ndependently of treatment.

Progressive diffused tuberculous disease of the lymphatic glands.—In the chapter on tubercle, affections of the lymph-glands were briefly alluded to (vol. i, p. 95). Instances are not very infrequent of nearly all the glands in the body being simultaneously, or in rapid succession, affected with

tubercle, and a fatal illness results. A case in point, which has sometimes been wrongly cited as an example of Hodgkin's disease, was recorded more than a century ago by Morgagni (Epist. lxviii).

No fewer than ten such cases appear in the reports of *post-mortem* examinations at Guy's Hospital during the fifteen years from 1868 to 1882. Two or three of them have already been published. Thus Dr Goodhart related in the 'Guy's Hospital Reports' for 1873 the case of a man who was admitted under Mr Cooper Forster for disease of the right knee-joint, and who also had a large mass of glands in the right posterior triangle of the neck. He died after amputation of the thigh. At the autopsy the glands in Scarpa's angle on the affected side were found to be enlarged, yellow, and of putty-like consistence. The inguinal, the iliac, and the lumbar glands on the right side were all very large, firm and yellow, those on the left side being healthy. Some of the glands in the portal fissure were as big as chestnuts. The bronchial glands on both sides were much enlarged, some being more than an inch long; they were cheesy, and disease seemed to be spreading from them into the lung itself on the left side, in the form of rounded yellow masses. The glands in the right axilla, and on both sides of the neck, were alike affected; and there was miliary tuberculosis of the lungs.

Another case was recorded by the author in vol. xxv of the 'Path. Trans.,' at p. 235. A woman aged thirty-five was admitted with a large suppurating glandular swelling in the left groin, and with another mass of swollen glands in the left side of the neck. She said she had been gradually wasting since her marriage two years before. She died at the end of a week. At the autopsy it was found that along the whole length of the spine there was a continuous mass of enlarged and suppurating glands. The aorta and the vena cava ran through a dense agglomeration of glands, some more than an inch long, many of which contained points of pus, while some were sloughing in the centre. The portal and bronchial glands were similarly affected. The right inguinal and the right iliac glands were in an earlier stage, and looked greyish white and granular on section. The axillary glands were little, if at all, involved. The spleen weighed twenty-four and a half ounces; it contained numerous yellow masses, some as large as walnuts. The lungs showed a few masses of the size of peas, but no ordinary tubercles; nor were there any tubercles in the intestines or the liver.

A third case was that of a man aged twenty-seven, admitted into a surgical ward on account of his having a swelling in one axilla, of the size of a hen's egg, attended with pricking pain; it was growing rapidly, but was moveable, and the skin over it, although red, was not adherent. It was excised, and was found to consist of large caseating and suppurating lymphatic glands. He died subsequently of pyæmia. At the autopsy similarly affected glands extended under the pectoral muscle, and all the mediastinal glands were tubercular; in the spleen there was a single tubercle softening into a cavity.

A somewhat similar case, also from our wards, was published in the 'Path. Trans.,' vol. xxvi, p. 202. The patient was a woman of forty-seven. Of the remaining cases five were in men, aged respectively eighteeen, twenty-four, thirty-five, thirty-seven, and fifty-four years; and one in a woman aged thirty. In every instance, with one exception, it is noted that the spleen contained tubercles.

The duration of this remarkable form of adult tuberculosis was generally

from six months to a year. It was attended with fever and with rapid wasting.

It must be of practical importance to the surgeon to recognise cases of this kind, since he is very apt to be led to excise some prominent mass of glands from the neck or the axilla, a proceeding which is not likely to be successful. To the physician their chief importance lies in the diagnosis from Hodgkin's disease, with which they are generally confounded.

It is interesting to note the great frequency with which tuberculosis of the spleen is met with in association with a similar affection of the lymphatic glands. This fact was long ago pointed out by Bright in vol. iii of the 'Guy's Hospital Reports.'

Chlorosis.*—From the time of Hippocrates this name has been applied to an affection characterised by a pale, sallow colour of the skin—a form of anæmia occurring chiefly, if not exclusively, in young women. Modern writers are apt to ignore any peculiarity of complexion in this as compared with other kinds of anæmia. But although the special tint is not observed in every case, it is characteristic of chlorosis whenever present. The "greenish" olive hue is best seen in those girls who are naturally of a dark complexion; indeed, χλωρός, as descriptive of the pallor of fear or of death, is more applicable to modern Greeks and Spaniards than to the xanthochroic inhabitants of northern Europe.

Ætiology.—At one time chlorosis was supposed to be commonly caused by disappointment in love. This is not the case; but it may be directly excited by a sudden shock, or by violent emotion, as in cases cited by Trousseau. In many instances it bears a close relation to the development of the menstrual function. Thus the age at which it shows itself is almost always between the fourteenth year and the twenty-fourth. It sometimes arises in a girl who has hitherto been robust, with a fresh complexion, and breasts showing no signs of immaturity; but Immermann remarks that such cases are seldom intractable. The catamenial discharge may have occurred for a certain length of time with more or less regularity, and there may be an interval after its cessation before the first signs of chlorosis are observed. Such cases have been supposed to indicate that amenorrhœa is the cause of chlorosis, but there seems to be no doubt that this is an error. Indeed, those who become the subjects of the disease have often been pale and delicate from childhood. Thus Dr Ashwell, in a paper in the first volume of the 'Guy's Hospital Reports' (1836), declared that chlorosis, when it afterwards assumes an aggravated form, has probably always existed from infancy. In many instances menstruation remains absent, and puberty can hardly be said to occur, the axillæ and the mons veneris continue to be devoid of hair, and the uterus may retain throughout adult life the form which is normally peculiar to childhood. Sometimes the catamenial flow is developed prematurely; but Niemeyer states that he never met with a case in which the menses appeared early while the breasts were still undeveloped, without obstinate chlorosis following.

One can readily understand how it happens that a girl in whom the blood-forming organs have never been capable of maintaining a sufficient

* *Synonyms.*—Morbus virgineus, Icterus albus, Leucophlegmasia virginum.—*Fr.* Pâles couleurs, maladie de vierge.—*Germ.* Bleichsucht. The Greek word used by classical writers is not χλωρώσις, but χλωρότης (from χλωρός, green, pale) of which the vernacular term, "green sickness," is the translation.

quantity of circulating fluid to give a healthy tint to the cheeks suffers more than others from the increased drain involved in the establishment of the menstrual function. In the same way we may account for the occasional development of a condition analogous to, if not identical with, chlorosis in pale and delicate boys when the glandular activity of puberty begins.

Virchow has endeavoured to show that in many if not in all of those who become chlorotic at puberty there is, besides a natural deficiency of blood, a congenital hypoplasia or imperfect development of the heart or of the blood-vessels. He finds that the aorta is much smaller, and that its walls are much thinner than in healthy persons of the same age. Thus, he speaks of instances occurring in the bodies of well-developed women, in which the aorta would hardly admit the little finger, whereas it ought to be large enough to receive the thumb; and he cites an observation, made by Rokitansky, of an abdominal aorta that was no bigger than an iliac artery should be, or even a carotid. But, on referring to a brief description of some of Virchow's cases, published in 1872 ('Ueber die Chlorose und Anomalien im Gefässapparate'), we find that in several of them there was stenosis of either the mitral or the aortic orifice. Now, in its whole length, the aorta has been repeatedly found extremely narrow in persons who were the subjects of obstructive disease of valves on the left side of the heart. But (with a few exceptions perhaps in the case of the mitral orifice) such affections are not congenital, but due to endocarditis occurring in childhood. Thus it seems that the hypoplasia of the aorta, instead of being itself a primary defect, is but a secondary result of the valvular lesion. Nor does it seem clear that when Virchow speaks of some chlorotic patients being dwarfed in stature he has been sufficiently careful to exclude the existence of acquired heart disease, which is certainly capable of interfering with the natural growth of the body.

The *symptoms* of chlorosis are in the main identical with those of other forms of anæmia (p. 752). It is worthy of notice that the patient does not grow thin; she may even become fatter than before. There is seldom, if ever, any dropsy beyond slight œdema of the ankles. It does not appear that retinal hæmorrhage has been observed, and certainly no other local bleeding occurs; nor is there menorrhagia, but amenorrhœa.

Beside breathlessness, feeble muscular power, and a rapid irritable pulse, the vascular murmurs of anæmia are seldom absent: a systolic bruit in the pulmonary area (probably dependent on dilatation of the trunk of the artery of the lungs) a systolic murmur in the carotid and other arteries, and a continuous humming sound in the internal jugular vein. This is sometimes better heard on the left, more often on the right side; it is increased in loudness during deep inspiration and has a slight respiratory rhythm even when the patient is breathing quietly; its locality, continuity, and loud harsh quality are very distinctive. This *bruit de diable* (in German *Nonnengeräusch*) is almost exactly imitated by holding a large univalve shell to the ear.

One symptom of anæmia in general is constant and often extreme in chlorosis: a torpid state of the colon. The constipation of chlorotic girls often lasts for many days and sometimes for a fortnight; even if the irritation of scybala occasionally produces what is called diarrhœa, the fæcal accumulation is still unaffected. This symptom, like the amenorrhœa, has been supposed to be the cause of the anæmia; but the first of the triad is certainly pallor.

The *diagnosis* of the disease rests on the age and sex of the patient, the

peculiar tint, the hæmic murmurs, the absence of loss of flesh, and the presence of constipation and amenorrhœa. Bright's disease, mitral disease, phthisis, lead-poisoning, all produce secondary anæmia, which, when it affects young women, may be mistaken for chlorosis.

Practically we have most often to make sure that the case is not one of gastric ulcer, phthisis, or idiopathic anæmia of Addison.

Many girls, apparently only chlorotic, have been unexpectedly attacked by acute and fatal peritonitis, and an autopsy has discovered the cause of both anæmia and death in a perforating ulcer of the stomach.

The early stages of pulmonary disease have been often overlooked in cases of anæmia, and the only way to avoid the mistake is to have the evening temperature frequently taken and to examine the upper lobes of the lungs from time to time.

In 1861 a girl aged eighteen, who was admitted into Guy's Hospital for chlorosis, gradually sank and died; a large scrofulous mass, which seems to have produced no marked symptoms, was found in the cerebellum, and there were a few scattered tubercles in the lungs.

Prognosis.—The disease is as a rule very amenable to treatment, but not always. Some women have to take steel year after year in order to keep free from chlorosis. Indeed, those who have been chlorotic in early womanhood are very liable to a recurrence in later life, especially if they become exhausted by child-bearing or by lactation.

Chlorosis is never directly fatal, but in exceptional cases it seems to be the starting-point of phthisis; hysteria, chorea, gastric ulcer, and exophthalmic goitre are particularly apt to occur in its course; and its presence greatly aggravates the danger of enteric or other fevers.

Treatment.—Chlorosis is happily distinguished from the other forms of anæmia described in this chapter by its amenability to treatment by iron. Splenic and lymphatic anæmia, leuchæmia, and the grave form of idiopathic anæmia to be next described, are seldom benefited, even for a time, by ferruginous medicines. But in most cases of chlorosis sufficient doses of steel act in a "specific" (*i. e.* constant and unexplained) manner. The famous Griffith's mixture (Mist. Ferri Comp.) is deservedly trusted in England, and the scarcely less famous Blaud's pills in Germany.* Mist. Ferri Co. and Pil. Aloes et Myrrhæ go well together. Another excellent combination is sulphate of iron, sulphate of magnesia, and aromatic sulphuric acid. The alkaline preparations are generally most serviceable, but now and then the Tinctura Ferri Perchloridi succeeds better. The rules are to exhibit laxatives along with the preparations of steel, to increase the latter until a decided effect is produced, and if it disagrees, to diminish the dose or change the preparation but not to abandon the drug.

The effect of iron on the number and colour of the blood-discs often appears marvellous, especially when they are watched day by day by means of the hæmocytometer. Thus, in a case of chlorosis recorded by Dr Gowers in the 'Lancet' for 1878, the red discs were at first found to be reduced to 26 per cent. of the normal (1,290,000 per cubic millimetre). Iron was administered, and at the end of a week their numbers rose to 40 per cent. (2,000,000 per cubic millimetre), while twenty-six days later they had reached 76 per cent. (3,800,000 per cubic millimetre).

The fact that iron is so important a constituent of hæmoglobin suggests

* ℞. Ferri sulph. gr. iiss, Pot. carb. gr. iss, Sacch. gr. j, Tragac. gr. ij—iv. Misce, ft. pil. Signetur: "Two, three, and at last four or five to be taken thrice daily."

that it acts directly as a food; but to make this view tenable one must assume that there has been a deficiency of iron in the dietary of those who are benefited by iron; and it is certain that this is not always or generally the case. One must therefore suppose that it stimulates the process by which red discs are developed.

In some cases in which there is much gastric disorder, it may be advisable to give only bland preparations, such as the ammonio-citrate or the potassio-tartrate or the tasteless solution of dialysed iron. But it often happens that the tincture of the perchloride, or the sulphate, can be borne by patients who have a pale flabby tongue, nausea, vomiting, and even pain after food; and such treatment is generally attended with signal success. Large doses seem to be much more serviceable than small. Probably their advantage lies mainly in the fact that only a small part of what is swallowed is absorbed into the blood; the greater portion is converted into a sulphide in the small intestine, and passes away in the blackened fæces.

Idiopathic or Pernicious Anæmia.*—In the year 1855, Addison, in his work on 'Disease of the Suprarenal Capsules,' remarked that the discovery of that disease had been made by him while seeking in vain to find a cause for a remarkable form of fatal anæmia, cases of which had for a long time occasionally come under his observation. To this affection, he added, he had been accustomed in his clinical lectures to apply the term "idiopathic," by way of distinction from such anæmic states as could be traced to "the usual causes or concomitants," among which he enumerated chlorosis. Ever since, it has been recognised by all who have been his colleagues or successors at Guy's Hospital; and allusions to it have been made in successive volumes of our reports and elsewhere, particularly by Wilks in the 'Reports' for 1857, and in his 'Lectures on Pathological Anatomy' (1859). Dr Frederick Taylor published in the volume of our 'Reports' for 1878 no fewer than twenty-three cases which had been recorded year by year since 1853.

We were therefore not a little surprised to find that when Biermer, of Zürich, described the same condition in 1868 under the name of "progressive pernicious anæmia," it was thought at first that a new disease was brought to light, and afterwards that it had been lost sight of by Addison's pupils until it was rediscovered by the Swiss observer.

Even before Addison's work appeared, isolated cases had been published by Coombe in 1823, by Piorry and Marshall Hall, and by Dr Barclay in the 'Medical Times' for 1851. Lebert, who was then at Zürich, wrote on it in 1858 as "essentielle Anämie." Cases recorded in New England by Channing, in 1842, have been discovered by Drs Pepper and Musser ('Philadelphia Med. News,' October, 1882).

The following is Addison's original description of this remarkable disorder, published in 1855:

"For a long period I had from time to time met with a very remarkable form of general anæmia occurring without any discoverable cause whatever, —cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease.

"Accordingly, in speaking of this form in clinical lectures, I, perhaps

* *Synonyms.*—Primary, essential or idiopathic anæmia—Addison's anæmia.—*Fr.* Anémie grave, anémie essentielle.—*Germ.* Progressive perniciöse Anämie—Anæmatosis (Pepper).

with little propriety, applied to it the term 'idiopathic,' to distinguish it from cases in which there existed more or less evidence of some of the usual causes or concomitants of the anæmic state. The disease presented in every instance the same general character, pursued a similar course, and, with scarcely a single exception, was followed, after a variable period, by the same fatal result.

"It occurs in both sexes, generally but not exclusively beyond the middle period of life; and, so far as I at present know, chiefly in persons of a somewhat large and bulky frame, and with a strongly-marked tendency to the formation of fat.

"It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to his earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness on attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless; the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitation being produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles. The debility becomes extreme, the patient can no longer rise from his bed, the mind occasionally wanders, he falls into a prostrate and half torpid state, and at length expires. Nevertheless, to the very last, and after a sickness of perhaps several months' duration, the bulkiness of the general frame and the obesity often present a most striking contrast to the failure and exhaustion observable in every other respect.

"With perhaps a single exception, the disease, in my own experience, resisted all remedial efforts, and sooner or later terminated fatally.

"On examining the bodies of such patients after death, I have failed to discover any organic lesion that could properly or reasonably be assigned as an adequate cause of such serious consequences; nevertheless, from the disease having uniformly occurred in fat people, I was naturally led to entertain a suspicion that some form of fatty degeneration might have a share, at least, in its production, and I may observe, that in the case last examined, the heart had undergone such a change, and that a portion of the semilunar ganglion and solar plexus, on being subjected to microscopic examination, was pronounced by Mr Quekett to have passed into a corresponding condition" (p. 212 in Addison's collected works, republished by the New Sydenham Society in 1868).*

* In the same year Dr Wilks remarked in the 'Guy's Hosp. Reports' (3rd series, vol. i) that "in that class of cases which had specially gained the attention of Dr Addison, and which he has designated idiopathic anæmia," no excess of white corpuscles is found in the blood.

Again, in his 'Pathological Anatomy' (1859), "We occasionally meet with cases of fatal anæmia where no disease is found in the body, &c.," and again, "Those cases of simple anæmia which have been called idiopathic."

Again, in the 'Guy's Hosp. Reports' for 1857 (3rd series, vol. iii, pp. 205—211), and 1859 (p. 108), he puts several cases on record, referring them all to the simple or idiopathic form of anæmia described by Addison. Dr Habershon published a case as "Idiopathic Anæmia" in the 'Lancet' for 1863 (vol. i, p. 518). Trousseau referred to Addison's observations in the first edition of his 'Clinique Médicale,' 1865 (tome iii, p. 533). Dr

A full discussion of the question of priority will be found in the writer's article in the 'Guy's Reports' for 1882 (vol. xlii, p. 236).

Ætiology.—Among the fifteen cases (all in women) described by Biermer in 1871, as also in those previously recorded by Lebert, and afterwards by Quincke and by Gusserow, there were many of severe anæmia in women which were not idiopathic but secondary to pregnancy or parturition, or menorrhagia, or were combined with amenorrhœa as extreme forms of chlorosis. Thus Lebert described his cases as "puerperal chlorosis," and Gusserow his as "hochgradigste Anämie Schwangerer."

In another group of cases of grave or even fatal anæmia the origin of the disease was in protracted diarrhœa, or in gastric ulceration, or (as in some of Immermann's and Quincke's cases from Basel and other parts of Switzerland) in privation and poverty.*

In other cases again the marrow of the bones has been found after death changed in the way described above (p. 757). It is doubtful whether in some cases this may not be a concomitant effect rather than the cause of anæmia. If the latter view is accepted these cases will fall under anæmia myelogenica (pp. 761, 766). But in many cases of fatal idiopathic anæmia the bones have been carefully examined, and no such change has been found.

Lastly, obscure cases of severe and incurable anæmia have been found after death to be due to the presence of *Sclerostoma duodenale* (p. 452), or, as believed, to that of *Bothriocephalus latus* (p. 442).

Instead of correcting their diagnosis, some writers have published these and similar cases as progressive pernicious anæmia due to medullary degeneration, or to gastric disease, or to parasitic worms, and have even hinted that like causes would be found in all instances if looked for.

But Addison's discovery was that patients may die from want of blood which is not preceded by hæmorrhage or exhausting discharges, which do not originate in malaria or starvation, and are not explained by organic or parasitic disease discovered after death. They are not accompanied by excess of leucocytes in the blood, nor by anatomical changes in the spleen, the lymph-glands, or the marrow, and they are not associated with amenorrhœa and the other symptoms of chlorosis. They are not uniformly "pernicious," they are not continuously "progressive," but they are "grave," and, so far as our present knowledge goes "primary," "essential," or "idiopathic."

Frederick Taylor, in the 'Guy's Hosp. Reports,' 3rd series, vol. xxiii; Dr Bramwell, in a paper published in the 'Edin. Med. Journ.' for November, 1877; Dr Stephen Mackenzie in a lecture published in the 'Lancet' in 1878; Dr Coupland, in his 'Gulstonian Lectures' (1881), and the present writer, in a paper published in 'Virchow's Archiv' six years before (Bd. lxv, 1875)—had proved that not only Addison's colleagues and pupils at Guy's Hospital, but also Dr Bristowe, Dr Quain, Dr Sutton, and Dr Howard, in Canada, were perfectly aware of the existence of the remarkable form of anæmia described by Addison. Lépine, in France ('Revue Mensuelle,' January, 1877); Gardner and Osler, in Montreal ('Canada Med. and Surg. Journ.,' March, 1877); and Pepper and Musser, in Philadelphia ('American Journ. of Med. Sc.,' Oct., 1875, and April, 1877), confirm the same judgment. It is surprising that in Professor Immermann's article on the disease in 'Ziemssen's Handbuch,' its recognition is still ascribed to Biermer.

* Dr Bramwell has recorded two cases of foreign sailors, in each of whom "pernicious" anæmia appeared to arise out of an attack of yellow fever. Dr Stephen Mackenzie, in his valuable lecture, cites three instances in which it followed a severe mental shock; one patient had accidentally poisoned her father instead of giving him his medicine, another had seen a child run over in the street, and the third had been attacked by a sheep in a field, immediately before the anæmia set in. Dr Wilks has quoted similar cases ('Brit. Med. Journ.,' Nov. 28th, 1884). So also Lépine, Coupland, and Musser.

No doubt they have their true causes; no diseases are idiopathic in the sense of being spontaneous. But their causes are not those which are known to produce anæmia; they are at present as unknown as those of idiopathic epilepsy or osteo-arthritis. No doubt some cases of apparently secondary anæmia or of chlorosis go on from bad to worse, uninfluenced by treatment, and at last develop the characteristic symptoms of the deepest and gravest anæmia, just as plumbism or cerebral tumours may produce typical epileptic fits, or as frequently repeated rheumatism or gout or gonorrhœal synovitis may produce at last, more or less completely, the characteristic lesions of osteo-arthritis. But the important clinical fact remains, that patients may die of extreme anæmia without any discoverable cause; while in the symptoms during life, in the distribution as to age and sex, and in the *post-mortem* appearances, these cases resemble one another and form a natural group.

Age and sex.—Idiopathic anæmia differs widely from chlorosis as regards the age and sex of those whom it affects. Among twenty-eight cases that occurred at Guy's Hospital up to the end of the year 1879, sixteen were in males, twelve in females. Two patients only were under twenty, both being boys; 4 occurred between twenty-one and thirty; 8 between thirty-one and forty; 5 between forty-one and fifty; 8 between fifty-one and sixty; and 1 at sixty-eight.

In the table published in the 'Guy's Hospital Reports' for 1882 (vol. xli, pp. 293—303), of 102 collected cases of Addison's anæmia, all adequately examined during life and all verified by autopsy after death, there were 6 between seven and fifteen years of age, 4 between fifteen and twenty, 29 between twenty-one and thirty, 26 between thirty-one and forty, 21 between forty-one and fifty, 13 between fifty-one and sixty, and 4 between sixty and sixty-nine. Thirty-six cases since collected from American sources by Dr Musser agree very closely as to the ages of the patients.

In the tabular statements of Heinrich Müller, Eichhorst, and some other observers, there is marked excess in number of female patients over male. Of 44 recorded at Zürich, only 9 were men. But this depends on including secondary anæmia, due to parturition, pregnancy, or lactation, cases of chlorosis, and cases which recovered or which were unverified by *post-mortem* examination. If from Eichhorst's list of only 30 men to 65 women we exclude all but primary cases, the numbers are 12 to 11. Among 107 cases collected by the writer, there were 48 in men and 59 in women. In Dr Coupland's 110 cases (some identical with the last) the figures were 56 to 54, and in Dr. Musser's 39 they were 24 to 15. So that there is either no sexual disposition to the disease, or the disposing factors in each sex are counterbalanced.

Idiopathic and fatal anæmia was at one time supposed to be particularly common among peasant women in Switzerland, but that probably depended on compilers including uterine and malarial cases. It is occasionally seen in its typical form in France, Germany, and America, as well as in Great Britain, in fact wherever there is a large population and competent observers.

Symptoms.—Almost always, the account which is given by a person suffering under this disease is that for some weeks or months he has been getting progressively paler, weaker, and more breathless.

The anæmia is extreme. But what is remarkable is that the complexion, instead of being white, is in many instances of a clear lemon-yellow tint, so that the disease has been mistaken for jaundice. If, however, the

conjunctivæ sometimes show a similar colour, this depends on the presence of unusually yellow adipose tissue beneath the mucous membrane.

As a rule there is no deficiency of flesh about the body generally, but, in exceptional cases, marked wasting has been seen. Most patients have a bad appetite, but some continue to eat well. Dryness of the mouth and of the throat is often complained of; the breath has been sometimes fœtid; nausea and vomiting are frequently present, especially in the morning; and sometimes there is more or less severe pain after food; the bowels have been constipated in some cases, relaxed in others. The pulse is rapid and jerking. Pulmonary and jugular murmurs are common.

There is always marked dyspnœa on exertion, and often palpitation. One man who came under treatment at Guy's Hospital said that every effort caused pain at the back of the head.

Many patients have epistaxis again and again; in women sanguineous vaginal discharges frequently occur; in some persons the gums bleed, or purpuric spots appear on the legs.

The *urine* is high coloured, but otherwise normal; urea is deficient. The ankles commonly become more or less œdematous, and sometimes the face is puffy. In the cases observed at Guy's Hospital there has been no considerable ascites, nor have the pericardium and the pleuræ contained more than four ounces of serum after death.

With the ophthalmoscope *retinal hæmorrhages* can often be detected. Biermer seems to have been the first to notice them. They appear either as linear striæ, or as rounded spots or patches, which may have whitish or yellowish centres, sometimes consisting (according to Manz) of accumulations of leucocytes. The optic discs are said to be swollen in some cases, their vessels to be tortuous, and the retinæ generally to have a peculiar smoky appearance. A boy aged ten who came under Dr Mackenzie's care, had well-marked optic neuritis. As a rule, there is no defect of vision; but the reason why one of Immermann's patients went to the hospital was because one eye had suddenly become blind.

Another characteristic symptom of Addison's anæmia is irregular *pyrexia*. It was first observed by Immermann, and it has repeatedly been observed at Guy's Hospital. Its course is uncertain, the thermometer sometimes rises to 104° or even higher, and after a few days of high temperature there is perhaps a more or less prolonged interval of apyrexia. As a rule, there are no subjective symptoms of fever. In some cases the temperature rises at an early period of the disease, but usually not until an advanced stage. Before death the thermometer often falls to 97°, or even to 95°.

The red discs of the blood are very often found altered in size and in form (*pœcilo-cytosis* of Quincke and Eichhorst), or with their hæmoglobin separated from their substance; but such changes are not peculiar to this form of anæmia (cf. p. 750). In some cases no abnormal appearances can be detected in the blood with the microscope.

The number of corpuscles is often diminished to 1,100,000 per c.c.—very unusual even in extreme chlorosis, where 1,300,000 is the lowest number the writer has seen. It may reach 750,000, 428,000, or (as in a case of Lépine's) 378,750. In a case under Worm Müller, of Christiania, reported by Laache, it fell to 360,000.

Some blood-discs are pale and large (8 or 9 instead of 7 or 7·5, or even 14 μ in diameter), others are small and deeply coloured (microcytes), as first noticed by Dr Leared, in 1858.

Diagnosis.—This depends in part upon our definition of the disease and on the rigour with which we apply the terms of our definition. Moreover, it cannot be certain until verified by an autopsy. But when the following characters are present, we may with considerable confidence make the diagnosis of idiopathic or essential anæmia: (1) Absence of organic disease and of any sufficient cause of anæmia,* particularly of amenorrhœa, menorrhagia, and pregnancy; (2) severe and ingravescent anæmia, with considerable diminution in number (to one half and under) of the red blood-discs, together with the presence of pale large and dark small corpuscles; (3) absence of emaciation; (4) occasional pyrexia; (5) hæmorrhages, and especially retinal hæmorrhage.

The diagnosis of primary pernicious anæmia has been made in cases which have afterwards proved to be secondary to internal cancer, ulcer of the stomach, phthisis, mitral disease, or morbus Brightii.

The likeness to *morbus Addisonii* remarked by Bristowe, Broadbent, Pepper, and other writers, is less frequent and less close than has been supposed. In the latter disease the patient is pale, but not excessively anæmic, the blood is normal or nearly so, and there is decided emaciation. The bronzed colour of the skin is in strong contrast with the clear yellowish pallor of Addison's anæmia; retinal hæmorrhage and pyrexia are absent, and there is frequent complication of phthisis or caries of the spine.

The distinctions from *chlorosis* have been already pointed out. Apart from the age and sex of the patient, the absence of hæmorrhage and pyrexia and the good effect of martial preparations distinguish all but very exceptional cases of chlorosis.

Experience shows that there is danger of mistaking essential anæmia for *jaundice*. In 1877 a man aged thirty eight was in the clinical ward of Guy's Hospital for seventeen days with what was believed to be either acute yellow atrophy of the liver or cirrhosis. He became delirious and violent, and had to be placed in a separate room; afterwards he was insensible, but his alarming symptoms ultimately subsided and he was discharged. However, three months later he returned to the hospital, and it was then obvious that his disease was Addison's anæmia. He sank and died, and the liver was found to be healthy. Other instances of the same kind, and scarcely less striking, have occurred.

In the year 1866 a man aged sixty-seven lay for a long time in the clinical ward of Guy's Hospital with what was believed to be the idiopathic anæmia of Addison, but at the autopsy it was found that the lungs were full of miliary tubercles, and that the liver, the spleen, and the kidneys also contained them in smaller numbers. In 1879 a woman died aged fifty-nine, whose skin had been of a lemon-yellow colour, and whose blood had been found to contain red discs of irregular form, so that the diagnosis was supposed to be beyond

* It must not be imagined that to determine the absence of proceeding loss of blood is always easy. From motives of delicacy persons will sometimes conceal the fact that there has been draining from hæmorrhoids, or from the uterus. Moreover, serious bleeding may occur per anum without the patient's being aware of it. In 1880 I was called to see, with Mr Earle, of Brentwood, a young lady who had fallen into a state of the most alarming prostration and bloodlessness. She had no doubt been ailing for some time, but she had been able to play lawn tennis until two days before my visit. She positively assured us that she had noticed no hæmorrhage from any of the mucous surfaces, and some days passed before her mother made the discovery that the evacuations from the bowel contained blood. Ultimately an ulcer was detected in the rectum. I am inclined to think that the suddenness of the girl's illness would almost have justified one in asserting that there must be some such cause for it.—C. H. F.

question. However, the heart weighed twenty ounces, the minute arteries in the pia mater were greatly thickened, and the kidneys, although large, were hard and glistening, and showed an excess of fibrous tissue under the microscope. It was a case of chronic Bright's disease.

When there is a doubt as to the possible presence of the *anchylostomum* in a case of anæmia, it is important to examine the fæces of the patient with a microscope, since the ova of the parasite can be recognised in them without difficulty.

The nearest allies of idiopathic anæmia are the various forms of anæmia lymphatica, particularly that in which the marrow of the bones is alone affected (anæmia myelogenica). Indeed, although the distinction between the two is well ascertained, the diagnosis must in some cases depend upon the result of inspection after death.

Morbid anatomy.—The only changes found *post mortem* are the result, not the causes, of the profound anæmia. The blood is pale, thin, and fluid, "like washings of muscle." Hæmorrhages are found in the retina and various internal organs. The fat is of a deep yellow tint, and the muscles of a remarkably dark hue. There is more or less effusion of dark-coloured serum in the great cavities. The heart is in a state of fatty degeneration (vol. i, p. 940), occasionally the diaphragm and other muscles, and sometimes the liver and kidneys also. The gastric glands have been found atrophied by Dr Austin Flint, Ponfick, Dr Bedford Fenwick ('Lancet,' 1877), and other pathologists. But this condition is not constant; it occurs without marked anæmia, and it would, if an efficient cause of disease, lead, one would suppose, to wasting rather than pallor. Degeneration of the semilunar ganglia was described by Queckett in one of Addison's original cases, and has since been observed by Eichhorst, Brigidi and others; but it is more frequently absent.

Course and prognosis.—The course of the disease is not uniformly "progressive." On the contrary, there are often decided pauses, and even apparent improvement. The duration is commonly from three months to a year. In 15 cases it is given as from five to eight weeks. In two cases which occurred at Guy's Hospital the disease is said to have run on for three years, and one patient stated that for seven and a half years he had been getting paler and weaker. In ten it lasted between eighteen months and two years.

According to Immermann the fatal termination is sometimes sudden from syncope; but usually it approaches very slowly, there being delirium, apathy, or (*coma-vigil*) complete insensibility for two or three days before death, while sometimes a cadaveric odour has been exhaled from the body.

As to the possibility of recovery from the disease there is good reason to believe that several cases have permanently recovered under, and we may hope as the result of, treatment. Addison himself met with one such case. Other writers have recorded a few, and twenty authentic cases are collected in the 'Guy's Hosp. Reports' (vol. xli, p. 304).

Treatment—In idiopathic anæmia the preparations of iron have proved to be altogether ineffectual. Too often no medicine can be found which will check its downward progress. *Arsenic*, however, has sometimes been very successful in cases which appeared to be of this nature. Dr Bramwell has recorded two such instances ('Edin. Med. Journ.,' November, 1877); one is that of a man aged thirty-eight, admitted to hospital for extreme

anæmia, with retinal hæmorrhages; he had been ill seven months, and became much worse under the administration of the tincture of iron, which was continued for a fortnight, but he completely recovered in the course of four months, while taking the liquor arsenicalis in doses gradually increased from two to twelve minims. Dr Finny had two successful cases ('Brit. Med. Journ.,' Jan. 3rd, 1880). In another, the patient, a man aged fifty-three, showed every sign of extreme anæmia, including retinal hæmorrhage. He was first seen by the editor in March, 1880, when he had been six months ill. He recovered under full doses of arsenic, and remained well in February, 1882, and again when seen in 1887.

Phosphorus has sometimes been thought to be useful. The editor had under his care a patient who regained his appetite for a short time under the exhibition of phosphorus in doses of one twentieth of a grain, and became able to get downstairs; however, he afterwards relapsed and died. In other cases it has completely failed, and perhaps has sometimes done harm.

It very seldom happens that the *transfusion of blood* can be employed with advantage in cases of anæmia that come under the care of the physician. Human blood only should be employed, and should be defibrinated, by stirring, before it is injected into the patient's circulation, on account of the danger of fatal embolism when it is allowed to retain its power of coagulating. That the red discs retain their structure after having been transferred from one person to another seems to be probable from the fact that no change in the urine is observed after the operation, whereas when lamb's blood is used, except in very small quantities, an invariable result seems to be the escape, by the kidneys, of hæmoglobin which has been set free by the disintegration of the foreign blood-corpuscles.

A sense of oppression, or of suffocative distress, is sometimes complained of while the operation is in progress; but this may to a great extent be obviated if the syringe is used slowly and gently. The largest quantity that should be injected at once is half a pint; in many cases from four to six ounces suffice.

Transfusion in extreme cases of anæmia has proved unsuccessful in the hands of Gusserow, Quincke, Bradbury, and Bramwell. It was practised in a case published by the writer in the 'Guy's Hospital Reports' for 1883 (p. 231) with only temporary benefit. But Quincke had one successful case, in which arterial transfusion was practised.*

The following diseases differ from the preceding group in that anæmia is a secondary condition, and hæmorrhage the primary and more important symptom.

* This remarkable operation was repeatedly tried on animals (1665) by Lower and Sir Christopher Wren, and afterwards on human beings (1667) by Denis and Emmerez at Paris, where the operation had been described nine years previously. The Dutch anatomist, Regner de Graaf, devised apparatus for the purpose. It then fell into disrepute from the many fatal results which followed, but it was revived by Dr Blundell 150 years later ('Med.-Chir. Trans.,' vol. ix; and 'Guy's Hosp. Rep.,' 1837, vol. ii, p. 256), and during the last twenty years has been extensively practised. Its history will be found detailed by Mr C. E. Jennings, together with a description of the form of instrument which appears to be most practically useful, in his book on the subject ('Transfusion of Blood and Saline Fluids,' 3rd edition, 1888). See also the papers by Dr Aveling ('Trans. Obstet. Soc.,' 1864) and Dr Braxton Hicks ('Guy's Hosp. Rep.,' 1869), and the Report by Professor Schäfer to the Obstetrical Society (vol. xxi, 1880).

SCURVY.*—*History.*—It is impossible to doubt, from what we know of the cause of scurvy, that men must have been liable to it ever since they began to live in masses under artificial conditions. But no positive proof of this can be found in the medical writings of antiquity, nor until the time of the Crusades. Towards the end of the fifteenth, and in the sixteenth centuries, this complaint became conspicuous from the ravages it committed among those who took part in the long sea-voyages which were then undertaken to the East round the Cape of Good Hope, and afterwards to America. But it was soon found that, beside the "sea-scurvy," there was a precisely similar "land-scurvy," which arose from time to time among the inhabitants of besieged towns, in the inmates of prisons and asylums, and among the poorer classes generally, when exposed to privation or famine. Unfortunately the subject was thrown into complete confusion by the publication of a work, 'De Scorbuto,' by Severus Eugalenus (1588), in which the symptoms and effects of almost every disease were jumbled up together. He seems to have had many followers; and one of the results of their teachings still remains in the popular use of the word "scurvy" as a name for eczematous and other eruptions, and in the traditional practice, in country districts of England, of calling obstinate chronic sores upon the leg "scorbutic ulcers." Indeed, much of the voluminous medical literature of the seventeenth century shows so complete an ignorance of the real characters of the disease that Hirsch ingeniously argues that it cannot have been common. A reaction, however, at length occurred, with Sydenham for one of its leaders; and in 1753 Dr James Lind, Physician to His Majesty's Royal Hospital at Haslar, gave an admirable description of scurvy, which has been followed by all later writers.

Onset and early symptoms.—Usually, but not always, the more definite symptoms of the disease are preceded by a general failure of health and strength. The face becomes pale and sallow, with a livid discolouration of the lips and cheeks. So characteristic is the patient's appearance, that its cause may often be known at a glance. When several are attacked at the same time, each is struck with the altered aspect of the others. The skin is dry and scurfy, and its hair-follicles are prominent and rough to the touch, as in the condition known as "goose-skin," and in *pityriasis tabescentium.* The muscles waste and become soft and flabby. The spirits are depressed and gloomy, the mind apathetic and indifferent, while great lassitude, a sense of fatigue and shortness of breath are felt after exertion. The patient is very sensitive to cold; and pains, which are commonly spoken of as rheumatic, are experienced in different parts of the body, especially in the loins and in the calves of the legs. These pains are worse after exertion, and are relieved by rest and sleep.

Hæmorrhage.—A week or two later an eruption is seen upon the skin, generally first over the lower limbs, but afterwards on the arms and on the trunk; rarely upon the head or face. It consists of reddish or purple spots, chiefly of small size, and presenting the peculiarity that most of them have a hair or a hair-follicle in the centre. The projecting state of the follicles may cause these spots to be slightly raised above the surface. In severe cases there may also be vesicles or bullæ, containing a sanguineous fluid,

* *Synonym.*—Scorbutus.—*Fr.* Le scorbut.—*Germ.* Scharbock or Schormund.—*Dutch.* Scheurbuik. The name is said by Immermann to occur for the first time in the Botanologicon of Euricius Cordus (1534) in the German form "Scharbock," derived from a Danish word, *Skörbeck,* signifying "disease of the mouth." *Scorbutus* is only a Latinised form of the same.

which presently dries up into crusts, while their bases often ulcerate. Much more common are larger subcutaneous extravasations of blood (*vibices*), the edges of which are ill-defined, fading off with varied tints; these sometimes break down, and form large ulcers, with spongy floor, exuding a thin, bloody, putrid fluid. One or more of the nails may be detached from its bed by effused blood, and cast off by an ulcerative process at its root.

Another and characteristic symptom is the formation of ill-defined brawny indurations in the connective tissue, especially of the hams, but also behind the ankles, along the back of the thighs, over the recti abdominis, or in the armpits. The skin over them may be free from discolouration, but they nevertheless consist of extravasated clotted blood, mixed perhaps with gelatinous inflammatory exudation. They are sometimes very rapidly developed, and may be hot and painful.

Again, blood is often poured out into the substance of the muscles, forming more or less obvious swellings, and rendering their contractions painful and difficult; or it may detach periosteum from the bones over a more or less extensive area, most frequently along the front of the tibia, where an enlargement results which has sometimes been mistaken for a syphilitic node. A similar change may also affect a rib, or the scapula, or one of the jawbones, and may sometimes lead to superficial necrosis and exfoliation. In yet other instances extravasation takes place along the epiphysial lines of growing bones, and causes their separation; or, if there is a recent fracture which has undergone repair, the callus may soften down, and the broken ends may again become loose. Or effusion of blood may take place into the joints, especially the knees and ankles.

But the most remarkable affection of all, and one which is scarcely ever absent, is that of the gums. Their edges become bluish red, spongy, and detached from the teeth, with which they should be closely in contact. They are also painful and tender, and they bleed at the slightest touch. They may even become black, and so greatly swollen as to rise above the level of the teeth or even to protrude from between the lips. This change is most marked opposite the incisors, but it may also extend round towards the molars. When there is a gap among the teeth the corresponding part of the gum remains healthy. In young infants, as well as in toothless old people, the gingival affection is said to be altogether wanting. M. Fauvel, at the Salpêtrière, had in 1847 a case in an old woman in whom a single remaining tooth was surrounded by a mass of swollen gum; the tooth was extracted, and the gum soon became level and firmer. When severe, a scorbutic state of the gums renders mastication and the ingestion of solid food an impossibility. There is a horrible fœtor of the breath. The teeth become loose and may fall out. Very commonly a greyish diphtheritic layer forms upon the surface of the affected parts, and the gums may slough, so as to expose the alveolar processes. It is a curious circumstance that the mucous membrane of the rest of the mouth remains perfectly healthy, or at most is slightly livid and puffy. The tongue is swollen and marked by the teeth at its edges. MM. Lasègue and Legroux, however, speak of having frequently seen ecchymoses on the palate during the epidemic which accompanied the siege of Paris in 1871.

Hæmorrhages from mucous membranes are of frequent occurrence in scurvy, epistaxis being most common. Bleeding may also take place from the stomach and intestines. In many epidemics dysenteric symptoms are present, but this appears to be due to a coincidence of the two diseases.

Blood seems not to be often expectorated from the lungs, except when gangrenous pneumonia sets in as a complication, as is sometimes the case. More frequently the pericardium, or one of the pleural cavities, or both together, are attacked with an inflammatory process, attended with abundant escape of blood as well as with the usual inflammatory exudation.

The face does not often present purpuric spots in scurvy, but the skin round one or both of the eyes sometimes becomes puffed out into purple swellings, while the conjunctiva covering the eyeball assumes a brilliant red colour and projects above the level of the cornea. Dr Buzzard, in 'Reynolds' System,' says that in many cases seen by him during the Crimean War, this condition (which was not inflammatory, being attended with neither pain nor discharge) constituted the chief symptom of the disease, and that they were usually severe cases and often ended fatally. Sometimes hæmorrhage takes place in the globe of the eye, especially into the anterior chamber, and may lead to iritis. Or there may be hæmorrhagic choroiditis, or panophthalmitis with sloughing of the cornea.

Other symptoms.—The most curious effect of scurvy is what is known as nyctalopia or "night-blindness."* The patient can see well during the day, and at night he can distinguish objects near a candle and even read. But when he has not the assistance of artificial light, he becomes so blind, even though the moon may be shining, that he has to be led about. The pupils may be dilated in such cases, but there are no ophthalmoscopic changes. Not infrequently a sudden night-blindness has been the earliest indication that the patient was otherwise than well. In vol. ii of the 'Ophthalmic Hospital Reports' (1859), papers on this subject will be found by Dr Bryson and others, founded upon observations made during the Crimean War, and in Her Majesty's ships in different parts of the world.

The order in which the various symptoms of scurvy develop themselves is very different in different cases. Sometimes the gingival affection is the first to appear; it may even constitute the sole manifestation of the disease; but sometimes it follows the purpura by a considerable interval. There may have been no sign of the patient's being otherwise than well until some part of the skin, which has received a trifling blow, becomes the seat of extensive extravasation of blood, or an ordinary purgative dose may, quite unexpectedly, be followed by profuse intestinal hæmorrhage, or an old chronic ulcer of the leg may be found to be spongy and the discharge from it sanious.

Course and event.—The course of scurvy is slow and protracted. There is no fever, except perhaps when there are inflammatory complications. The appetite is bad, but sometimes there is said to be a longing for vegetables and fruit. Thirst is often marked. The pains in the limbs become so severe as to interfere with sleep. Anæmia and emaciation advance rapidly, and subcutaneous œdema is present in many cases. The urine is sometimes albuminous, even though the kidneys may be subsequently found to be healthy. The pulse becomes extremely small and weak, and the heart's impulse may be imperceptible. The muscular weakness is frequently so great that the patient faints if he attempts to sit up in bed. Indeed, this is sometimes the cause of a fatal termination at a comparatively early stage

* Amblyopia nocturna. Hemeralopia or Hemeropia would better express the modern meaning of the word, for *νυκταλωπίασις* or *νυκτάλωψ* (an irregular compound of *νύξ* and *ὤψ*) is used by Aristotle and Galen to signify *day-blindness*, "a defect of sight incident to children with black eyes," only able to see by night (v. Littré *sub voce*).

of the disease, which otherwise seldom destroys life, even in bad cases, until after the lapse of some weeks. Accordingly, at the "Dreadnought" Hospital Ship, it used to be a rule for all patients admitted with scurvy to be hoisted up the ship's side in a recumbent position. It is rare for hæmorrhages from mucous membranes to destroy life directly. Death is more often due to gradual exhaustion and prostration, and then the mind is usually clear to the last. In other cases it is the result of some complication, such as dysentery or acute pneumonia (which may or may not pass into gangrene) or ulcerative endocarditis; or, again, it follows extravasation of blood into the cerebral membranes, or the hæmorrhagic forms of serous inflammation already mentioned.

But in the immense majority of cases—at least when the patient comes under medical observation—recovery takes place. The improvement produced by proper treatment is often immediate and striking; but nevertheless many weeks or even months pass before the health is completely restored. The purpuric spots undergo changes of colour like those which are seen in bruises, and gradually disappear; the smaller ones round the hair-follicles merely turn brown. The brawny indurations slowly subside, but they not unfrequently leave behind them thickenings and fibrous bands which may cause permanent contractions of parts of the limbs, especially at the knee or the ankle, with atrophy of the corresponding muscles. Even the joints themselves may be reduced to a state of ankylosis.

The late Dr Carrington's experience was, that at present these protracted cases are not seen at the Seamen's Hospital at Greenwich, and that during six years no permanent injury had followed scurvy. In the worst cases the rations had been short and the water bad.

Cause.—Our knowledge of the ætiology of scurvy is so far perfect that we habitually prevent its occurrence under circumstances that would inevitably give rise to it but for our interference; and there are very few diseases of which as much can be said. Still, several points require to be carefully and separately discussed. One is whether the chief or ordinary cause of scurvy, which we have under our control, is present in all cases without exception. Now, beyond all doubt that chief cause is the absence of a due supply of *fresh vegetable food.* The question therefore is whether scurvy ever arises when a proper quantity of such food has been taken, and to this a negative answer may probably be given. Immermann, indeed, cites in 'Ziemssen's Handbuch' a few instances in which he says that the disease prevailed notwithstanding that there was an abundant supply of vegetables. But on looking up the first of them, an epidemic which occurred in the barracks of Rastatt in the winter of 1851–52, we find that Opitz, who recorded it nine years later in the 'Prager Vierteljahrschrift,' says expressly that the poor lived almost entirely on soup and beef and dumplings, because vegetables were very scarce and dear. And even had they been cheap it would have proved nothing, unless those persons who were actually taken ill could be shown to have partaken freely of them. So, again, it is not sufficient to assert that vegetable food has been duly served out in rations to a body of men, some of whom are found to have scurvy; we want to know that they have eaten it, and have not thrown it away, or parted with it to their comrades. It does not appear that any case is on record in regard to which these conditions are satisfied.

Potatoes have a high antiscorbutic value; the disease was exceedingly prevalent in Ireland after the failure of the potato crop in 1846, and at

Millbank Penitentiary in 1832 an outbreak occurred which was directly traced to the introduction, a few months previously, of a new dietary from which the potato was omitted. On the other hand, peas and beans are incapable of preventing scurvy, and the same is the case with rice and other cereals. The modern methods of preserving vegetables in a succulent condition leave them with their antiscorbutic properties almost intact; but complete desiccation seems to destroy their usefulness. All fruits, including apples, tend to ward off the disease; cider is said to have no such power, but the acid wines of France are believed to be antiscorbutic.

It is, however, undoubtedly true that scurvy does not always show itself in those who fail to get a proper share of vegetable food. We have therefore to ask, in the second place, whether the disease requires accessory causes for its production; or, if not, what circumstances are capable of counteracting its chief cause. Now, there are some conditions which affect the frequency of the occurrence of scurvy, and which are mentioned by writers on its ætiology, but which really have only an indirect effect. Thus it is more common in cold climates generally than in hot ones, and in winter than in summer, only because a low temperature is unfavourable to the growth of plants. Still, there are grounds for believing that the development of the complaint is favoured by a variety of depressing influences, among which may be mentioned want of sunlight, residence in narrow, cold, dark, damp dwellings (such as cellars or the casemates of a fort), overfatigue, excessive indulgence in ardent spirits, and despondency of mind. It is also said to be particularly apt to occur in persons who are convalescent from ague or dysentery, in those who have syphilis, and in soldiers who are recovering from severe wounds.

The withdrawal of fresh vegetables from the dietary can be neutralised in several different ways. Thus fresh meat, if eaten in large quantities, is an efficient antiscorbutic, especially when raw or but slightly cooked. Indeed, one of the earlier views with regard to the ætiology of scurvy was that it was a direct effect of the salt pork which constitutes so large a part of the diet of sailors; but it has often occurred when no salted provisions of any kind had been taken. The antiscorbutic value of milk was discussed in a valuable essay by the late Dr Parkes in the 'Med-Chir. Review' for 1848. The conclusion seems to be that the ingestion of milk in quantities of a pint or a pint and a half every day does not always supply the omission of vegetables in preventing scurvy; but children and others who live mainly on milk undoubtedly remain free from the disease. On the other hand, infants suckled by scorbutic mothers have often been attacked; and it is very likely that the milk of cows fed almost entirely upon hay may fail to possess the same properties as that of animals which have had plenty of grass.

The most important of all antiscorbutic agents, however, in the absence of fresh vegetables, are the juices of certain fruits, especially the orange and lemon. Their value was recognised as far back as 1573, by Solomon Albertus; and since 1795 *lime-juice* has been regularly furnished to ships in the Royal Navy, with the result that scurvy, which used to commit the most fearful ravages among our sailors, is now scarcely ever seen. Indeed, were it not for the systematic use of this agent, every long voyage, when vegetables are no longer to be had, would probably be an experiment demonstrating the real cause of the disease. The usual plan is to serve out an ounce of the juice daily to each man. In the spring of 1876 an out-

break of scurvy took place among the men of the sledging parties sent out from the ships "Discovery" and "Alert" engaged in the Arctic Expedition. These men had no supply of lime-juice with them, and received only very small quantities of potatoes.* Nevertheless it was afterwards argued that the defects in their dietary were not the cause of their falling ill, chiefly on the ground that cases arose from ten to twenty-seven days after the commencement of the sledging operations, and must consequently have had their origin in the unfavourable conditions under which the men had laboured during the previous long winter. But the Admiralty Committee appointed to inquire into the matter reported that the disease was really due to the absence of lime-juice. In future, it would, under similar circumstances, be well to concentrate the juice so as to render it more portable; and glycerine might be added to prevent freezing. No case of scurvy from the Royal Navy has been admitted into the Seamen's Hospital since 1879. Most of the cases there treated occur in Norwegian sailors.

Why the absence of vegetables from the food should cause scurvy, and how lime-juice is able to take their place and to prevent the development of the disease, are questions upon which there has been much speculation, but hitherto with no positive result. In the last century dilute sulphuric acid and vinegar were largely made use of, in the vain hope that they too might prove to be antiscorbutics; and crystallised tartaric and citric acids have since been tried, but have generally failed.

Dr Garrod, in 1848, propounded the theory that the essential cause of the disease was the absence of a due supply of potass in the food. He showed that there was a great deficiency of the salts of this alkali in dietaries which were known to be liable to give rise to scurvy, and that it was present in abundance in all the substances which possessed antiscorbutic properties. It is curious that whereas his views have never been widely accepted in this country, they are with a slight modification upheld in Germany by the most recent writers at the present time. This modification consists in regarding as valueless those potass-salts that pass out of the body unchanged, and in attaching importance to those alone which undergo conversion into carbonates and may be supposed to enter into the composition of red blood-discs or of muscle. It has in fact been demonstrated that nitrate of potass is incapable of preventing scurvy; but it has not yet been shown that the pure citrate or the tartrate of potass possesses antiscorbutic value at all comparable with that of lime-juice or fresh vegetables.

Histology.—There can be little doubt that the immediate cause of the purpura and of the other hæmorrhagic symptoms of scurvy is a morbid condition of the walls of the smaller vessels. But hitherto no visible change in them has been detected with the microscope. In 1871 Lasègue and Legroux examined the capillaries in seven fatal cases which had occurred during the epidemic in Paris at the time of the siege; they could discover nothing but some scattered fat-granules. Nor have we as yet any certain knowledge as to the alteration in the composition of the blood which must be supposed to form an intervening link between the dietetic cause of the disease and its varied phenomena. After death the blood within the body has sometimes been found to be coagulated; sometimes it has been liquid. It has not seldom been pale and watery; but this is only equivalent to saying that

* They had been given extra rations of lime-juice for some time before starting "in order to saturate their systems."

anæmia is a symptom of scurvy. For the same reason it is doubtful whether much importance can be attached to observations proving that the red discs are deficient in number. Such oligocythæmia necessarily involves a deficiency of potass salts and of iron, which has in fact been shown to occur by several chemists. Laboulbène (1861) made out a slight degree of leucocytosis; but this again may probably have been merely relative, depending upon the scarcity of red discs.

Dr Garrod, Dr Ralfe, and others have carefully investigated the state of the urine, in the hope of indirectly throwing light upon the constitution of the blood in scurvy. Dr Ralfe's conclusions are that the uric acid is increased but that the acidity of the urine is diminished, and that there is a great reduction in the amount of alkaline phosphates. He has propounded the theory that the primary change in the blood is a diminution in its alkalinity, citing in support of this view certain experiments upon animals by Hoffmann and others, in which it has been shown that food yielding only an acid ash produces, after a time, effects like those of scurvy, namely, dissolution of the corpuscles, ecchymoses, and purpura.

Treatment.—In the treatment of persons already ill with scurvy, the administration of lime-juice is almost as important as in its prevention, and the presence of diarrhœa does not contra-indicate its use.

If fresh meat, mashed potatoes, cabbage, and salad can be eaten, there is no objection to the patient's having them; but when the gums and teeth are very tender, the diet often has to be limited to milk, beef-tea, and eggs beaten up with wine. On the Continent the yeast of beer is given.

Dr Buzzard says that a daily application of solid nitrate of silver to the gums affords great relief when they are sloughing and bleeding. Washes of chlorine, Condy's fluid, chlorate of potash, alum, decoction of oak-bark, may also be freely employed. For the hard swellings in the legs, friction with soapsuds and water is said to have been used with success in the Turkish hospitals during the Crimean War. Iodide of potassium is recommended when effusion occurs under the periosteum. It is advised that scorbutic ulcers should be dressed with lint soaked in lemon-juice, or with the bruised substance of succulent herbs, such as the house-leek.

Diagnosis.—The diagnosis of scurvy is chiefly determined by our knowledge of its ætiology. Most observers are of opinion that, assuming as a positive fact that a patient has eaten an adequate amount of fresh vegetable food, one is justified, on that ground alone, in denying the scorbutic origin of his complaint whatever his symptoms may be.

A case in point was brought by Dr Stephen Mackenzie before the Ophthalmological Society in 1880. It was that of a lad who was extremely anæmic, and who had cutaneous purpura and spongy gums, but who (it was stated) had not been deprived of vegetable food. There were, however, hæmorrhages in each retina, with patches of degeneration like those which occur in Bright's disease. This in itself is a reason for thinking that the complaint was not scurvy, in which retinal hæmorrhage is very rare.

As a rule, however, the gingival affection constitutes a safe criterion between scurvy and other purpuric affections, including the "morbus maculosus," to be presently described. But in splenic leuchæmia Mosler and others have sometimes found the gums swollen and ulcerated, as well as inclined to bleed; and in some cases of malformation of the heart slight morbid conditions of the gums have been.

Whenever there is a doubt as to the diagnosis it may be quickly settled

by the administration of lime-juice. And the same may be said with regard to the exceptional cases of scurvy in which the gums remain in a normal state, as well as of those other rare instances in which there are no symptoms except the spongy state of the gums.

But the most important point of all is the danger of overlooking the slight forms of scurvy which occur sporadically among the poor of London and other cities. Dr Buzzard remarks that such persons, who probably never brush the teeth, attach no importance whatever to an unhealthy condition of the gums; nor do they notice a slight petechial eruption upon the legs. What they are likely to seek relief for is a supposed "rheumatic" complaint attended with muscular weakness. It is essential, therefore, to be on the look-out for the other symptoms of scurvy, including the peculiar sallow complexion, when advice is asked for vague pains in the limbs. In out-patients' practice we are thus able to detect the disease in men who have been living without vegetable food, although they had not the slightest idea of the cause of their symptoms.

Infantile scurvy.—This is an occasional consequence of bad feeding, as is now established by the observations of Dr Cheadle and Dr Thomas Barlow. It often occurs with rickets, and when thus complicated has been described by German writers under the title of "acute rickets," by Mr Thomas Smith as hæmorrhagic periostitis, and by Dr Gee as "osteal or periosteal cachexia." The fact that young children are often given fruit and potatoes, with other improper food, probably saves many of them from scurvy. Beside the other symptoms, effusion among the muscles of the thighs, under the periosteum, or at the junction of the epiphyses with the shafts of the long bones is particularly common (see Dr Barlow's paper 'Med.-Chir. Trans.,' 1883). This condition has often been mistaken for infantile syphilis. The gums are only occasionally spongy, perhaps from the absence or fewness of teeth, for the child is usually under eighteen months old.

Dr Eustace Smith recommends the administration of raw meat-juice and orange-juice, together with free ventilation of the nursery and taking the child out in the open air whenever the weather is suitable. Cod-liver oil is also useful.

PURPURA.*—This affection, "the purples," consists in the formation of spots of hæmorrhage in and beneath the skin, and occurs in many different diseases, viz.:—1. In scorbutus. 2. In hæmophilia. 3. With rapidly diffused sarcoma (vol. i, p. 3). 4. In Hodgkin's disease, splenic leuchæmia, and grave anæmia. 5. With cirrhosis of the kidneys, acute rheumatism, and ulcerative endocarditis. 6. As a complication of "erythema multiforme." 7. In scarlatina and variola (vol. i, pp. 229, 237, 213).

In all these varieties the cutaneous hæmorrhages are secondary, nor are they by any means universally present. But there is another form of purpura of which they constitute the fundamental and essential symptom, and for which at present no cause can, as a rule, be discovered.

In Germany this disease is commonly spoken of as the "Morbus maculosus Werlhofii." It was described in the last century by Werlhof, a physician who held a Court appointment to George III in Hanover, and died in 1767. In his 'Opera Medica,' collected by Wichmann, a well-marked

* *Synonyms.*—Purpura simplex et hæmorrhagica—Idiopathic purpura—Morbus maculosus Werlhofii.—*Germ.* Blutfleckenkrankheit.

case is recorded; the patient was a girl previously healthy, who also had epistaxis, hæmatemesis, attacks of syncope, and a small and very rapid pulse; she ultimately recovered

Of course it cannot be pretended that Werlhof would have exactly limited his morbus maculosus to what we now regard as primary or essential purpura. Nor have we even now any certain warrant for assuming that this may not hereafter be still further broken up into separate affections, due to different causes. There is, indeed, already a subdivision, accepted by most writers into "P. simplex," confined to the skin, and "P. hæmorrhagica," attended with bleeding from various mucous membranes and with ecchymosis of the deeper structures of the body. But this distinction is artificial and unnecessary, for the less severe "simple" cases of purpura are very apt after a few days to pass into the "hæmorrhagic" form.

Ætiology.—The morbus maculosus seems to be more common about the age of puberty than at any other period of life; females are said to be more subject to it than males, and this applies particularly to children.

The patient is often well nourished and fresh coloured, having had good food and having been apparently quite well up to the time when the cutaneous spots are observed, or when hæmorrhage begins from some mucous surface. Sometimes he is anæmic and weakly, or he may have recently recovered from an acute disease, such as enteric fever. Immermann (in 'Ziemssen's Handbuch') remarks that in these cases the purpura usually shows itself when the appetite is returning and when the first attempts to stand are being made. Now and then, when there has been no antecedent malady, there is a short prodromal stage of malaise, anorexia, and headache, lasting two or three days or even a week.

But in some cases the occurrence of purpura is directly attributable to the medicinal administration of the iodide of potassium, the corresponding sodium-salt being generally incapable of producing a similar effect. This "iodic purpura" appears to be in all respects identical with the ordinary form of the disease. Descriptions of it, with copious references, are given in papers by Dr Stephen Mackenzie (in the 'Medical Times and Gazette' for 1879) and by Dr Duffy (in the 'Dublin Med. Journ.' for 1880). In one case—that of a syphilitic infant, five months old—it is said to have directly followed a single dose of two and a half grains of iodide of potassium; but it is generally not seen until the salt has been taken for some days or for several weeks. The spots may sometimes subside, notwithstanding that the patient goes on with his medicine; but in most cases, should he have discontinued it, a fresh crop appears as soon as he attempts to resume it.

Symptoms.—Not infrequently the legs are alone affected by purpura, or the spots may appear there earlier than anywhere else, the forearms being the next parts to be attacked, and the face suffering last, or not at all. But sometimes ecchymoses come out simultaneously over nearly the whole of the cutaneous surface in untold numbers. They present differences of colour, which chiefly depend (as Dr Hyde Salter pointed out in the 'Medical Times and Gazette' for 1856) on the depth in the skin at which the blood is extravasated. Thus the more superficial of them, which are seen through only a thin layer of tissue, appear bright red and sharply defined; the deeper ones are of a purple hue and fade off gradually at their edges; they are also generally larger, because the meshes of the tissue in which they lie

are more open. Very rarely the cuticle is raised into a dark bleb by blood poured out upon the surface of the rete. The spots bear no definite relation to the hair-sacs, such as has been described in scorbutus. Nor is there so marked a tendency to the formation of large subcutaneous vibices; but the eyelids are sometimes surrounded by broad black rings, and extensive effusions of blood may sometimes be seen in other regions, especially when there have been blows or other injuries. Occasionally retinal hæmorrhage occurs; Dr Goodhart describes a case in a child four years old.

Among the mucous membranes, that of the nose is perhaps more apt than any other to bleed in the morbus maculosus; and epistaxis is often the earliest symptom. Hæmorrhage also frequently occurs from the stomach, the intestines, the urinary passages, or the female genital organs; and occasionally from the bronchial tubes. Blood may ooze from the gums, and collect round the bases of the teeth in dark red or black crusts; but when these crusts are removed, the gingival tissues are never found to be swollen, spongy, nor of a purple-red colour, as in scorbutus; they are either perfectly normal in appearance, or more or less anæmic.

In the more severe cases of purpura, when there has been a considerable loss of blood, the patient may rapidly pass into a condition of extreme anæmia, with waxy pallor of the skin, a rapid feeble pulse, and liability to faint on the slightest exertion, or even on attempting to sit up. Under such circumstances fever may be present, as in all other forms of extreme anæmia. Immermann further suggests that the temperature may sometimes rise as the result of the reabsorption of the extravasated blood, or in consequence of the setting up of local inflammatory changes by its irritant action upon the tissues among which it lies. It seems still to be doubtful whether fever is ever present as an initial symptom, or at least whether the occurrence of a high temperature at the beginning of an attack of purpura does not show that the case is of a peculiar kind.

With regard to the condition of the blood within the vessels in purpura there have been different statements. It has been said to be deficient in coagulating power, but this seems to be a mistake. There is always diminution of red corpuscles. Immermann estimated the proportion of leucocytes in a severe case at Basle; during the first few days it was normal, but afterwards there was a slight excess, as is usual after all kinds of hæmorrhage. Laache, however, found leucocytosis from the first.

Event.—As a rule, purpura ends rather quickly in recovery. In some cases fresh spots may cease to come out after a few days, the old ones fade and disappear, the mucous membranes cease to bleed, the anæmia is quickly repaired, and within three or four weeks the patient is as well as ever. If he should get up too early it often happens that a new crop of spots may be seen upon his legs within an hour or two of the time when his feet are first allowed to touch the ground; but these soon undergo absorption in their turn. Sometimes, however, the disease runs on for several weeks, or it may recur again and again, with intervals of many months, during which the health appears to be perfectly good.

Beside the ordinary cases of idiopathic purpura which are confined to the legs, and readily cured by laxatives and arsenic or steel, we occasionally see more severe cases in previously healthy young patients. Two of these have been lately under the editor's care in Guy's Hospital. One was a healthy lad aged fourteen, whose case is published in the 'Pathological Transactions' for 1884. The disease came on without known cause, and ended fatally in

about ten days. While lying almost unconscious the day before his death a house-fly settled on his face, and before it could be brushed off had left a bleeding mark. Here micrococci were found in venous thrombi after death. The other case occurred in a robust young man aged twenty-two. The onset was unexpected, and the symptoms well marked. Severe epistaxis was effectually stopped by Billoc's apparatus (p. 283), and active treatment checked the hæmorrhage from the bowels, stomach, and kidneys. But all the ill symptoms returned, and he died on the fourteenth day. Here micro-organisms were looked for by the editor and by Mr Cheyne, who had seen the former case, but none were found; nor were they in Dr Wickham Legg's two cases ('St Barth. Hosp. Rep.,' 1884; and 'Path. Trans.,' 1885).

Anatomy.—After death the lining of the stomach, intestine, uterus, kidneys, and bladder are generally found spotted with ecchymoses, as are also the pleura, the pericardium, the arachnoid, the peritoneum, and even the substance of the lungs and the medulla of the bones. In some cases an effusion of blood upon the surface of the brain or into the ventricles is the direct cause of death. Thus a man aged thirty-four was admitted into Guy's Hospital for purpura, and appeared to be doing well, when he became insensible and paralysed on the right side. Afterwards there was loss of power in the left limbs also, and he died on about the twentieth day of his illness. A quantity of blood was found extravasated on the left hemisphere of the brain beneath the pia mater, and also within the ventricular cavities. Another case, which occurred in 1871, was that of a woman aged twenty, who, while in the hospital for phthisis, was attacked with a severe form of purpura, and with epistaxis. At the end of about a week she became delirious for some hours on two successive days. Then the bleeding ceased, and the spots disappeared, but she sank gradually a fortnight later with diarrhœa. The dura mater over each hemisphere was found lined with a uniform layer of blood, which was yellowish in tint, and almost membranous.

In other instances some of the more delicate structures of the body seem to slough as the result of the infiltration of blood into their tissues. Thus a girl aged nine was admitted into Guy's Hospital for gangrene of the external genitalia and purpura. So far as could be learned she had not been affected with any one of the contagious exanthemata. There was a fœtid discharge, and she died in four or five days. The bladder, the vagina, the uterus, and the Fallopian tubes were all found intensely inflamed and covered with spots of hæmorrhage. Some years later a man aged twenty-three died after an illness of eight days' duration, which began with a purpuric affection of the right thigh. The lower end of the ileum, for about one foot of its length, was of a purple colour, its coats thickened and infiltrated with exudation, its serous surface coated with lymph, and its mucous membrane slightly excoriated. Zimmermann has related in the 'Arch. f. Heilkunde' for 1874 a case in which several intestinal ulcers formed and sloughed through into the peritoneum, setting up a fatal peritonitis. See also Dr Legg's second case above referred to.

Pathology.—There are good analogies in support of the view that the morbus maculosus depends primarily upon a peculiar alteration in the blood; but it seems clear that, before the hæmorrhages occur, this must have led to a morbid state of the walls of the capillaries, perhaps by impairing their nutrition. Immermann suggests that the purpura which occurs during convalescence from fever is possibly due to the circumstance that

the recovery of the minute vessels is sometimes retarded beyond the time at which the volume of the blood is restored and the heart regains its vigour. In a case recorded by Dr Wilson Fox, in the 'Med.-Chir. Rev.' for 1865, the arterioles and capillaries of the skin in the neighbourhood of purpuric patches were found to be obviously altered in appearance; they were brittle, had a glistening waxy look, and assumed a most intense reddish-brown colour with iodine. The patient, a man aged thirty-three, had been affected for about a month with a syphilitic eruption, which followed an indurated chancre at five months' interval. He had taken iodide of potassium for some time, but not continuously. It seems doubtful whether any of the viscera were lardaceous, except perhaps the adrenal bodies and the intestinal mucous membrane. Many of the muscles were in parts pale and waxy looking; their fibres had lost their striation, and become deeply stained by iodine, while their blood-vessels showed changes similar to those observed in the affected parts of the skin. Dr Thin ('Med.-Chir. Trans.,' lxii) described the minute blood-vessels as obviously altered and disorganised within the area of a bulla caused by the administration of iodide of potassium; and he suggests that iodic purpura is due to a more extreme change.

In a case of the editor's, in 1883, referred to above, Mr Watson Cheyne found strings of micrococci in the tissues. In a case of Dr Russell's, of Carlisle, he had previously found bacteria ('Path. Trans.,' vol. xxxv, p. 408). In other instances no microphytes have been found, and their presence is probably accidental.

Diagnosis.—The diagnosis of the morbus maculosus rests upon the exclusion of all the various diseases which may give rise to symptomatic purpura. The possibility of the sporadic occurrence of scurvy must not be forgotten, but even mild scorbutic cases are generally distinguished by positive characters, of which the chief are the debility and anæmia that precede the cutaneous affection, the swollen and spongy state of the gums, the brawny induration in the hams, and the formation of each of the spots round the mouth of a hair-sac. In cases of purpura, hæmorrhages from the mucous surfaces are generally much more profuse than they are in scorbutus. Malignant sarcomatous growths must always be carefully sought for, the heart and the urine must be examined, and the state of the spleen and lymphatic glands must be investigated.

Treatment.—There is reason to believe that certain medicines are capable of preventing the formation of fresh spots of purpura and of averting the hæmorrhages from mucous membranes which constitute the most serious part of the disease. Of these *arsenic* appears to be the most valuable. Dr Habershon recommended it in the 'Guy's Hospital Reports' for 1857; it has since been commonly employed in our wards, and in one case it succeeded at once, when many other drugs had failed. Turpentine is strongly recommended by Dr Gee, and seems to be the most efficient hæmostatic in some instances; while ergot, acetate of lead, or gallic acid succeed better in others. Immermann says that it is important not to treat the consecutive anæmia by ferruginous preparations for some time, and that their administration has sometimes led to a relapse. The patient should be kept in bed in a cool room; he should have a light milk diet, and his bowels should be kept open by laxatives. Stimulants are often needed. Dr Eustace Smith recommends in previously healthy children oil of turpentine in castor-oil, which acts as a drastic purgative.

HÆMOPHILIA.*—From the commencement of the present century it has been known that in certain families the males during successive generations are liable to protracted and sometimes fatal hæmorrhage after injuries of no great severity: such persons are called "bleeders." Attention was drawn to the subject in 1803 by an American physician, Otto, who gave an account in the 'Philadelphia Medical Repository' of a family in which this morbid tendency had existed for seventy or eighty years. It is said to be first mentioned in the writings of an Arabic author who died at Cordova A.D. 1107. In 1784 Sir William Fordyce recorded the case of a Northamptonshire family affected in a similar way. A monograph on the disease was published by Dr Wickham Legg in 1872.

Ætiology.—The isolated occurrence of hæmophilia is exceedingly rare. When a "bleeder" is born of healthy parents, it almost always happens that some or all of their subsequent children are also affected; and probably the disease had already existed in a grandparent or some ancestor.

The preponderance of males among those who suffer from hæmophilia is nearly as thirteen to one. Moreover, when it does affect women, hæmophilia is much less severe, and scarcely ever fatal: its signs are often limited to the occurrence of cutaneous ecchymoses, spontaneously or after slight injuries—or, in other cases, to epistaxis, menorrhagia, or excessive post-partum hæmorrhage. Immermann, however, quotes an instance, recorded by Reinert, of a family of sons and daughters, in which the daughters alone were bleeders, while the sons were all free.

It is very remarkable that the inheritance of the complaint takes place mainly through the female line. The sons in a hæmorrhagic family do not all invariably suffer; and if any escape, their children are almost always exempt. Even those sons who are affected, if they live to beget offspring, may have some boys who are healthy. But the daughters of such a family, though they have not themselves shown the slightest indication of hæmophilia, are almost certain to transmit it to their male children. Obviously these facts are very important in regard to the advice which should be given if one is consulted as to the propriety of marriage on the part of members of a hæmorrhagic race. At Tenna, in the Grisons, there were until lately two families, not known to be related to one another, in which the disease had been known to exist for a century. In 1855 the females of these families determined to renounce marriage, and in 1879 Immermann was able to state, on the authority of Dr Hörsli, of Tusis, that there was no longer a well-marked example of hæmophilia in the village.

A curious circumstance is that persons with an inherited tendency to the disease seem to be unusually prolific, the average proportion of children to each union being nine, whereas among the population generally it is five.

Hæmophilia occurs in those who are lean as well as in those who are fat, and in persons whose hair and eyes are of all varieties of colour. Its victims are, however, said to have generally a thin, delicate, and transparent skin, with full subcutaneous veins.

The disease has repeatedly been observed in European Jews, and if a large proportion of recorded cases have occurred in nations of Teutonic as compared with those of Latin origin, the reason probably is that it is only in certain countries that much attention has yet been given to the subject.

* *Synonyms.*—Hæmorrhaphilia (Schönlein), a barbarous word, intended to mean love of hæmorrhage (φιλία)—Hæmorrhagic diathesis.—*Fr.* Hémophilie.—*Germ.* Bluterkrankheit.

Course and symptoms.—Hæmophilia does not usually appear at the time of birth. The detachment of the navel-string has but rarely been attended with bleeding in children who were afterwards to suffer from it. But about the end of the first year, or at least before the close of the second year, definite symptoms generally manifest themselves. The latest age at which it is said that hæmophilia commenced and assumed a serious form is the twenty-second year.

The hæmorrhages which characterise the disease are commonly divided into those which are traumatic and those which are spontaneous, but the distinction is only partially applicable, since slight injuries are very apt to be forgotten, which in "bleeders" may give rise to considerable extravasations. Thus spots and patches of effused blood in and beneath the skin may sometimes be traced to the pressure of the clothes. But there can be no doubt that they sometimes arise independently of any such cause, especially where they are small and such as would be termed petechiæ. Indeed, successive crops of cutaneous purpura may be observed in this disease exactly as in the "morbus maculosus" and in so many other morbid states. The spontaneity of hæmorrhages from mucous surfaces is said to be sometimes shown by their being preceded by well-marked signs of "fluxion" —throbbing of the heart and of the arteries, redness and heat of the cheeks, ears and lips, headache, giddiness, restlessness, and irritability of sight and hearing. Epistaxis is the most common form of mucous hæmorrhage, especially in children; according to Grandidier it is four times as frequent as any other. Next in order of occurrence comes bleeding from the gums and mouth; this, however, may be traumatic, for Dr Legg mentions that some patients cannot use an ordinary tooth-brush without drawing blood. Again, there may be hæmorrhage from the stomach, the intestines, the lungs, the urinary passages, the female genitalia, or even the lachrymal caruncle. Blood does not often escape from the unbroken skin, but cases are on record in which it has oozed from the finger-tips or the ears.

In marked contrast with these spontaneous or quasi-spontaneous forms of hæmorrhage, are those which result directly from blows or cuts. Even slight superficial scratches, such as would scarcely be noticed in a healthy subject, may bleed so as to endanger life. There are, however, considerable differences as to the amount of injury that can be borne, not only in different hæmophilic patients, but in the same patient at different periods. One cut may cause but slight loss of blood, whereas there may be the greatest difficulty in checking the oozing from another precisely similar cut on a later occasion. The operation of ritual circumcision has several times proved fatal. So have venesection, the application of leeches or cupping glasses, and, above all, the extraction of a tooth. Indeed, although Dr Legg says he has seen a tooth drawn without there being any remarkable amount of hæmorrhage, both he and all other writers are agreed that extraction is a dangerous operation in those who are "bleeders." The slight punctures which have to be made for vaccination have scarcely ever given rise to much bleeding.

It must be remembered that the risk of hæmorrhage from a wound continues until it is completely healed; for a thin cicatrix has been known to give way after having formed.

The deeper structures may also be the seat of extravasations of blood, which reach an enormous size, and are almost peculiar to hæmophilia. A subcuta-

neous tumour may be formed, as large as an apple or even as a child's head. It sometimes seems to rise spontaneously, but very often it is due to some slight injury. Thus, in a case of Sir William Jenner's, cited by Dr Legg, the fall of an india-rubber air-ball upon the thigh filled the connective tissue with blood from the knee to the trochanters. The thigh is, indeed, one of the favourite seats of such swellings, and Immermann says that they are also frequently seen under the false ribs, and upon the back. They are generally of a black or dark-blue colour, surrounded by a zone of red. They are sometimes very hard, sometimes soft or fluctuating. They may be hot, and painful and tender to the touch; and in some cases they suppurate, discharging a mass of altered blood with shreds of broken-down tissue, after which hæmorrhage is apt to go on for a considerable time. If they are punctured by the surgeon, dangerous bleeding commonly follows; but if left alone they slowly subside and at last disappear.

In hæmophilia the effusion of blood into any one of the large serous cavities seems to be rare. Immermann cites two cases in which the peritoneum was the seat of hæmorrhage, and four in which it occurred in the cerebral membranes; but in at least three of these latter there had been a fall or a blow upon the head. He says that extravasations into the pleural sac or into the pericardium have not yet been observed. It may therefore be worth while to note that in a case in which Dr Goodhart made an autopsy, the right pleura was closed by adhesions which were in part stained of a deep orange colour.

Articular affection.—One of the most remarkable features of the disease has still to be mentioned, namely, the occurrence of swelling in one or more joints, especially the knees. This affection usually begins between the seventh and the fourteenth year. It is sometimes the direct result of a blow, and sometimes it immediately follows a long walk; but Dr Legg says that the most common cause is exposure to cold or the occurrence of damp and chilly weather, and that it is most frequently seen at the beginning of spring and at the end of autumn. The enlargement often occurs rapidly, and appears precisely like that due to rheumatism, or to synovitis from injury. Many writers have therefore expressed the opinion that it often depends upon simple serous effusion into the articular cavity. But all the pathological evidence which at present exists seems to point to the conclusion that the primary lesion is extravasation of blood. It is true that when a joint has been affected for a length of time the cartilages show signs of chronic inflammation, and that the projecting folds of synovial membrane in its interior are thickened and swollen. This was markedly the case in the knee of a boy who was under Mr Bryant's care at Guy's Hospital in 1880, and who had had the joint more or less swollen for three years. But in the same patient there were other joints in which at the *post-mortem* examination all the structures were found healthy, except that they were stained by orange-coloured pigment and had stringy masses of ochre-brown fibrin adherent to them. It might perhaps be supposed that if a great quantity of blood were poured out into a knee-joint, a discolouration must be visible through the skin, but this is negatived by observation. The usual course of the articular affections of hæmophilia is slowly to subside under treatment, but to return again and again at intervals of months or years.

Prognosis.—In some very exceptional cases of hæmophilia, the hæmorrhagic tendency is said to cease during childhood or youth, and the patient afterwards remains free from the disease. But by far the larger number of

those who are affected with it die before they are eight years old.* When adult life has been reached the danger is less, but it is by no means at an end, for fatal bleeding has been known to occur as late as fifty or sixty years of age.

The habitual condition of bleeders, even when they have long suffered from the disease and are perhaps still troubled with a joint-affection, is not generally one of permanent bloodlessness; they often have as much colour in the face and lips as other people. But the immediate effect of a profuse loss of blood is of course an extreme degree of anæmia. The quantity poured out is sometimes enormous. One case is related in which, after the extraction of a tooth, half a gallon was lost in less than twenty-four hours. It often happens that oozing goes on at the rate of three or four pints a day for several days. When the source of the hæmorrhage is visible, it seems to come, not from a single vessel, but from a surface, as from a saturated sponge. Sir William Jenner has remarked that it has generally appeared to him to be venous rather than arterial. As the bleeding goes on, the patient becomes blanched, pulseless, delirious and unconscious, and death is often preceded by convulsions. But sometimes, when his vital powers are reduced to the lowest ebb, the oozing, which may have resisted all treatment, ceases spontaneously; he remains apparently on the brink of dissolution for several days, and then slowly revives. Writers generally say that the blood undergoes restitution in such cases more rapidly than might be expected, but Dr Legg remarks that the anæmia remains for at least four or six months.

Diagnosis.—The diagnosis of hæmophilia is not difficult in confirmed cases. But one must keep in mind the possibility that a joint-affection may be due to this cause, even when it occurs in a person who may not be anæmic, who exhibits no purpuric spots, and who does not mention that he is a bleeder. The real nature of less marked examples of the disease, such as occur in the female sex, could probably never be positively determined without reference to the history of family predisposition. Many women have a tendency to bruise very readily, and others are subject to the recurrence of spontaneous hæmorrhages, especially "hæmatidrosis," in which blood oozes from the mouth of the hair-sacs or sweat-glands.

In the 'Medical Times and Gazette' for 1871, Dr Legg has recorded two cases, in women, of a "hæmorrhagic diathesis," in which the resemblance to hæmophilia was nearly complete, there being a great liability to hæmorrhage from slight wounds, and also to epistaxis, menorrhagia, and purpura. In each instance the abnormal state had been present for some years, but in neither of them did it exist before puberty. This last circumstance, however, could hardly be held to exclude hæmophilia, because many of the slight forms which are seen in women seem not to be recognisable during childhood. But what appeared to be conclusive was that each patient had borne male children who were not bleeders, and that no family history of hæmophilia could be elicited.

Pathology.—With regard to the nature of hæmophilia very little can as yet be said. Some of the older writers supposed that it depended upon a defective coagulability of the blood. But it is now known that this was a mistake; there is no proof whatever of any abnormal condition of the blood, except as the result of the continuance of hæmorrhage. So, again,

* Grandidier (quoted by Eustace Smith) found that of 150 boys who were bleeders, more than half died under seven years of age, and only nineteen reached twenty-one.

the fatty change sometimes found in the substance of the heart is clearly consecutive to the anæmia which commonly precedes death (p. 752). Several observers, however, have noticed a peculiar thinness and transparency of the arteries—not only of the aorta and pulmonary artery, but also of such branches as the temporals and radials. Others, indeed, have failed to discover anything of the kind; and the most that can be said is, that although not itself the cause of the symptoms of the disease, it may possibly point to the existence of a similar abnormality of the capillaries, and that this may really be the cause.*

Dr Legg suggests that hæmophilia may depend upon a backwardness of growth or imperfect development of the vascular system generally. Immermann also speaks of a "hypoplasia" of the vessels; but he thinks that the essential thing is a disproportion between the capacity of the circulatory apparatus and the volume of the blood. He imagines that in hæmophilic males there is an actual overgrowth of the blood itself, and he endeavours to account for the transmission of the disease by females who themselves are not bleeders by supposing that they have the thin vessels, without the excess of blood. Sir William Jenner's authority may be quoted ('Brit. Med. Journ.,' 1876, ii) for the view that there is in hæmophilia "a tendency to plethora of the smaller vessels." He remarks that it is when the patient is looking his best, that injuries have the worst effect and that spontaneous hæmorrhage is most likely to occur.

Treatment.—These views are not without bearing upon the management of bleeders. Their diet should be light. Jenner recommends "a considerable proportion of white meats." An aperient dose of sulphate of soda may be given every week, and a mercurial purge every third week. Dr Legg has found that after taking iron, patients have been less liable to spontaneous bleeding and have lost less blood after wounds. A warm, dry climate is desirable.

When actual hæmorrhage is going on, the tincture of iron, or ergot, or gallic acid may be used internally. Locally there appears to be no styptic so valuable as the perchloride of iron; a strong solution may, if necessary, be applied to the interior of the rectum; and when the socket of a tooth bleeds after extraction, solid crystals of the salt may be introduced.

* See a paper by Dr Percy Kidd ('Med.-Chir. Trans.,' vol. lxi, p. 243), and Dr Legg's account of four other cases ('Path. Trans.,' xxxvi, p. 490); also Dr Theodore Acland's account of the state of the thymus (ibid., p. 491), and Dr Laache's observation (loc. cit., p. 43).

GENERAL DISEASES AFFECTING THE JOINTS

GOUT*

History—Onset and symptoms of a first attack—Subsequent course—The affected joints—Lithate of soda—Tophi—Pathology of gout—Ætiology—Diagnosis—Course and prognosis—Gouty kidneys—Treatment by drugs, diet, and baths.

THIS remarkable disease was well known in classical times. Hippocrates gives a good account of gout. Lucian wrote a burlesque tragedy in which *Podagra* is one of the dramatis personæ.

In modern times, the literature of gout dates from Sydenham, who was very competent to write about this disease, having himself suffered from it for thirty four years. His masterly description was copied by Cullen, and has formed the basis of almost all that has since been written upon the subject.

The fit of gout.—In a large proportion of cases, when gout occurs for the first time in a young subject, it begins in a remarkable way. The patient is asleep in bed, when he is awakened, about two o'clock, or between two and five, by a pain in one of his feet, generally in the ball of the great toe, but sometimes in the heel, ankle, or instep. This pain is described by Sydenham as being like that of dislocation; and yet, he says, the parts feel as if tepid water were poured over them. Then follow chills and rigors and a little fever. The pain which was at first moderate, becomes more intense. It is characterised as a grinding, crushing, wrenching pain; or as a burning, such as would be caused by a hot iron pressed into the joint; or as if a dog were gnawing it. The patient keeps changing the position of his foot, in the vain hope of finding a place in which to lie in comfort. He cannot bear the bedclothes to touch it. The least vibration of the floor causes him extreme distress, so that those about him have to tread the room with the lightest possible step, and the passage of a waggon along the street below sends him almost mad with rage. For, as Sydenham puts it, "a fit of gout is a fit of bad temper."

Towards morning the patient has a sudden and slight respite, which he perhaps imputes to having at last found a comfortable position. He perspires gently and falls asleep. He wakes freer from pain, and then finds that the part is swollen. Till then, the only visible swelling had been that of the veins round the painful joints. For the next two or three days the pain becomes worse towards evening, and abates in the morning. Then, however,

* *Gk.* Ποδάγρα. The terms χειράγρα, ὠμάγρα, and similar compounds were also used.—*Lat.* Podagra: in Mediæval Latin Gutta, whence *Gout*, *Goutte*, and *Gicht*. "Arthritis" is sometimes used as a synonym by classical and by modern writers.

not infrequently, the other foot begins to swell, and the whole tragedy, as Sydenham calls it, is acted over again.

A joint affected with gout, besides being painful and swollen, is also of a deep red colour, tense, and shining. As soon as the patient can bear the pressure of the finger, one finds that the skin over it pits, or (in other words) that there is œdema. Subsequently the cuticle peels off; and the part then itches very much.

The amount of febrile disturbance is proportionate to that of the local inflammation. This is a point of some consequence, as it constitutes one of the distinctions between gout and acute rheumatism; in the latter disease there may be high fever, with scarcely any joint affection.

In robust patients, who have not had more than a few previous attacks, the duration of a fit of the gout is about a fortnight. But in persons of advanced age and broken-down health, it may last two months or even longer. In that case, however, the length of the attack is rather apparent than real; for it is in fact made up of a series of irregular minor fits, which gradually become milder.

It is generally said that before a first seizure of gout the patient appears to be in his usual health. Certainly he seldom has any idea of what is about to befall him. Sydenham, however, observed that indigestion and flatulence precede the attack; and Dr Garrod says that the premonitory symptoms are sometimes very distressing. Trousseau remarks that in addition to symptoms of hepatic disorder the patient often has an irregular and capricious appetite, preferring acids and meats strongly spiced, as if he felt the necessity of stimulating his torpid organs of digestion.

After an attack of gout, on the other hand, a man often feels much better than for some time before. He is more active, and free from many uncomfortable feelings that had before troubled him. Sooner or later, however, these return, and are the prelude to a second seizure. Sometimes this does not occur for two or three years after the first; but often the interval is not more than a twelvemonth, and it may be less. The second attack is in its turn succeeded by others, and always at shorter intervals; until, at last, the patient may be scarcely ever free from the disease.

Probably in consequence of Sydenham's graphic and accurate description of a first attack of acute gout, it has been too often assumed that such is its usual or constant mode of onset. But in many cases, perhaps in the majority, the first attack is far less acute, severe, and dramatic: the tragedy is more slowly played out. And in not a few patients gout begins in no acute or sudden form, but stealthily and almost imperceptibly. A man about fifty complains of his boots being tight or sprains his ankle while shooting, or feels a twinge in one knee as if it were strained. There is no pain at night and no redness or swelling to be seen, but a moderate walk is more and more apt to be followed by a "sprain," and at last the great toe or instep or ankle is found to be decidedly swollen, tender, and slightly reddened, so that the suspicion, which the patient had long silenced, becomes certainty,—he has the gout.

Locality.—It has already been stated that the ball of the great toe is the joint most frequently first attacked by gout. Sir Charles Scudamore found that this joint was affected on one side or the other in 373 out of 512 first seizures. When any other part shows the earliest manifestation of the disease, the reason appears generally to be that it has in some way been injured. Thus, according to Garrod, the knee may be attacked before any

other joint, if the patient has injured it in falling from his horse, or otherwise; and a considerable time may pass after the accident before the result follows. Next to the great toe, the joint most obnoxious to gout is probably the metacarpo-phalangeal articulation of the index finger; certainly not that of the thumb.

Whatever may be the part first affected, other joints afterwards suffer in almost all cases. In the very first seizure, two or three different articulations may be attacked. These are generally some of the small joints, such as those of the toes or fingers, the ankles or wrists. Even in the most advanced and intractable cases the largest joints of all—the shoulders and hip-joints—are usually spared.

Anatomy.—When a patient has suffered from repeated attacks of gout, the affected joints become greatly deformed. This is more marked in the fingers than in the toes, no doubt because their natural movements are more free. The fingers become bent irregularly in all directions; sometimes inwards, sometimes outwards. A very common state is for the metacarpo-phalangeal and the second phalangeal joints of a finger to be stiffly flexed, while between them the first phalangeal joint is over-extended, its knuckle being represented by a deep hollow.

Sometimes the sides of the fingers are greatly enlarged. Sydenham compares their appearance to that of a bunch of parsnips. But in many cases they are very little altered in form.

Lithate of soda.—In long-standing cases of gout there are found in the tissues round the joints masses of a white material having the consistence of putty or mortar, or hard and dry like chalk. These masses are called *tophi*,* or "chalk-stones." They do not, however, consist of carbonate of lime, but mainly of urate of soda. This salt is also found deposited in the articular cartilages of the affected joints. It there looks as if it were upon the surface of the cartilages, covering them more or less completely, and appearing just as if it had been laid on by a brush. However, on making a section of the cartilage one finds that the deposit is really in its substance. Examined by the microscope, it proves to consist entirely of crystals. There are bundles of very fine needles, the centres of which (according to Cornil and Ranvier) commonly correspond with the cells of the cartilage. It is in the superficial part of the cartilage that the crystals are most densely crowded; they often make it opaque to transmitted light. Towards the articular end of the bone they are more thinly scattered; here they often traverse the whole thickness of a cartilage cell, which is, as it were, impaled on them.

The synovial membrane may contain similar deposits of the urate; but they are much more apt to occur in the fibrous structures outside the joints, particularly the surrounding ligaments and tendons.†

The same salt is also deposited in many cases, even at a distance from

* *Tophus* or *tofus*, the Greek τόφος, seems to have been applied to rough crumbling rock, the disintegrated volcanic *tufa*. Virgil associates it with chalk: *Et tophus scaber, et nigris exesa chelydris Creta* ('Georg.,' ii, 214).

† There are some discrepancies in the statements of writers as to the deposition of lithate of soda in the *bones*. Dr Garrod says that he has never been able to find it there. But Cornil and Ranvier teach that it is not uncommon in the cancellous tissue. They mention one case in which they observed it, and in which the bones forming the metatarso-phalangeal joint of the great toe had entirely lost their cartilages and were ankylosed together, and say that absorption of the articular cartilages is a common remote effect of urate of soda in a joint. This, however, is not in accordance with our experience in England. Bony ankylosis is unknown, and it is very rare to find the cartilage gone in a gouty joint.

the joints. The writer remembers seeing a man who had a number of small ulcers, some of them in the middle of his thighs and legs, from which masses of urate of soda were discharged. In 1886 and 1887 a patient was in Philip Ward, who had tophi of lithate of soda in the skin of his legs and arms, as well as in the joints and ears. The integuments of the limbs, however, very rarely present such deposits, except over articulations. But there is one particular region where they are very often to be found. This is in the external ear, generally in the helix. Garrod remarks, with his wonted accuracy, that these deposits in the pinna of the ear are at first fluid, the skin over them forming a vesicle of a milk-white appearance. He says that some months elapse before they become the white, hard, bead-like masses, which are commonly seen. Is any fluid present when the deposition of the urate of soda first occurs in the articular cartilage ?

The bursæ have still to be mentioned as parts which are very liable to receive deposits of urate of soda in gout. This is particularly the case with those over the olecranon, which may become enlarged until they are almost as big as oranges.

Sometimes when these tophi are large, the skin wears through. Nodules in the ears may in this way be cast off, and the patient be freed from their presence. Sir Thomas Watson tells the story of a gentleman who had exposed tophi on his fingers, and who used, when playing at cards, to score the game upon the table with his gouty knuckles.

Another change to which tophi are liable is the occurrence of suppuration in the tissues round them. And in connection with this subject it may be remarked that although a joint affected with acute gout often looks so inflamed that one might imagine it ready to point, yet as a matter of fact this never happens, nor is pus ever formed. But, on the other hand, abscesses round the extra-articular deposits in chronic gout are very common, and the lithate of soda is freely discharged from them mixed with pus. Garrod speaks of as many as five or six abscesses of this kind being open at one time on each hand, and others on the feet, and he remarks that they give rise to very little constitutional disturbance.

Formerly it was universally supposed that deposits of urate of soda were to be found only in advanced cases of gout, and, indeed, that their presence was somewhat exceptional. Sir Charles Scudamore asserted that they did not occur in one gouty case in ten. This, however, is true only so far as concerns masses of the urate deposited outside the articulations, and large enough to be seen and felt. There is reason to believe that within every joint affected by the disease urate of soda is always present in greater or less quantity. Garrod mentions the cases of two patients, of whom one had only a single attack of gout thirteen years before his death, while the other had had two attacks within the last two years of his life. In each case a small quantity of urate of soda was found as a white deposit upon the surface of the joints which had been affected by the disease.

Pathology.—In considering the theory of gout we must start from the fact that the blood in this disease contains an excess of uric acid.

Garrod has not only shown that uric acid can be detected in the blood in gout by an elaborate chemical analysis, but also that for clinical purposes a simple method of determining its presence may be employed. About two drachms of the serum of blood are put into a flat glass dish, somewhat larger than a watch glass, and acetic acid is added so as to give a slight acid reaction. A fibre from a piece of linen cloth is then placed in the fluid, and

the dish is set aside until its contents have acquired a gelatinous consistence by evaporation. If the blood contains uric acid in excessive quantity, it is deposited upon the fibre, and this becomes studded with crystals, the characteristic appearance of which can readily be identified with a pocket lens. The serum from a blister will also yield crystals of uric acid when examined in this way, but only when the blister is placed at a distance from a joint affected at the time with gouty inflammation. Garrod states that the acid cannot be detected in the fluid from a blister in the neighbourhood of a gouty articulation, and he infers that gouty inflammation causes a local destruction of uric acid. However this may be, it appears to be certain that an attack of gout has in some way the effect of clearing the blood from its impregnation with the acid. In patients who had partially recovered from an acute seizure, a very marked decrease in its quantity was found by Garrod; and, indeed, in the intervals between the early attacks of gout he failed to detect any appreciable amount of lithate in the blood. This tendency, which gouty inflammation possesses, of freeing the blood from the lithic acid which had accumulated in it, accounts for a fact which appears to be well established, and which certainly needs explanation, namely, that an attack of acute gout often leads to the rapid disappearance of certain other symptoms from which the patient had before been suffering. The symptoms in question are those which are commonly described as "irregular" gout, the ordinary affection of the joints being, on the other hand, spoken of as "regular" gout. Other epithets sometimes used instead of "irregular" are "atonic," "latent," "lurking," "masked," and "misplaced." Most of the symptoms have already been described as effects of lithæmia. Garrod has demonstrated by direct experiment the fact that uric acid was present in the blood "in some cases where symptoms of irregular gout were present without any accompanying joint disease."

The next question is, what is the distinction between a state of lithæmia and one of gout? To this question also Garrod gives an answer. He shows that lithate of soda is invariably present in a gouty joint. Still more original is his suggestion that the salt is deposited, not as the result of the inflammation, but before it occurs—that, in fact, the presence of urate of soda in a joint is a condition antecedent to an attack of gout, and very probably its cause.

In the pinna of the ear, in fact, gouty concretions commonly form without any indications of previous inflammatory action. In some cases the patient experiences sensations of heat and pricking and the part is tender, but more often he is quite unconscious of the fact that such concretions in the pinna are present. Judging from the observations which have been made by Dr Moxon and the author in the *post-mortem* room of Guy's Hospital, one would infer that in the interior of joints also the deposition of urate of soda sometimes takes place no less slowly and with the same absence of symptoms. At any rate, we have found the articular cartilages of the great toe joints encrusted with the salt in many cases in which no mention of gout had been made during life. So that perhaps the occurrence of a definite gouty seizure is often more or less of an accident in the course of an essentially chronic process.

This, however, is not exactly Garrod's view. He states that among a large number of autopsies on persons who are not known to have gout, there were only two in which even a slight trace of urate of soda was found

in the great toe joints. He supposes that the deposition of the salt in a joint is always the immediate precursor of a gouty seizure.

There are two conditions which may be supposed to cause a rapid increase in the lithate of soda in the blood, and so lead to its deposition in the joints and to provoke an attack of gout: namely, (1) the ingestion at a particular time of such food as "disorders the liver" and produces lithæmia, and (2) the failure of the kidneys to excrete lithic acid.

The first of these conditions is well known to be a frequent exciting cause of a gouty seizure. Scudamore mentions the case of a gentleman without any hereditary tendency and with no reason to suspect that he would be attacked, who was seized with gout for the first time after three or four days of excessive conviviality. Another striking case is that of a gentleman who had never had gout in the summer and who, persuaded of his security, drank six or seven glasses of champagne; in twelve hours he had a fit of the gout. In three instances patients had sat down to dinner with scarcely the sensation of gout, but when rising to leave the table found themselves completely disabled.

Garrod, however, believes that in many cases the other condition (that of the kidneys) is really operative. He found that in severe cases of acute gout the amount of uric acid contained in the urine was on an average less than four grains daily—the normal amount being eight or ten grains. It is true that in such cases the urine as it cools often deposits lithates, which are of a bright pink or red colour such as has generally been supposed to be characteristic of lithæmia, and to indicate an excessive secretion of these salts. But this conclusion is fallacious; for, first, the quantity of urine passed in the twenty-four hours is diminished, and secondly, its acidity is much increased, so that the whole of the lithates are deposited. As gout becomes more and more chronic, uric acid is excreted by the kidneys in diminished amount even in the intervals between the attacks, and in advanced stages of the disease it may be entirely absent from the urine. But the *blood* in chronic gout always contains excess of uric acid.

It still remains to inquire why one part of the body more than another should be selected for the deposition of the crystalline lithate of soda. Garrod's hypothesis is that the crystals are formed especially in those parts which are not vascular or in which the circulation is sluggish. This applies both to the pinna of the ear and to the articular cartilages, and in reference to the former he says that persons with cold ears are most apt to have gouty concretions in them. In the knee-joints, too, the parts which are in contact with the vascular fringes remain free.

The reason why the metatarso-phalangeal joint of the great toe should be attacked by gout in preference to all other joints is supposed by Garrod to be the fact that it is subjected to so much pressure from the weight of the body, and also to sudden shocks. When any other joint has been previously injured it is frequently made the first victim of a gouty seizure.

For some reason it has become common to ascribe bronchitis, dyspepsia, gastralgia, iritis, gravel, cystitis, and urethritis, phlebitis, eczema, and even psoriasis to a gouty diathesis. But the evidence is very slight, and the "gout" to which such evidence as there is applies, is the destillation of morbid humours which belongs to a bygone pathology, not deposit of urate of soda in the tissues. There is no reason to believe that gout ever "flies to the stomach," but over-indulgence at the table may produce acute dyspepsia as well as inflammation of the great toe; elderly people are liable to gravel,

gout, stone, and cough; and while lead and drink may lead to gout and chronic Bright's disease, cirrhotic kidneys favour an attack of gout.

Nevertheless it appears probable that the deposition of lithate of soda causes inflammation in other tissues beside the joints; it does, as we have seen, in the ear and occasionally in the skin, and it or the preceding lithæmia may possibly excite neuritis of the sciatic nerve, or inflammation of a varicose vein, or of the conjunctiva or the bronchia. The proof, however, must be given in each case.

Ætiology.—In the first place, the disease is decidedly *hereditary*. Garrod found that more than half of all his gouty patients could prove an inherited disposition to this disease; and he says that among persons belonging to the upper classes the proportion is considerably greater.

Sir Charles Scudamore found among 522 patients suffering from gout, 190 in whom no hereditary taint could be traced. Of the remaining 332 patients, 181 inherited it from the father, 59 from the mother, and 24 from both parents, while the other 68 had grandparents (44 cases), or uncles (21) or aunts (3) who had suffered from the same disease.*

Mr Jonathan Hutchinson ('Med. Times and Gaz.,' 1876) believes that the younger children in a family are more likely than the elder to suffer, and to suffer severely. The popular idea that the disease often misses a generation is probably unfounded.

There is a particular *age* at which a first attack of gout is especially apt to occur, namely, between thirty and thirty-five years of age. In persons less than twenty years old the disease is very rare; but Garrod says that he has seen it in the great toe in youths of sixteen. In early cases there is always a strong hereditary predisposition, and the habits of the individual are also generally such as favour its development. Scudamore mentions two cases in boys of eight and twelve, but neither cases were seen by him and they were possibly not genuine. His youngest patient was fifteen. Sydenham said, "I have not hitherto found children or very young persons affected with the true gout." The aphorism of Hippocrates is still true: *παῖς οὔ ποδαγρικὸς πρὸ τοῦ ἀφροδισιάσμου* (vi, 30).

Beyond the age of sixty-five a first seizure seldom occurs; Scudamore had never seen a case, but Garrod records one at eighty and another in a woman nearly ninety. Among sixty-one cases collected by the editor, the first attack occurred once at seventy, and one of the worst cases of (hereditary) gout was in an abstemious young woman of twenty-one.

Gout is much more common *in men* than in women. This doubtless depends mainly upon the fact that their habits are more frequently such as tend to develop the disease. The occurrence of the catamenia during a large part of female life perhaps also assists in warding off gout, for it seldom attacks women until after the cessation of the menstrual function.† Among seven female patients with gout at Guy's Hospital, in three it was hereditary and in three it was caused by drink.

High living, with excessive indulgence in *animal food*, is generally supposed to be a cause of gout.

* The hereditary factor in gout seems to have been unknown to the ancients. It is clearly stated in Pirkheimer's 'Apologia seu Podagræ laus,' published at Nürnberg in 1522, and afterwards translated into German (1577) and into English ('Praise of the Gout,' translated by W. Est, Lond., 1617); and it was made part of the definition of true gout by Cullen.

† Here again the aphorism of Hippocrates holds: *Γυνὴ οὐ ποδαγρικὴ ἣν μὴ τὰ καταμήνια αὐτέῃ ἐκλίπῃ*, vi, 29.

As regards the power of *intoxicating drinks* to produce gout, there is no doubt whatever; but all liquor does not act equally to this effect. The fact that malt liquors are more apt than ardent spirits to produce gout is well shown by comparing the working men of London with those of Edinburgh and Glasgow; the former drink beer and porter and are very liable to the disease; the latter drink little but whisky, and although they are by no means sparing of this, they scarcely ever have gout. The rarity of gout in many of the cities on the Continent, where distilled spirits form the chief intoxicating beverage, is another proof that they have but little tendency to produce it. Garrod says that even the pale bitter ale may, if taken freely, be the sole cause of the complaint. With regard to the different kinds of wine, he recognises the special danger of indulgence in port, champagne, Burgundy and Madeira; and he also gives a caution against sherry—however dry and pure—which he says is by no means so innocent as many people imagine. Cider, according to this writer, when sweet and partially fermented, is apt to cause gout; but rough cider is comparatively harmless. With regard to the causes which render one kind of alcoholic drink more injurious than another, no positive conclusion can as yet be drawn. Their comparative effects in causing dyspepsia, and on the other hand in leading to free diuresis, are probably important.

A fact which Sydenham long ago observed, in his description of gout, is that the first attack generally occurs in the *winter season;* he says towards the end of January or the beginning of February. Why this should be the case is not very clear; but it is well known that deposits of lithates in the urine are very apt to occur in cold weather. In many patients the disease returns for two or three years in the spring only; after a time, a second attack occurs in the autumn; and at length the seizures occur quite independently of the season.

The influence of cold in the development of gout is shown by the effects of change of *climate.* Garrod says that a gouty man may often escape his accustomed winter attacks by going to Malta or Egypt. In hot countries, this disease appears to be very rare, or unknown. Even in the south of Europe it is much less frequent than in England; but we must remember that the habits of the people of different countries are very different.

Certainly the disease seems to have been more common in ancient Greece and Rome than in modern Italy, Spain, and France. Among sixty-four autopsies at Vienna, the editor only once saw a case of gout, and the deposit in the joints was not recognised by anyone but Rokitansky himself. It is believed to be more common in Paris and Berlin than it used to be, and also in the United States. It is said to be rare in Russia and Scandinavia, notwithstanding cold and alcohol; and to be more frequent in Holland and Flanders. It is undoubtedly less common in Scotland and Ireland than in England. Pagenstecher speaks of it with respectful envy as the heritage of "die reichen Söhne des gesegneten Albions."

A remarkable fact in the causation of gout has been established by Garrod, namely, chronic *poisoning by lead.* He found that about 30 per cent. of the gouty patients in his hospital practice had been exposed to the influence of lead. Some of them were painters and plumbers, others workers in lead mills, and others "composite-doll" makers. A careful inquiry into the habits of the men failed to show that they had been less temperate in their habits than other men of the same class. That this is a true cause is sufficiently established by everyday hospital experience in

London, but the way in which lead produces gout is at present doubtful. Probably the explanation of this fact involves that of another which Garrod believes he has established: that persons who are already gouty are more susceptible than others to be affected by lead. He says that in several instances he has found those patients to be of gouty habit, or to have already had severe attacks of gout, in whom the medicinal administration of the preparations of lead has produced colic or a blue line upon the gums with unusual rapidity. Possibly the excretion of lead in the urine leads to irritation of the kidneys, and thus to chronic Bright's disease, and retention of uric acid. Dr Lorimer, of Buxton, has published an account of 107 cases of saturnine gout ('Brit. Med. Jour.,' July 24, 1886). He finds that it usually befalls patients earlier than hereditary or than alcoholic gout, that the fits are less severe but more lingering, and that albuminuria is almost constantly present. (See also Wilks in the 'Guy's Hosp. Rep.' for 1870.)

An occasional exciting cause of gout is *mental fatigue or anxiety.* Sir Charles Scudamore mentions the cases of two female patients, in each of whom a severe first attack of gout was brought on by sitting up for several nights in succession, nursing a sick relation. Severe intellectual labour, watching, sorrow, and exhaustion from sexual excesses, may each be the occasion of a fit of gout.*

Diagnosis of gout is in some cases very easy, in others exceedingly difficult. According to certain writers, the difficulty in determining the real nature of a case in which the joints are swollen and painful arises from the occurrence of inflammation intermediate between gout and rheumatism; or from the existence of both. But there is in reality no *tertium quid.* Either the affected joint contains urate of soda or it does not: in the former case the disease is gout, in the latter it is not. In the *post-mortem* room doubts never arise as to the gouty nature of disease in a joint. As for the simultaneous presence of gout and rheumatism in the same patient, its theoretical possibility must be admitted, but practically it does not occur oftener than coincidence of typhus and enteric fever, or of measles and scarlatina, or of syphilis and psoriasis. The term "rheumatic gout" is either a bad name for osteo-arthritis, or is a mere excuse for shirking a sometimes difficult diagnosis.

The following characters suggest an affirmative answer to the question whether a case is one of gout. (1) That the small joints are affected, and particularly the great toe joint; (2) that the attack began suddenly in the night, especially if it be a first attack; (3) that the skin over the affected joint is tense, shining, and red, with distended veins around it, after a day or two œdematous, and finally desquamating; (4) that the febrile disturbance is moderate, and in proportion to the local inflammation. The sex, age, and family history of the patient have also to be taken into account; and the state of the heart and kidneys must be investigated. All those parts which are apt to be the seat of tophi should be examined. If a single deposit of urate of soda can be found, it settles the question. But care must be taken not to mistake other kinds of enlargement of the finger-joints for those caused by gout, and it is needful to distinguish from tophi

* The ancients laid stress on the latter exciting cause:

"Λυσιμελοῦς Βάκχου καὶ λυσιμελοῦς Ἀφροδίτης
Γεννᾶται θυγάτηρ, λυσιμελὴς ποδάγρα."

in the pinna of the ear comedones of the sebaceous glands, and that occasional nodule in the edge of the helix which Darwin discovered.

It must not be forgotten that there are other diseases besides rheumatism which may be mistaken for gout. Garrod mentions a case in which the great toe was swollen, tense, red, and hot; in which, in fact, the joint looked exactly as though it were affected with severe gouty inflammation; but the disease turned out to be *pyæmia*. The diagnosis from osteo-arthritis and gonorrhœal synovitis will be best considered in the following chapters.

Course and prognosis.—Acute gout in the joints appears never to be directly fatal; a prognosis is required with regard to the ultimate effect on the health of the patient.

It was once deemed rather an honour than a misfortune to have the gout; it showed that not only the man himself, but perhaps his father and grandfather before him, had been able to afford good living. Sydenham himself, after suffering for thirty-four years, speaks of it as a comfort that gout, unlike any other disease, kills more rich men than poor, more wise men than simple. "Great kings," he says, "emperors, generals, admirals, and philosophers have all died of gout."

At the present day any consolatory reflections of this kind may be checked by the consideration that all insurance offices charge an additional premium to anyone who has had even a single attack of gout. Their actuaries know well that the disease tends to shorten life. However slight it may have been, a seizure of gout is always an admonition that the patient's habits of life are incompatible with the preservation of sound health. Sir Thomas Watson says that "in not a few instances men of good sense, and masters of themselves, having been warned by one visitation of the gout, have thenceforward resolutely abstained from rich living and from wine and strong drinks of all kinds; and they have been rewarded by complete immunity from any return of the disease; or at any rate, its future assaults upon them have been few and feeble." "I am sure," he adds, "it is worth any young man's while, who has had the gout, to become a teetotaler." For the earlier the age at which a first seizure occurs the worst is the prognosis; and particularly if there be an inherited predisposition. Garrod says that he has known thirty-five years elapse between a regular attack of gout in the great toe and the patient's death, which latter event took place when he was seventy years old. He has also seen several cases in which the disease, after having recurred periodically for many years, gradually declined in intensity and duration, and at last altogether disappeared.

The repetition of attacks of gout is a serious matter, if only on account of the crippled state of limbs which it induces, preventing the patient from taking exercise, and destroying his enjoyment of life.

But the main risk connected with the gouty condition is its liability to induce "chronic interstitial nephritis," or renal cirrhosis, so that Dr Todd called the "small, red, contracted kidney" the "gouty" kidney. When the disease is caused by gout, white streaks are often seen, running in the course of the straight tubes in the pyramids; these white streaks consist of prismatic crystals of urate of soda and of amorphous masses, blocking up the tubes, and also embedded in their walls.

The signs by which this affection of the kidneys may be detected have been fully discussed already (p. 643). A prolonged examination of the urine, both chemical and microscopical, is often necessary. But merely looking at the secretion may be enough to excite suspicion. Sydenham

remarked that in cases of long-standing gout the urine, "no longer high coloured, is pale and copious, like the urine of diabetes." He did not understand the significance of this, but we now know that it points to the fact that the kidneys are diseased. We also know that hypertrophy of the heart is an almost constant attendant upon this form of renal affection, and that apoplexy frequently results. The slightest indication of cerebral mischief must therefore be watched very carefully in such cases, and it is often right to warn the patient or his friends that he should not be left alone at night, nor while out of doors.

Evidence of Bright's disease was found in a third of the editor's sixty-one cases. In many of them atheroma was present, and it also has been called gouty. The calcareous deposit, however, contains no urate of soda.

Bronchitis in gout may be arthritic or renal or senile; not infrequently it proves the immediate cause of death in long-standing cases.

It has been said of gout, as of cancer, that it is incompatible with phthisis This, however, is not the case. The fact that they affect different periods of life, so that few have gout who die under thirty, explains their seldom being met with together, but in the small number of sixty-one cases above quoted, three times the ordinary symptoms of phthisis occurred in patients with well-marked gout. In another case of gout tubercles were found after death in the lungs, and at the present time (February, 1888) a patient in Philip Ward with unmistakeable tophi is also the subject of phthisis of the larynx and lungs.

Treatment.—Gout is one of the few diseases for which modern medicine has found a specific remedy. When Sydenham wrote he spoke of the possibility of such a discovery, which he said would delight him above all other physicians; but he knew of no specific for gout. Cullen, after him, advocated patience and flannel alone. The ancients had recommended hellebore and hermodactyl; and the latter plant has been identified with *Colchicum autumnale*, and with *Iris tuberosa*. Towards the end of the last century a secret remedy for the disease became widely known under the name of the *eau médicinale de Husson*. Its success was unmistakeable, and it was at length discovered that the plant used in making it was meadow saffron or colchicum. Since then, however, there has been much discussion as to the use of colchicum in gout. Perhaps it seemed to derive a taint from its introduction as a quack nostrum. At any rate, many writers have depreciated its value. Some have asserted that when it acts beneficially it is only by purging; others that even when it removes the local symptoms it leaves "the disposition to the disease much stronger in the system." This was the opinion of Scudamore, and Trousseau advised his hearers to cross their arms and look on, doing absolutely nothing to subdue an attack of acute gout.

Very different from this is the teaching of Watson and of Garrod. The former writer prescribes forty minims or a drachm of the colchicum wine in a saline draught at bedtime, and half a drachm more in a warm draught the next morning, repeating the sequence if the disease continues. The latter gives about twenty minims of the wine every six hours. Both say that the effect is almost magical; the pain is calmed, and the swelling reduced often within a few hours.

It is certain that the curative action of colchicum is not dependent upon its purgative operation; for it is often effectual when it does not act upon the

bowels at all. Sometimes, however, this remedy produces a peculiar change in the fæces, which Garrod says may enable a patient accustomed to its use to tell that he is taking it.

It is probable, as Sir Thomas Watson observes, that the striking efficacy of colchicum in attacks of gout has often led men to disregard those precautions of regimen and diet by which alone the disease can be kept at bay. Having what they deemed a specific, they have cast aside all restraint, and before long they have had a fresh seizure.

Alkalies, again, appear to be decidedly useful in gout, and they may often be advantageously given with colchicum. Probably the best way in which these remedies can be given is in the form of salts of citric, tartaric, or acetic acid; and in the hope that the base may combine with uric acid and lead to its elimination by the kidneys it is best to prescribe potass rather than soda; for the urate of potass is much more soluble than that of soda, and potass also possesses greater power as a diuretic. The salts of lithia were introduced by Garrod as possessing some advantage over those of potass in the still greater solubility of the urate of that base; perhaps this gain does not outweigh the disadvantage of their higher price. The carbonate of lithia may be given in doses of five to ten grains dissolved in aerated water, or the citrate in doses of eight to twelve grains or more. Whatever alkaline salt is prescribed, it should be largely diluted with water, and taken at least an hour before meals,when the stomach is empty.

Sarcosin, which diminishes the excretion of uric acid, has been recommended on theoretical grounds as a remedy for gout by Schultzen.

The *diet* in an attack of acute gout should for the first few days consists of milk, arrowroot, sago, tapioca, and the like, with water or toast and water. A little brandy may be given if the previous habits of the patient render it necessary, but not otherwise. It must, however, be added that even a first seizure of gout sometimes occurs in a patient so broken down in constitution as to require as much nourishment as the stomach can readily dispose of, such as beef-tea, soup, and eggs.*

The *local treatment* of gouty joints is not very effectual. They may be covered with flannel or cotton wool; or, as Garrod recommends, oil-silk or gutta-percha sheeting may be carefully applied, so as to keep in the moisture exhaled from the skin, and form a kind of vapour bath. A solution of atropine and hydrochlorate of morphia may be used as a sedative, in the proportion of one grain of the former and eight grains of the latter to the fluid ounce, applied on a small piece of lint beneath the oil-silk. A lotion containing a drachm of the spiritus ætheris sulphurici to six ounces of water has sometimes proved serviceable. Scudamore recommended a lotion composed of one part of alcohol and three of camphor water, made agreeably lukewarm and applied on thick linen compresses.

Garrod gives a strong caution against the application of leeches to joints affected with gout. He has seen irremediable injury from their use: great toe joints, stiffened after a few attacks, and two cases in which the patients completely lost the use of both knees. Hot poultices, again, are said by Scudamore to be very injurious.

Another plan of treatment, which was followed in his own person by no less an authority than Harvey himself, but which appears certainly to be mischievous, is the application of cold. Sir Thomas Watson mentions that

* "Potus theæ et caffe inter reliqua remedia calculosis et podagricis excellunt."—Baglivi: Prax. Med., lib. i, p. 117.

Dr Parry, of Bath, had at one time two patients under his care, each of whom had attempted to cut short or to ease a paroxysm of gout by plunging the affected part into cold water. This gave instant relief to the pain, and the inflammation presently abated, but in each case hemiplegia occurred a few hours afterwards. Trousseau relates a case that occurred to Dr Demarquay of a gentleman who applied cold water compresses to his foot, which was affected with very severe gout. The pain was almost immediately relieved, but a few hours later the patient fell into a state of apoplectic semi-stupor, which disappeared under the use of sinapisms to the foot, and consequent return of the articular inflammation. These cases certainly resemble one another so closely that it is difficult to believe that the cerebral disturbance was a mere accident.

The *treatment of chronic gout* must be in some respects different from that of an acute attack. But it is a great mistake to suppose that colchicum is useless, even when the joints are deformed and crippled.

Other remedies which are very useful in certain cases of chronic gout are guaiacum and iodide of potassium. Garrod says that he has given the former of these medicines extensively, especially for the asthenic gout of old subjects; a patient may go on taking it for a whole year at a time. The latter is particularly useful when the pains are increased at night by the heat of the bed; and also when the joints contain fluid of which the absorption is slow. Bark and quinine are also useful in some cases.

Certain *baths and mineral springs* have so great a reputation for the relief of gout that there are few patients, among the richer classes, who do not sooner or later visit one of them. Of such resorts the chief is Vichy; the main ingredient of its springs is carbonate of soda in the proportion of about forty grains to the pint. Another very similar water is that of Vals. Many persons who are robust and of full habit derive much benefit from these springs; but to feeble patients they often do harm. Garrod says that in very chronic cases a tendency to the formation of concretions about the joints has appeared in his experience to be hastened by the use of Vichy water. Trousseau advised that as a general rule alkaline waters should not be taken for more than ten or twelve days at a time, and only in small quantities, for not a year passed in which he did not see evil consequences from their prolonged use. Mineral waters should never be taken when an acute attack is present or threatening; nor by patients who have organic disease of the heart or kidneys.

For patients with symptoms of hepatic disorder, the waters of Carlsbad and the other springs mentioned under lithæmia (p. 491) are often very useful. When the circulation is sluggish or the secretions deficient, Wiesbaden is indicated, the water of which contains a large amount of chloride of sodium; or Aix-la-Chapelle. Old or infirm persons may be sent to Bath or Buxton, where the waters are hot and the active saline ingredients small in quantity; or Teplitz or Gastein may be chosen, if more distant places are preferred.

RHEUMATISM*

Definition—Onset, course, and symptoms—Anatomy of the inflamed joints—The pyrexia, sweating, &c.—erythema—cardiac complications—pleurisy, tonsillitis, nodules—Ætiology and pathology of rheumatism—Prognosis: fatal cases—Rheumatic hyperpyrexia—Diagnosis, particularly from pyœmia—Treatment by older methods—Observation of the natural course of the disease—Treatment by salicin and salicylates—Treatment of hyperpyrexia in rheumatism.

Definition.—By ancient writers, from Hippocrates downwards, the words *rheuma* and *catarrh* are used as having similar meanings, their etymology being also alike, since the one term was derived from ῥέω (I flow), while the other came from the same verb with the prefix κατὰ. The notion was that of an acrid humour, generated in the brain and distributed over the body. In the course of time, diseases of the mucous membranes became known as catarrhs, while the name of rheumatism was applied to painful affections of the limbs and elsewhere, on account of their being so often of an erratic character. Baillon or Ballonius, who wrote in 1642, is said by Bright and Addison to have given it this meaning, and he also distinguished it from gout, or *gutta*, another name witnessing to the humoral doctrines of Galen. *Destillatio* was a term applied in common to catarrh, rheuma and gutta. The conception of rheumatism became modified in some countries so as to include the notion of its production by external cold. All sorts of complaints have accordingly been termed rheumatic; some, of which the cause was unknown because they were painful; others, although painless, because cold was the supposed cause.†

But there is a very common, definite and important acute disease which was first accurately described by Sydenham in 1670, and for which we have no other name than rheumatism or rheumatic fever. We will include under the same name genuine cases though their course may be "subacute," for they have no claim whatever to a separate designation.

With regard to the so-called "chronic rheumatism" there is more difficulty. We shall describe separately the complaint which is known as *osteo-arthritis* or *arthritis deformans*, and which probably bears no relation whatever to acute rheumatism. But there are other cases in which a chronic joint-affection has clearly arisen from, and can only be described as a sequel of, the acute disease; and it is impossible to deny that a similar affection may, as a rare exception, be chronic from the first, and yet be of the same nature.

The use of the adjective "rheumatic" ought, of course, to be limited in precisely the same manner as that of the substantive; but in practice it is often employed loosely for various affections of the muscles and of other parts, of which the only common character is that they are caused by cold. Nevertheless, it is much better to speak of "myalgia," than of "muscular

* *Synonyms.*—Rheumatic fever—Acute and subacute Articular Rheumatism—Arthritis vaga—Polyarthritis rheumatica acuta—Rheumarthritis.

† This confusion is still common with good German writers, so that it is often as difficult to know what is meant by "rheumatic" as to know the meaning of "typhus."

rheumatism;" for the common vague use of the term implies a connection, of which there is no proof, between "rheumatic" affections and true rheumatism.

Onset and course.—The beginning of the disease is commonly rather gradual. For a day or two the patient feels uncomfortable, and perhaps complains of pains in the limbs. There is seldom headache, and still more rarely rigors or vomiting. Pyrexia gradually sets in, and has commonly reached its height on the second day. Presently one of the joints, generally a large joint, is found to be swollen as well as painful. In many cases inflammation of the joint is the first thing noticed.*

This affection, which is characteristic of rheumatism, is synovitis, acute or subacute, serous, comparable to pleurisy, but not leading to adhesions, nor ending in suppuration or destruction of the tissues.

It is not easy to determine clinically the fact that fluid is effused into the interior of the shoulder-joint, the elbow, or the ankle; for there may be a more or less elastic swelling at those points where the synovial membrane is most able to yield, and one may hesitate to assert absolutely that it cannot be due to exudation into the sheaths of tendons or into bursæ. But in the knee very small quantities of fluid may be detected with certainty. One has only to grasp it gently between the two hands, pressing the sides of the synovial cavity upwards, so that the fluid may accumulate beneath the patella; a slight tap upon this bone with one forefinger will then bring it down upon the condyles, giving a sensation which is unmistakeable. In those exceptional cases which prove fatal all the joints which have been affected are found at the autopsy to show distinct signs of inflammation. The synovial membrane is not, indeed, always injected, but there can be no doubt that in this, as in so many other tissues, redness may subside after death; and a considerable quantity of fluid is almost always still present. This is often cloudy, with floating shreds of fibrin. Not infrequently a separable layer of lymph lines the synovial membrane, which is then very deeply reddened; or it may cover the surface of the cartilages, as, for example, in the knee-joint. Leucocytes seem to be always discoverable in the exudation, but not in numbers to produce the appearance of pus. In one case which was examined by Dr Moxon the sheaths of the extensor tendons of the wrist were full of opaque serum and of masses of greenish-yellow lymph. Articular cartilages have in rare cases been found eroded, but in all probability this was accidental and unconnected with the rheumatism.

That the anatomical changes in the joints in this disease must be slight in comparison with those which occur under other circumstances might, indeed, be inferred from its clinical history. One of its most striking features is the rapidity, and even suddenness, with which it flies from one part to another. Thus a joint which is extremely swollen one day may be quite normal on the next day; and a little later it may be again affected as severely as before. There is not always any obvious reddening of the skin; generally some ill-defined pink patches or striæ are seen, but they are often

* Some years ago Dr Chambers, of St George's Hospital, and Dr Francis Hawkins endeavoured to draw a distinction between two varieties of acute rheumatism, one of which they termed "fibrous," the other "synovial," and their views were adopted in Sir Thomas Watson's 'Lectures.' They were, however, altogether mistaken in supposing that the disease ever attacks the parts outside a joint rather than the articular cavity itself, and the clinical distinction they drew, so far as it is real, corresponds to that between true rheumatism (fibrous) and osteoarthritis (synovial).

near the articulation rather than over it. Even if, as occasionally happens, the joint shows a redder surface, which looks like gout, the surrounding veins are not dilated.

The patient dreads the gentlest touch or the jar of a passing footstep; with his head and limbs immoveably fixed he turns his eyes with anxiety as a stranger approaches his bed. He lies perfectly helpless, unable to feed himself, or even to turn in bed; if the joints of the lower jaw are affected he can scarcely open his mouth. Hospital patients often speak of having lost the use of their limbs, or of being paralysed. The pain is usually worse by night than during the day.

Garrod remarks that the different joints are often attacked in symmetrical order: the right ankle, then the left; the right knee, then the left; and so on. But in many instances the distribution of the disease is quite irregular. All four limbs suffer usually more or less. The knuckles and other small joints are often affected, but perhaps never without some one of the large joints being also attacked. According to Lebert, acute rheumatism sometimes attacks the synchondroses, as, for instance, those of the pelvis.

In some exceptional cases rheumatic inflammation of a joint, instead of subsiding, passes on into a chronic stage, and ends in hydrops articuli, most likely of the knee. Ankylosis or disorganisation of the articular structures is so extremely rare that its occurrence throws doubt on the diagnosis of rheumatism.

There is no evidence whatever that rheumatic inflammation attacks any but synovial and serous membranes. It has no predilection for fibrous tissues, whether the fibres be white, elastic, or muscular.

The *pyrexia* of acute rheumatism is in the majority of cases severe in proportion to the number of joints involved and to the intensity of the inflammation in them. But to this rule there are many exceptions. Sometimes the articular affection is well marked while the temperature is scarcely raised at all. In other cases there is considerable fever, whereas very few of the joints suffer, and these but slightly. Indeed, as Graves long ago pointed out, it is possible for the fever to occur alone, without any joint-affection at all. The case which he cites was one of relapse, the patient having previously passed through two attacks of the usual kind; but the converse may occur,—an attack of pyrexia, with pericarditis or endocarditis, or perhaps even with pleurisy alone, may be followed by a relapse which is attended with synovitis, and thus reveals its real nature. It is therefore certain that acute rheumatism is a *general* disease with localisation in the joints, not a local disease of the joints with symptomatic fever. Yet even Wunderlich was unable to recognise any typical course; the maximum temperature, which is usually about 104° F., sometimes occurs as early as the fourth or even the second day, but more often not until the end of the first or the beginning of the second week. The *pulse* is often extremely rapid, and is commonly large, full, and bounding. The breathing is not proportionably hurried. The *tongue* is generally flabby, marked by the teeth at its sides, and coated with a thick, white fur. There is usually thirst and anorexia, but the patient can sleep if not kept awake by the pain. He is seldom delirious unless his temperature runs very high, or unless he has been a drunkard, in which case a form of delirium tremens is often developed. There is no tendency for the fever to assume a "typhoid" character.

The *urine* is characteristically febrile, scanty, dense, high coloured, acid,

depositing pink urates in abundance, and often crystals of uric acid; occasionally it contains a trace of albumen for a day or two.

One of the most striking symptoms of acute rheumatism, though not invariably present, is *sweating*. The patient lies bathed in water, so as to make even the blankets damp. This must, of course, carry away much heat by evaporation from the surface of the body, but it has no obvious influence in lowering the pyrexia or in relieving the articular pains. The perspiration has usually a very sour smell, which is often taken into account in diagnosis. This, however, seems to be due to the quantity of sweat rather than to its containing an unusual quantity of free acid. Sir William Gull used to point out that the reaction to litmus paper often varies upon different parts of the skin, being acid, alkaline, and neutral in the same patient. Sweat when first secreted is not acid and only becomes so by decomposition of the sebaceous compounds mingled with it. Senator suggests that the sweat may become alkaline by conversion of urea into carbonate of ammonia, particularly between the toes and in the armpits.

There is frequently developed a copious eruption of *sudamina*—minute transparent vesicles, containing an acid fluid which is no doubt sweat. They glisten when a bright light falls upon them, but they are colourless, so that they often can more easily be felt than seen. In many cases, however, their bases become slightly inflamed and reddened, and their contents opalescent and alkaline: at this stage they are sometimes called *miliaria*. This eruption bears no special relation to acute rheumatism, for it occurs in other diseases attended with sweating, and also in healthy people during hot weather.*

In some cases of acute rheumatism there is another very remarkable eruption, the characters of which vary rather widely, but which generally assumes the form of erythema, urticaria, or purpura. It has often been described under the name of *Peliosis rheumatica*, originally invented by Schönlein. Dermatologists have broken it up into several varieties, which they have termed "Erythema papulatum," "E. tuberculatum," "E. circinatum," "E. marginatum," &c., according to the appearance it happens to present; and another modification has been named Purpura urticans. The best name is that proposed by Hebra, "*Erythema multiforme*;" and it will be described under that name in the chapter on diseases of the skin.

Cardiac complications.—In every case of acute rheumatism it is necessary from day to day to watch with the stethoscope for indications of inflammation of the pericardium or of the valves of the heart, for these affections often give rise to neither pain nor dyspnœa nor any other symptom. The frequency of inflammation of the heart in acute rheumatism cannot be stated with numerical accuracy. The results of *post-mortem* examinations

* In this connection it is perhaps worth while to mention the "febris miliaris" which occurred as an epidemic on the Continent, and especially in France, throughout the eighteenth century and down to the year 1856; it was attended with a red papular or vesicular rash. The name is still used in Italy, but probably for scarlatina, enteric, and other febrile diseases attended by sweating and sudamina. In the English "sweating sickness," of which there were five terrible epidemics between 1485 and 1551 in this country, there seems to have been no such eruption; at least, Dr Guy makes no allusion to it in the graphic description of that disease in his work on public health.

See the account given by Dr Kaye (the founder of Caius College) in his tractate 'De Ephemera Britannica.' The years of the invasion of this serious epidemic were 1485, 1506, 1517 (the worst, according to Stowe), 1528, and 1551. Additional facts will be found in Hecker's 'Epidemics of the Middle Ages,' translated by Dr Guy Babington, and in Hirsch's work translated by Dr Creighton. It is believed by Dr Payne that this curious malady still lingers in an endemic form in Picardy.

are not available for the purpose, because cases which prove fatal are more severe than the average; and the auscultatory signs which afford our best means of detecting cardiac complications during life are open to sources of fallacy, particularly the difficulty of distinguishing between organic and functional murmurs. Probably, however, we shall not be far from the mark in estimating that about 50 per cent. of all first cases of rheumatism are accompanied by pericarditis or by endocarditis, or by both. Among forty-five cases which ended fatally at Guy's Hospital, and in which there had been no previous chronic disease of the valves, the heart was healthy in eight only, both pericarditis and endocarditis existed in nineteen, pericarditis alone in ten, endocarditis alone in eight.*

Turning to clinical records we find that Dr Peacock ('St Thos. Hosp. Rep.,' 1873) recorded cardiac murmurs in a third of his cases. Mr Manser and Mr Shadwell abstracted 500 cases from the clinical records of Guy's Hospital, and found only 221 that were free from cardiac murmurs; while of the remaining 279 cases, 110 were believed to be pericardial and 169 endocardial. At St Bartholomew's Hospital, out of Dr Church's 574 cases in which the state of the heart was ascertained, it was affected in 371.

There is no doubt of the fact that acute inflammation of the heart is sometimes, and especially in children, the sole indication of a rheumatic attack, and that in other cases it is attended with only slight and fugitive articular pains, which may easily escape notice altogether. Nevertheless, as a rule, there is in acute rheumatism a direct relation between the degree of severity of the articular affection and the frequency, as well as the intensity, of cardiac complications. Thus Sibson found that the affection of the joints was severe in only one fourth of those cases in which the heart showed no sign of inflammation, but in two fifths of those in which endocarditis was diagnosed, and in three fifths of those in which there was pericarditis, with or without endocarditis.

Age, however, has a very marked influence upon the frequency of inflammation of the heart in acute rheumatism. The younger the patient, the greater the liability to it.† In young female servants, who make up a large proportion of the cases of rheumatism seen in hospital practice, the heart scarcely ever, according to Sibson, fails to show some signs of being attacked. Latham also said that maidservants with rheumatic fever in hospital always get pericarditis. On the other hand, many older patients escape; and here a curious difference is found to exist between men and women. Pericarditis seems to be three times as frequent in men above the age of twenty-five as in women of corresponding age. The reason for this is believed to be that a laborious occupation greatly augments the liability to cardiac complications in acute rheumatism; most women who are not young work less hard than men of the same age, whereas the girls of the lower classes are very apt to have their strength overtaxed.

Of the other complications of acute rheumatism, *pleurisy*, affecting

* These figures differ widely from those which Dr Sibson gave as the result of an analysis of 325 unselected clinical cases in his elaborate article in the fourth volume of 'Reynolds' System.' According to these, endocarditis was present nearly three times as often as pericarditis. Bouillaud, who first on the Continent called attention to cardiac inflammation in rheumatism, estimated its occurrence as 80 per cent., but that was in patients who were bled and leeched remorselessly. Budd put it at 48 per cent., Fuller at only 23 per cent., and the same figure was fixed by Lebert and by Wunderlich.

† This is strikingly shown by the third table in Dr Church's paper in the 'St Barth. Hos. Rep.,' vol. xxiii, p. 273. The percentage of cardiac affections in successive decades from under 10 to 50 runs thus:—83, 69, 51, 30, 21.

chiefly the left pleura, is the most frequent. Lebert speaks of it as occurring in 10 per cent. of all cases; and it would probably be far more often diagnosed than it is, if the pain produced by moving rheumatic patients did not interfere with stethoscopic examination of the back. Bronchitis is occasionally present; the lungs are often œdematous; and, in fatal cases, the microscope may show them to be affected with slight catarrhal pneumonia. Lobar pneumonia, with hepatisation of even a part of one lung, is of somewhat rare occurrence, and when present commonly leads to death.

Rheumatic peritonitis and iritis are both very doubtful, and rheumatic meningitis, cerebral or spinal, may be said not to exist.

In one fatal case that occurred at Guy's Hospital the *tonsils* were suppurating, and acute tonsillitis sometimes begins an attack.

Rheumatic nodules.—An additional concomitant of rheumatism, of both pathological and diagnostic interest, consists in small, subcutaneous, fibrous nodules, usually but not always in the neighbourhood of joints, often upon prominent points of bone, like the olecranon, the tibia, and the acromion. They were described by Meynet, of Lyons, in 1875, by Rehn in Germany (1878), by Hirschsprung in Denmark (1881); and a full account of them by Dr Barlow and Dr Warner will be found in the 'Transactions of the International Congress of 1881,' vol. iii, p. 116, with twenty-seven cases.*

Ætiology.—In causing acute rheumatism, *cold* is generally regarded as the most important factor. But this is open to question. Exposure to cold winds, or travelling in winter or in cold climates, or insufficient clothing does not cause it. The only efficient kind of cold is getting wet through. The patient is not infrequently attacked within a few hours after being out for several hours in the rain; but when there has been an interval of a week or even a fortnight since such an occurrence, the causal relation of events is more doubtful. Sometimes the exposure may have been repeated again and again for a considerable time before any harm seems to result; it may either be supposed to have a cumulative action, exerted slowly and from day to day; or the patient does not suffer until his power of resistance is so diminished by over-fatigue, or in some other way, that he now for the first time gets "a chill."†

Acute rheumatism is stated to be less common during the months of July, August, and September than at other seasons of the year. But the number of patients admitted into the wards of hospitals in London at different times is liable to wide variations which are not attributable to changes in the weather; for several weeks hardly a case may present itself, whereas afterwards a great many may appear within a few days of one another.‡

* Numerous cases have since been recorded by Dr Cavafy ('Path. Trans.,' xxxiv, p. 41), Drs Lees, Duckworth, Money, Drewitt, Stephen Mackenzie and Fowler, in the 'Transactions' of the Pathological Society for 1883 and of the Clinical Society for 1883–84. A remarkable case is described and figured by Drs G. S. Middleton in the 'American Journ. of Med. Sc.,' October 1887.

† I recently had a general servant under my care, who during the whole of one December had been obliged to sleep in a very damp washhouse because her mistress had chosen to take in a lodger; she was attacked at the end of about a month with acute rheumatism. Some years ago a footman was admitted into hospital from a house in Eaton Square; he attributed his illness to sleeping in a damp room, and he said that more than one of his predecessors had suffered in a similar way.—C. H. F.

‡ A review of the statistics at Guy's Hospital during ten years, made for this edition by Mr Capes, showed that the largest numbers of admissions were in September (158), and November (136); the next in October, January, and April (127 each). There were

The *geographical distribution* of the disease is as yet too imperfectly known to enable one to state positively its bearings upon this question. Hirsch gives a large amount of information; but with regard to many of the statements which he cites, it is difficult to tell whether acute rheumatism is meant, to the exclusion of the vague affections which are so often grouped together as "rheumatic." Thus, after stating that rheumatism is prevalent among the Esquimaux, in Iceland, and in Polar regions generally, he sums up by declaring that acute rheumatism is a disease mainly of the temperate zones. One striking circumstance is that in Cornwall and South Devon it is comparatively infrequent. In dispensary practice during four and a half years in Cornwall, only four cases occurred; and medical men practising in Exeter say that it seldom is seen in that city. The Isle of Wight and Guernsey are said to enjoy a similar degree of immunity from the disease. On the other hand, in the United States "rheumatism" appears to be far more frequent in the Southern than in the North-Eastern States, where the climate is comparatively cold and changeable. It is said to be rare in Canada and in Australia.

There can be no doubt that the members of certain families are especially liable to suffer from rheumatic fever; several children of the same parents are often affected by it, and others have chorea. Garrod traced an *hereditary predisposition* in about a quarter of his patients, Fuller in 27 and Chomel in 33 per cent. Among 400 hospital patients, the editor found it in 68, *i.e.* in 17 per cent.

Whether the disease is more apt to occur in persons of one complexion than in those of another is very doubtful. According to Laycock, those of "the rheumatic diathesis" are usually well built and well nourished, the complexion is of a healthy florid tint, the teeth are regular, sound, and firm, the hair is abundant; the skin greasy and thick. Mr Hutchinson (following Bazin) is convinced that there is a "diathesis" which is termed by him arthritic, and which is common to gout, rheumatism, and arthritis deformans. A very general impression is that most patients with rheumatic fever have light hair and grey eyes. But this applies to all diseases in England, because our population is chiefly xanthochroic.

Rheumatism is a disease of *youth:* a large proportion of first attacks take place in children and in young adults; and it scarcely ever happens that a person falls ill with it for the first time after the age of fifty. In infants, however, it is very uncommon; only a few cases have been recorded, one at the age of twenty-three days by Widerhofer, another at four weeks by Stäger, and one at ten months by Henoch; all quoted by Senator.

About four-fifths of first attacks occur between 11 and 30. At Guy's Hospital, out of 365 first cases, 22 fell between 5 and 10, 179 between 10 and 20, 118 between 20 and 30, 34 between 30 and 40, and only 12 above 40. Even second and third attacks become less frequent as the patient grows older. The editor has met with one in a woman of 61, and one in a man of 73.

Putting together 683 cases (whether first or later) from St Bartholomew's Hospital and 620 from Guy's Hospital:—Out of the total 1303, 48 occurred under 10, 521 between 10 and 20, 441 between 20 and 30, 195 between 30 and 40, 69 between 40 and 50, and 30 above 50.

There does not seem any difference in the liability of males as com-

not so many in May, June, and August (108—112); fewer in December, February, and July (about 100); and fewest in March (69).

pared with females to acute rheumatism more than can be explained by their harder work and greater exposure to weather. This seems to be shown by Dr Goodhart's cases in children below 14, where of 69 patients there were 27 boys to 42 girls.

Among 400 cases at Guy's Hospital the numbers were 223 men to 177 women. Among 654 cases obtained by the Collective Investigation Committee ('Brit. Med. Journ.,' Feb. 25th, 1888), 375 were men and 279 women.

Muscular *exertion* is, according to some authorities, another factor in the ætiology of the disease. It not infrequently sets in immediately after a long march, and its great frequency in domestic servants is supposed to be due to their being so often sadly over-worked. Some of the occupations which furnish large contingents of cases involve arduous muscular work; thus it is frequent in smiths and in carpenters.

A striking feature of acute rheumatism is its tendency to recur, apart from the relapses which so often follow the first attack within a fortnight. One attack is a predisposing cause of a second. The interval is commonly from three to five years, but it may be far longer.

Lastly, rheumatism not infrequently follows *scarlet fever*. It usually appears during the stage of desquamation.* A similar affection sometimes occurs as a sequel of dysentery (p. 185). The puerperal state is also believed to bear a causal relation to rheumatic fever, which is not infrequently developed in women who have been recently delivered, or have miscarried, especially when there has been profuse hæmorrhage.

On the whole, we must confess that the true cause of rheumatism is unknown. Practically all that can be said is that those who are young, those who are poor, and those who have suffered from it already are the most likely to be attacked by the disease.

Theory of rheumatism.—The pathological allies of rheumatic fever are chorea and erythema. Its only known antecedent is scarlatina, its only known consequence disease of the heart.

The essential nature of rheumatism is quite unknown. It has been ascribed with great confidence to a diseased humour, to a disturbance of nervous centres or of trophic nerves, to the presence of a bacillus, and to an organic poison in the blood.

The author of the present work, like other physicians, was attracted to the view, originally suggested by Prout, that the poison is lactic acid. The peculiar sour smell of the perspiration perhaps prompted the theory, but we have seen that the sweat is not more acid than usual. Analogy, a fallacious guide, argues that rheumatism, like gout, is due to some acid in the blood. In 1858 Dr B. W. Richardson published a series of experiments upon dogs and cats, which were believed by him to show that the injection of lactic acid into the peritoneal cavity was capable of setting up endocarditis; but three years later Reyher, in 'Virchow's Archiv,' pointed out that appearances precisely similar are constantly seen in the cardiac valves of healthy cats and dogs. After this the lactic acid theory languished, until, in 1871, Dr Balthazar Foster, of Birmingham, recorded in the 'British Medical Journal' two cases in each of which the administration of this acid (in doses of ɱxv to ɱlxxv), with a view to check diabetes, was followed by the occurrence of painful swellings of the joints; one patient had no fewer than six well-marked attacks, the symptoms of which seem to have resembled those of acute

* See papers by Dr Thos. Barlow and Dr Ashby, 'Brit. Med. Journ.,' Sept. 15th, 1883.

rheumatism. Külz in his 'Beiträge zur Path. u. Ther. d. Diabetes' related a case in which lactic acid set up pains called "rheumatic" in the hip and thigh. It is true that the same drug has been repeatedly administered to other patients and to healthy persons without result; but, as Senator remarks, such negative facts perhaps only prove that a "personal susceptibility" is necessary. Lastly, a plausible hypothesis is that under the influence of cold the lactic acid which is always formed as the result of muscular exertion fails to be destroyed by oxidation, as it should be, and that when so accumulated it acts as an irritant to the joints.

Prognosis: duration and relapses.—In the great majority of cases an attack of rheumatism when left to itself slowly and gradually improves, the pains diminish, the joints recover, and the fever subsides. This takes place in a variable time, occasionally in a week or ten days, often not until three weeks have elapsed, and sometimes only after six or eight weeks. The duration of rheumatism, however, is now happily so much modified by treatment, and the estimation of its natural length is so much concerned in the judgment we form as to the results of treatment, that this question will be more particularly considered in the following section.

Of all acute diseases, rheumatism is the most prone to relapse. Its relapses are not like those of the disease known as relapsing fever (vol. i, p. 157), an essential part of the process: in most cases they are absent. But in a large proportion—varying in different times and places and under different modes of treatment from a sixth to a fourth or a third of the whole number—only a few days after the first attack has subsided, a second comes on; this runs a similar though usually a milder and a shorter course, and the heart, if it has escaped before, is seldom affected now. A second relapse is common, and a third and a fourth are sometimes seen. Dr Church has proved that relapses are much more common after second than after first, and still more so after third attacks. The question of how far they can be prevented by regimen and drugs will be presently considered (p. 827), for it is one important object of treatment. There is no doubt that prolonged care and continued treatment during convalescence are influential in preventing relapses. Hence they are comparatively rare among patients who can afford a prolonged period of easy convalescence, while it is one of the disappointments of hospital practice to see a patient, who has been sent out perfectly well after an attack of rheumatism, return to the ward a week or two later with all his symptoms renewed.

The joints are very rarely permanently injured. Occasionally hydrops articuli remains behind, and after many attacks the joints may undergo the changes described in a subsequent chapter as osteo-arthritic. In proportion to the severity of the attack, anæmia is a marked effect and is scarcely ever absent, while return of muscular strength is often very slow.

The liability to peri- and endocarditis, with their subsequent effects, has been already considered.

Mortality.—Apart from the ultimate effects of rheumatic diseases of the valves, a fatal result is very rare. When not due to acute pericarditis, or to some accidental cause, death usually results from hyperpyrexia, a remarkable condition to be presently considered, or from pneumonia.

Senator puts the average mortality at from 3 to 3·7 per cent. of those who are attacked, and at Guy's Hospital it was in the 400 cases collected by the editor nearly the same, 3·75. So also in the 655 cases of the Collective

Investigation Report the mortality was 3·3 per cent. But at St Bartholomew's Hospital Dr Church found only 10 deaths in 693 cases (1·3 per cent).

The number of deaths in our wards varied widely in different years. From 1855 to 1867 inclusive only *ten* fatal cases altogether appear in the records. From 1868 to 1884 there were *fifty* fatal cases. The numbers in the several years were as follows :—Two in 1868, three in 1869, five in 1870, two in 1871, seven in 1872, two in 1873, seven in 1874, six in 1875, three in 1876, six in 1877, one in 1878, three in 1879, one in 1880, two in 1881, and none in 1882–3–4.

In three of these sixty cases the fatal termination was due to stenosis of the mitral valve, which had itself resulted from previous attacks of acute rheumatism. In only four of the fatal cases was there found a severe and recent lesion of the valves as the direct result of the rheumatism.

In eighteen instances death was attributed to *pericarditis*, which was sometimes severe, the heart being covered with lymph, and there being from ten to sixteen ounces of fluid, deeply stained with blood. In several of these cases there was also effusion into one or both of the pleural cavities ; twice there was inflammation of the mediastinum ; in seven cases the muscular substance of the heart was obviously involved in the inflammatory process. Each of the complications in question may be supposed to have helped in bringing about the patient's death ; and it is not improbable that myocarditis was sometimes present when it was not noticed. Although endocarditis existed in eleven of the eighteen cases, it was so slight that it could not be considered to have affected the issue.

In twenty-seven fatal cases of acute rheumatism collected by the editor, death occurred from *hyperpyrexia* with delirium in five, the ages of the patients ranging from twenty-three to thirty-one. One died from delirium tremens with acute *pneumonia*, four from pneumonia with cardiac lesions, thirteen from severe pericarditis or valvular disease or both, the immediate cause of death in two being embolism, complicated in both cases by chorea. The remaining three died from accidental causes : diphtheria, enteric fever and epilepsy, which supervened while under treatment for rheumatism.

Rheumatic hyperpyrexia.—That cerebral symptoms sometimes develop themselves in the course of rheumatic fever, and rapidly prove fatal, has long been known. By Sir Thomas Watson and Dr Latham it was thought that they depended upon cardiac complications, and particularly on pericarditis ; but in many cases the heart is found free from all signs of inflammation. The existence of meningitis has also been disproved.

In 1867 Dr Ringer related three cases of rheumatic fever in which extremely high temperatures were observed, the thermometer having risen from 104° to 105° to 109·2° or even 110·8°. Fahr. Dr Kreuser noticed the same fact in Würtemberg about a year previously. Many similar cases have been since recorded, both in England and abroad ; at Guy's Hospital, in seven years fourteen patients died of hyperpyrexia in acute rheumatism.

The most probable pathology of this terrible complication is that while the development of the high temperature is itself consequent upon an exhaustion of the heat-regulating centre in the bulb, the cerebral symptoms in their turn result from the action of the heated blood upon the brain. Unlike the occasional high temperatures observed in severe cases of pneumonia and fever shortly before death, in acute rheumatism hyperpyrexia may suddenly bring to a fatal issue an illness which had seemed to be attended with little or no risk. One of Dr Ringer's patients was supposed to have

recovered, and was to leave the hospital the next day, when cerebral symptoms set in, and he died in two hours, with a temperature 110°.

The first indication of the onset of hyperpyrexia is often that the patient loses his pains and finds that he can move all his limbs without suffering. He and his friends are naturally rejoiced at what seems to be a great improvement in his condition; but such an occurrence, unless the temperature falls at the same time, should be to the physician a warning of impending danger, and should lead him to employ the thermometer at regular intervals of ten, twenty, or thirty minutes. Another warning symptom is that the skin ceases to perspire.

The cerebral symptoms, which rapidly supervene, vary in character in different cases. Sometimes the patient becomes drowsy, appears to fall asleep, and so passes into a state of unconsciousness, with contraction of the pupils, which ends in death. Sometimes he grows violently maniacal, jumping out of bed, and fighting with nurses and attendants. Sometimes he is seized with convulsions, or with tonic spasms and opisthotonos. His pulse becomes very rapid, from 140 to 186, and towards the last it may become imperceptible. His breathing is much accelerated, and his face sometimes exhibits the dusky purple flush of congestion, with eyes suffused, and more or less complete cyanosis.

The interval which elapses from the commencement of the hyperpyrexia to the fatal termination is very variable, as is well shown by an analysis of twenty-two cases made by Dr Wilson Fox. In one instance the thermometer rose from 103·5° to 109° in two hours; in two other instances a period of twenty-four hours passed before a similar point was reached. Even when the temperature is 107° or 108° the patient may now and then continue to live for some hours; and at 110° there may still be an interval of from one to two hours before the fatal termination.

Dr Fox thought that hyperpyrexia is more frequent in first attacks of rheumatic fever than in subsequent ones; and subsequent experience has confirmed this. As shown in a valuable report based on 67 cases, rheumatic hyperpyrexia is more common in men than in women ('Clinical Trans.,' vol. xv).

Diagnosis.—Acute rheumatism is, as a rule, very easy to recognise. But there are not a few diseases which may be mistaken for it, and in some exceptional instances it may be impossible to decide.

The author was called into the hospital one Sunday by the house physician to see a girl who had been just admitted for rheumatic fever, and whose temperature was very high. The peculiarity of her case was that she was covered all over with a bright scarlet rash. She had had acute rheumatism on a former occasion, and the real nature of her illness was not suspected until the following day, when the papular eruption of *smallpox* was found upon her. In several instances the pains in the limbs produced by the growth of *multiple sarcomata* in the body, especially about the vertebræ, so as to affect the spinal nerves near their origins, have been supposed to be due to acute or to subacute rheumatism (vol. i, p. 111).*

Ulcerative endocarditis sometimes complicates rheumatism. In some cases so regarded, it is permissible to doubt whether the rheumatism was really

* In a case of multiple sarcomata of the skin, secondary to a growth in the cæcum under the editor's care, there were pains and swelling about the joints which he mistook for rheumatism, and when the sarcomata appeared he supposed them to be purpuric erythema—peliosis rheumatica.

present or whether the endocarditis was not primary. It formerly often happened that affections of the spinal cord were in their early stages regarded as rheumatic on account of the pains to which they gave rise, until the supervention of paralysis showed a mistake to have been committed. But it must not be forgotten that in some spinal cases, and notably in locomotor ataxy, the joints do actually suffer. The same remark applies also to *syphilis*, the effects of which are not infrequently set down to rheumatism. Lastly, *scorbutus* and *hæmophilia* must be mentioned as diseases with regard to which it is by no means difficult to make a similar blunder.

The diagnosis from an acute attack of *gout* is occasionally difficult when the latter occurs for the first time in a young subject, but such cases are usually hereditary, and the heart being unaffected would be unusual at that age in rheumatism. The diagnosis from gonorrhœal synovitis and from acute osteo-arthritis will be given hereafter (pp. 838 and 841).

A useful means of diagnosis in doubtful cases is afforded by the presence of the "rheumatic nodules" described above (p. 815).

Perhaps the most common and serious mistake is mistaking for rheumatic fever one of those forms of *pyæmia* which come particularly before physicians because they are not secondary to a wound or obvious source of suppuration. The most frequent primary lesion is caries of the petrous bone—which may be recognised by otorrhœa, or by fœtor of a fragment of cotton wool left in the meatus even when there is no obvious discharge of pus, by perforation of the membrana tympani, and by tenderness over the mastoid process or the jugular vein. Almost as frequently, the pyæmia is due to acute osteo-myelitis of a long bone, and then the affected limb will be found hot, tender, and œdematous. Occasionally acute periostitis with suppuration and secondary synovitis may in like manner simulate rheumatism.

Sometimes, in spite of all care, diagnosis is impossible till after death, and difficult even then. In 1879 Dr Goodhart made an autopsy in the case of a boy aged sixteen, who had been lying for five days in a medical ward in a typhoid condition. He had a systolic murmur; his temperature had been 103·8° on admission, his pulse 132, his respirations 48. He had been attacked with pains and with chilliness five days before his admission. Numerous abscesses were found in the lungs and in the kidneys. Evidently, therefore, the case was one of pyæmia; but it was not until after nearly every other bone had been examined that Dr Goodhart discovered suppuration beneath the periosteum of the lower end of the right fibula. On the tricuspid valve there was a vegetation of the size of a pea, with a little ulcer beneath, which had been torn through some of the chordæ.

How closely pyæmia may resemble acute rheumatism is well shown by a case which occurred to Dr Moxon in Guy's Hospital in 1877. A patient was admitted who had nine days before been attacked with headache, sickness, and rigors, and in whom these symptoms were followed by profuse sweating and by pains in the joints. Salicylic acid was prescribed, but the temperature rose and delirium set in, so that cold baths were employed on several occasions. The question whether the disease could be pyæmia was formally discussed, and negatived in favour of the diagnosis of acute rheumatism. He made no complaint of the thigh, as being more painful than other parts, when he was being moved into or out of the bath. Yet at the autopsy not only was there osteitis of the lower part of the shaft of the right femur and of the adjacent epiphysis, but the bone was denuded of its periosteum, and there was a large collection of dirty pus beneath the

muscles. There was also suppuration about the shoulder, and pyæmic abscesses in the lungs and heart.

An important distinction between acute rheumatism and pyæmia is that in the former disease the pain and the swelling are apt to fly about, leaving one joint, and after a few hours attacking another. But that this criterion is not perfect is proved by the case of a boy who died of ordinary pyæmia in a surgical ward in 1870, and who for a time had flying pains in the joints.

It is worthy of notice that almost all the instances that have occurred at Guy's Hospital within the last few years of pyæmia from osteo-myelitis have been in boys or young men between ten and twenty years of age. As a rule, the diagnosis is rendered comparatively easy by the severe constitutional disturbance which is present, by the presence of rigors, and by the skin being dry instead of sweating; the joints, too, show a deeper blush, and are more frequently hot. One very important question refers to the significance of certain eruptions which are sometimes associated with pyæmia. Beside the scarlet rash which is comparatively common in surgical wards, and which has now been proved to be really scarlatina, there are cases in which the skin presents appearances of a still more remarkable kind. Thus in 1861 a boy of thirteen was admitted into hospital who had for a week been treated for rheumatic fever, but whose disease was at once recognised to be pyæmia, there being a large abscess of the thigh. Towards the last his body became covered with "a purplish rash, resembling the mottled rash of typhus, partly consisting of petechiæ (probably fleabites), partly of papules which became vesicular at the apices and slightly scabbed." In another patient, in 1874, pustules are said to have appeared on the back and on the abdomen two days before death. Pustular eruptions are mentioned in two instances of "spontaneous pyæmia" collected by the Pathological Society's Committee in 1879.

In regard to all such cases, however, it is necessary to be careful not to overlook the presence of *glanders*. That disease is itself sometimes mistaken for acute rheumatism at its commencement.

Another class of cases which are very likely to be set down to acute rheumatism are those in which *pyæmia* results *from gonorrhœa*. In 1872 a young woman was admitted into Guy's Hospital with what was supposed to be rheumatic fever; an hour later she was delivered of a child which survived for some days; she was now seen to be suffering from pyæmia, and four days afterwards she died. At the autopsy an abscess was found in the subserous tissue near the right ovary, and there were softening thrombi in the adjacent veins; but it was thought that these lesions were themselves secondary, and that the starting-point of the disease was a vaginal discharge from which she had been suffering, and which was probably of a gonorrhœal character.

In ordinary surgical practice it seldom happens that pyæmia is mistaken for acute rheumatism. But sometimes the converse blunder is committed, and when a rheumatic affection of the joints arises in a person who has been recently operated on, or who is suffering from an accident, pyæmia may at first be feared.

Treatment.—Until lately, nothing could be more unsatisfactory than the treatment of rheumatism. A great variety of medicines were prescribed, each of which appeared to be highly successful in some cases, while it utterly failed in others. Some physicians, and especially the late Dr Fuller, of St George's Hospital, recommended the administration of alkalies, in such doses as to main-

tain an alkaline reaction of the urine; some, following Dr Rees, pinned their faith to lemon-juice. Some gave quinine, others the tincture of iron, others colchicum, others ergot, others propylamine (or rather trimethylamine). The late Dr Herbert Davies advocated the employment of blisters to all the affected joints; surrounding the knees, for example, with strips of emplastrum cantharidis three inches wide. It seems to be certain that the blistering plan is often followed by a rapid subsidence of the local inflammation, but there is the disadvantage that when carried out fully it is apt to produce strangury, and (as Senator has shown in vol. lx of 'Virchow's Archiv') it sometimes leads to the presence of fibrin in the urine, which may not only contain flakes when voided, but even coagulate afterwards. Moreover, it may produce local sloughing.

It would be difficult to say when observers of disease first began to entertain the suspicion that the apparent success of various methods of curing acute rheumatism might, after all, be fallacious, and that the result would have been the same if nothing had been done. Sir Thomas Watson used to cite, but without assenting to it, the dictum of Warren, who, when asked what was good for acute rheumatism, replied, "Six weeks." But it is only within the last few years that careful observations have been systematically made and recorded of cases left to themselves, or in which no drugs were administered that could be supposed to disturb their progress. Such an "expectant treatment," as it has been termed, seems to have been first tried by Lebert, who in 1860 published the results in nine patients. A few years later Sir William Gull and Dr Sutton, in the 'Guy's Hospital Reports' for 1866, placed on record the details of twenty-five cases.*

From these observations it is clearly apparent that acute rheumatism is altogether unlike those among the specific fevers which have a fixed or definite duration. Among Sir William Gull's patients the length of time during which active symptoms continued, including that passed before admission as well as subsequently, varied from nine to thirty-four days. On striking an average, the course of the disease, as measured by the period at which there is freedom both from pyrexia and from joint-pains, is much shorter than that named by Dr Warren. The English physicians made it 19 days; Lebert gave 16 days as the average time when the symptoms first greatly improved, 22 days as that for convalescence.

In attempting, however, to compare cases of acute rheumatism left to themselves with those submitted to treatment by drugs, there is a fundamental difficulty. Strictly speaking, those cases only are comparable, in which the treatment was begun on the very day on which the patient was taken ill. But to include only such cases would be not only to limit the

* It is, however, wrong to consider the "expectant" treatment carried out by Dr Gull at Guy's Hospital as doing nothing. It was the same rational treatment used in cases of pneumonia, of enteric or scarlet fever, and of fractured limbs. The patients were kept perfectly undisturbed, no strangers were allowed to approach their beds, they were screened from draughts, and the light was shaded. Their joints were covered with cotton wool dipped in laudanum, and protected from pressure by cradles. Except under very special circumstances, no laxative was allowed. They were given effervescing ammonia julep, or any cooling drink preferred, and a grain of opium was administered every night, or oftener if the pain was severe. Under this treatment, if there were few brilliant recoveries, there was on the whole far less pain and distress, a shorter average course, less cardiac disturbance, and a more safe and rapid convalescence, than under the antiphlogistic treatment of Bouillaud, the lemon-juice of Dr Rees, the alkaline treatment of Dr Barlow (each of which the writer had the opportunity of comparing with Dr Gull's), or any other plan which was in use before the introduction of salicin.

field of observation almost entirely to private practice, but also to ensure that the cases accepted should be of far more than average severity, since only patients seized with violent symptoms are likely to seek medical advice at once. It is therefore impossible to reject cases which have come under treatment at varying periods of the disease. But now arises the question whether one should take into account the time which the disease has lasted before the patient is seen. It is clear that if treatment is effective the fact must be brought out most strikingly when this time before treatment is left out of consideration; on the other hand, if drugs are useless, we should take the whole duration of the cases as a basis for comparison.

It is indeed easy to prepare both sets of figures, as was done by Gull and Sutton; and, in dealing with average results, no serious error is likely to arise whichever set be adopted—provided the cases are numerous, so that each group may contain its proportion of patients brought under treatment at an early stage, and of those in whom the disease was already far advanced.

But there remains an insurmountable objection to the employment of averages, which invalidates the comparisons made by Gull and Sutton of the results obtained by the "expectant" with those by other methods. The objection is that some cases of acute rheumatism run a very protracted course, which, instead of terminating in a few weeks, is measured by months. One cannot be surprised that no such instances appear to be found in Gull's and Sutton's lists; for it would be scarcely practicable to keep the patients week after week under observation without attempting something for their relief; and afterwards in tabulating results, the very fact of treatment having been adopted would be sufficient to exclude them. But when one is dealing with a series of cases submitted to any particular treatment, it becomes impossible to reject the cases in question, some of which may perhaps have been discharged from hospital still uncured; and when an average is taken, a few such cases would swamp the rest in which treatment may have been successful.

TABLE I.—*Natural Course of Twenty-four Cases.*

Duration of Symptoms while in Hospital.	Number of Cases.	Duration of Symptoms before Admission.	Average of the same.
3 days	1	21 days	Average 21 days.
4 days	1	5 days	„ 5 days.
5 days	1	Uncertain	—
6 days	2	7 days, 5 days	„ 6 days.
7 days	3	3 days, 3 days, 14 days	„ 6·3 days.
8 days	1	5 days	„ 5 days.
9 days	1	9 days	„ 9 days.
10 days	2	8 days, 5 days	„ 6·5 days.
11 days	3	8 days, 5 days, 5 days	„ 6 days.
12 days	2	6 days, 10 days	„ 8 days.
13 days	1	6 days	„ 6 days.
14 days	1	14 days	„ 14 days.
16 days	2	12 days, 6 days	„ 9 days.
18 days	1	12 days	„ 12 days.
21 days	1	4 days	„ 4 days.
27 days	1	7 days	„ 7 days.
Duration before admission of 23 cases in which it could be determined—180 days; giving an average of			„ 7·8 days.

It appears that the only fair way of using Gull's and Sutton's cases is to tabulate the length of time that symptoms lasted after admission: the list is then as follows, a fatal case being excluded (see Table I).

It is, of course, to be wished that the number of cases had been larger; but from the regularity with which they are distributed over different parts of the column one may perhaps conclude that the result represents an average. A point which comes out clearly from the last column is that a comparatively long duration of the disease after admission is by no means peculiar to patients admitted at a very early period of their illness. On the other hand, it may be noticed that the case which subsided most rapidly while in the hospital had already been running on for three weeks; and this was not possibly a mere accident, for on tabulating ten other cases in which rapid recovery took place without treatment (or under ineffectual treatment), we find that the average duration of the disease before admission was 13·7 days.

Let us now compare the natural course of the disease with the results of three different methods of treating acute rheumatism, as recorded by their several advocates (see Table II). It seems clear from these figures that no

TABLE II.—*Results of Treatment by Lemon-juice, Alkalies, and Blisters.*

Duration of Symptoms after Commencement of Treatment.	Lemon-juice. Dr Owen Rees ('Guy's Hosp. Rep.,' xii).	Bicarbonate of potass. Dr Garrod ('Med.-Chir. Trans.,' xxxviii).	Free Blistering. Dr Davies ('Lond. Hosp. Rep.,' i).
1 day	1	—	—
2 days	—	1	—
3 days	—	3	—
4 days	1	8	—
5 days	—	6	1
6 days	1	6	2
7 days	2	7	1
8 days	—	5	2
9 days	1	9	1
10 days	2	—	—
11 days	—	2	—
12 days	—	4	—
13 days	—	—	1
14 days	1	Doubtful	5
Total cases	9	51	13

striking success can be claimed for any of the three plans of treatment. The most favourable seems to have been the alkaline method, but even under this the cases in which the symptoms subsided within five days amount to only eighteen out of fifty-one, or less than 36 per cent.

Salicin.—On the other hand, it has now been conclusively shown that the duration of rheumatism may be greatly shortened by the administration of salicylic acid or its alkaline salts or salicin. It was in January, 1876, that Dr Stricker, assistant in Traube's Clinic in Berlin, drew attention to the use of salicylic acid in rheumatism. It had been used there for some months, and the same medicine had been employed at Basle by Buss. Salicin was originally advocated by Dr T. J. Maclagan, in March, 1876, in the 'Lancet.' He had first prescribed it in 1874.*

* Among the Hottentots and the Boers of South Africa willow tea has, according to Mr F. Ensor, long been a traditional remedy for rheumatism.

After a period of reasonable scepticism and trial, the salicyl treatment became firmly established in this country and in Germany, and was introduced into France by Professor Sée. The immense majority of physicians and practitioners in Europe and America now use it.

Salicylic acid, as originally used in Germany, is a very unsuitable form of the remedy. It is insoluble, bulky, unpleasant, and irritating. It is now always neutralised by ammonia, potash, or soda, and the soda salt is the one generally used. It is probably less depressing to the heart than salicylate of potass, but it is often well to add ammonia or alcohol so as to obviate this result. Salicin is an agreeable remedy, and now scarcely, if at all, more expensive than the salicylates. It is also less irritating to the stomach and bowels, and milder in its general effects. But it sometimes fails when other salicyl-compounds succeed, and therefore is best used as a substitute for sodium-salicylate when its effects are found to be undesirable.

The author took pains to tabulate not only his own experience at Guy's Hospital, but also that of all his colleagues, in the treatment of acute rheumatism with salicin or salicylates from the spring of 1876 to the end of 1880; and obtained the following results (see Table III).

These figures obviously compare very favourably with those given in Table I as the result in Gull's and Sutton's cases. The symptoms were arrested within five days in no fewer than 180 of our 355 patients, at which period only three of their twenty-four patients had lost their symptoms. It must be added that the rapidity with which relief is afforded to the joint-pains is actually far greater than appears in the table. In many of the cases which are set down as having become relieved at the end of five or six or seven days, the patient within two or three days was almost without pain, or had almost a normal temperature, or was almost free both from pyrexia and from pain. It is no uncommon thing for the patient to be conscious of experiencing great relief from the first two or three doses of the medicine; and house physicians have repeatedly remarked the striking contrast, even on the first night after admission, between patients treated with salicylic acid or salicin and those to whom no medicine had as yet been administered; the former lie quiet, even if they do not sleep; the latter often cry out during nearly the whole night, disturbing everyone else in the ward. Moreover, in many of the cases placed lower down in the list, the drug was given for a few days, or even for twenty-four hours only, and was then withdrawn; or the doses in which it was administered were such as are probably too small to arrest the disease. In the above table every case has been included in which as much as sixty grains of salicylic acid or of salicin was given in the twenty-four hours. With a single exception the tabulation is drawn up, in every respect in such a way as to place the facts in the light more adverse to the success of the treatment.*

Relapses.—The exception just alluded to has reference to the relapses which occurred in ninety-three of the 355 cases. This list does not include certain instances in which during convalescence (perhaps when the patient first got up) pain returned for a day or two in a single joint, or in which without pain the temperature rose once or twice to a point between 99° and 100°. Such

* Among other statistics of the effect of salicylates on acute rheumatism, the reader is referred to an elaborate analysis of 210 cases at St George's Hospital by Dr Isambard Owen ('Lancet,' 1881), to the 158 cases reported in the 'Lancet' for 1879 by Mr R. H. Lucas, of Bury St Edmunds, and to the results of 536 cases reported in the 'Brit. Med. Journ.' for 1888, vol. i, p. 395. See also Dr Bristowe's paper read at Cardiff in 1885, and the discussion which followed ('Brit. Med. Journ.,' August 22nd).

TABLE III.—*Results of Treatment by Salicin or Salicylates at Guy's Hospital, 1876—1880.*

Period at which Patient became free from Pyrexia, as well as from Joint Pains, reckoned from commencement of Treatment.	Number of Cases.	Number of Cases in which Relapses occurred.		
		1 Relapse.	2 Relapses.	More than 2 Relapses.
1 day	7	2	—	—
2 days	41	8	1	1
3 days	40	11	4	—
4 days	42	10	3	1 (six relapses)
5 days	50	17	7	3 (in 1, five relapses)
6 days	23	4	2	—
7 days	21	6	1	—
8 days	20	9	2	—
9 days	13	7	3	1
10 days	14	3	2	1
11 days	14	2	1	—
12 days	6	2	1	—
13 days	5	2	—	—
14 days	6	0	—	—
15 days	4	2	—	—
16 days	2	1	—	—
17 days	2	0	—	—
18 days	3	1	—	—
19 days	0	—	—	—
20 days	0	—	—	—
21 days	2	—	—	—
22 days	1	—	—	—
23 days	3	2	1	—
24 days	2	—	—	—
25 days	1	—	—	—
26 days	2	—	—	—
27 days	0	—	—	—
28 days	2	1	—	—
29 days	3	2	—	—
30 days	1	—	—	—
32 days	1	—	—	—
35 days	1	—	—	—
36 days	1	—	—	—
37 days	1	—	—	—
41 days	1	—	—	—
49 days	1	1	—	—
55 days	1	—	—	—
60 days	1	—	—	—
Indeterminate	15	—	—	—
Fatal cases	2	—	—	—
Totals	355	93	28	7

occurrences were not infrequent, but as no treatment was required, and as recovery was in no way retarded, one may leave them out of consideration. The actual relapses more or less closely resembled primary attacks of acute rheumatism, so that the patients had again to be kept in bed and on low diet. In one case a relapse lasted twenty-one days ; and the average duration of thirty-one relapses (some treated, some left to nature) was between five and six days.

Now, there are two ways of looking at the relapses of acute rheumatism. One is to regard them as continuations of the original illness, and to suppose

that it has been interrupted and postponed, but not really cut short by the administration of the remedy. This view is especially applicable to cases in which the symptoms return very soon after the discontinuance of treatment. Thus in four instances the disease reappeared within about twenty-four hours; and in nine others before a week had elapsed. In a case of Dr Habershon's in which the urine was tested with perchloride of iron for several days after the administration of salicylate of soda had been left off, the purple reaction indicative of the presence of a salicyl compound was obtained as late as the eighth day; it is therefore quite possible to set down all the thirteen cases just alluded to as examples of the "recrudescence" of the primary attack. But on the other hand there were six cases in each of which the relapse began when a period of from three weeks to two months had passed after the subsidence of the primary attack, and when the medicine had long been discontinued. Such cases must be looked at in the same light as those which relapse when no treatment at all has been adopted; this happens not infrequently, but in what numerical proportion of cases it is not possible at present to state. It does not seem likely that relapses are more apt to occur when salicin or salicylate of soda has been given than when the disease has been treated in other ways, or left to run its own course. However, those observers who (as will presently be mentioned) consider salicylic acid a very depressing agent would perhaps be not unwilling to entertain such an opinion. The increased liability to relapse in enteric fever when antipyretic methods are adopted cannot be cited as analogous, because the two diseases are believed to differ so completely in their pathology. In twenty-eight of the 355 cases treated with salicin or salicylic acid at Guy's Hospital the original attack was followed by two relapses, in five of them by three, in one by four, and in one by six. It may easily be imagined that such patients spent many months in the hospital wards. It is probable that the frequency of relapses is greatly diminished by systematically continuing the administration of moderate doses of salicylate, or of salicin, until after the lapse of several weeks.

Doses.—The dose of salicylate of soda which is adequate to arrest acute rheumatism with rapidity appears, as a rule, to be about twenty grains, given at intervals of two or three hours; but sometimes a larger quantity is required. One patient of the author's took twenty grains of salicylate of soda every two hours without any marked result for two or three days, but the disease at once yielded when thirty grains were given. On the other hand, there are cases in which a dose of ten grains, repeated every six hours, seems to be effectual.

Salicylate of soda may be dissolved in aqua menthæ piperitæ, or in aqua carui. Or the acid may be dissolved in solution of citrate or of acetate of ammonia (the proportion being gr. xx of acid to ʒiij of liq. amm. acet.), and sweetened with extract of liquorice.

It is generally necessary to use a larger quantity of salicin: Dr Maclagan recommends that from twenty to forty grains should at first be given every hour. It may be prescribed as a powder stirred up in cold water, or twenty grains may be dissolved in an ounce of warm water.

Influence on the heart.—As regards the possible influence of salicylic acid in preventing the development of cardiac complications in acute rheumatism, it is very difficult to make any definite statement. One cannot but remember that for each new method of treatment introduced—even for Dr Davies' treatment by local blistering—the assertion has been made that it lessens

the liability to inflammation of the heart. On the other hand, Gull and Sutton showed that in cases in which the organ is healthy at the time of the admission of the patient into hospital, it seldom becomes subsequently attacked. Still, one may reasonably expect that any remedy which possesses the power of arresting acute rheumatism, so that after its administration fresh joints no longer become affected, must also hinder the development of what is believed to be an analogous morbid process in and around the heart. And although in sixty-nine of the 355 cases auscultation revealed some changes in the character of the cardiac sounds while the patient was in the hospital, there was hardly one in which there was reason to believe that pericarditis set in at a time when the action of the remedy was fully established. Almost all of them were cases in which was detected a systolic murmur of extremely difficult interpretation.

On the other hand, it would certainly seem that salicylic acid has no power of controlling or arresting the cardiac complications of acute rheumatism when they have once developed themselves.*

Drawbacks.—In certain cases the administration of salicylates is attended with inconveniences, and sometimes with symptoms which are alarming. A not infrequent effect is nausea and *vomiting*, accompanied with pain at the epigastrium. But this is seldom so severe as to make a change of treatment necessary.

Another effect is *enfeeblement of the heart's action.* At Guy's Hospital this has rarely attracted attention; in a few instances it is noted that the pulse became weak, and sometimes that it was irregular or intermittent; in one case it fell, after nine days, to fifty-two beats in the minute; in two the first sound of the heart became inaudible, and the heart's impulse could no longer be felt. The administration of stimulants has, however, been very seldom deemed necessary. On the other hand, Dr Greenhow ('Clin. Trans.,' 1880) says that in his patients, who were treated with salicylic acid, "more or less weakening of the pulse, requiring the free administration of brandy, occurred in nearly every case. This was accompanied by great weakening of the impulse of the heart, and in ten cases by almost complete obliteration of the first sound." Dr Goodhart recorded a case in the same volume in which sudden death in the night took place, probably as the result of failure of the heart, for the pulse had been rising in frequency. He was disposed to attribute this result to the administration of salicylic acid, but only sixty grains in all had been given, and none of the known effects of the drug were observed. At the autopsy early pericarditis was found.

Far more obvious than the cardiac are the *cerebral symptoms* to which salicylates (but very rarely salicin) sometimes give rise. Deafness is a very frequent and an early effect of the remedy. It is often accompanied with a sensation of giddiness, and with noises in the ears, which are described by the patients as buzzing or ringing, or are compared with the noise of a train or the rushing of water. In some cases there is headache, which may be very intense; or patients become delirious, screaming and struggling to get out of bed. It often happens that when such effects are produced the patient has already lost his pains, and that his temperature has fallen to 99° or to 100°; the latter circumstance distinguishes the cerebral symptoms

* Dr Church, comparing his cases under salicylic treatment with those of the late Dr Latham at the same hospital, long before its introduction, believes that the liability to endocarditis is much the same, but that pericarditis is less frequent, and pleurisy and pneumonia far less common and far less severe.

due to salicylic acid from those of hyperpyrexia. The face may be deeply flushed and bathed in perspiration, and in one case the delirium is reported to have been attended with "typhoid symptoms, so that the prognosis for a time was grave." As a rule the patient becomes rational in a few hours, or in a day or two, after the medicine is discontinued. A fatal result has very rarely followed, and in these exceptional cases we have usually found some other cause after death. The drug has often been resumed after a few days' interval without further ill-effects; but sometimes delirium has set in on successive occasions.

Another occasional effect, whether of salicin or of salicylic acid, is *epistaxis*. It was particularly noticed by Dr Greenhow, and has occurred in many cases treated at Guy's Hospital. It often recurs several times, but is not attended with evil consequences, except that it must no doubt tend to increase anæmia, and so to retard convalescence.

Dr L. E. Shaw has recorded in the current number of the 'Guy's Hospital Reports' (vol. xliv, p. 125) three cases of severe *hæmorrhage* occurring during the administration of salicylate of soda for acute rheumatism. In the first patient, a boy aged fifteen, taking a scruple of the drug every three hours, delirium appeared on the fourth day (eleventh of the disease), with epistaxis and retinal hæmorrhage. He recovered, but the other two cases proved fatal. In one, a wardmaid aged twenty-one, after taking the same dose of salicylate every two hours, became delirious and passed blood in her urine. After death there was no cardiac lesion found, and the kidneys were normal, but the renal pelvis and the bladder were covered by ecchymoses. In the third patient, a woman aged twenty-six, taking the same gr. xx dose every three hours, for a severe febrile attack of doubtful nature, delirium and hæmaturia appeared, and after death, beside lesions due to latent enteric fever, a precisely similar condition of kidneys and bladder was found.

Taking the cases of rheumatic fever which occurred in our wards in two years (1881 and 1886), as specimens, Dr Shaw found that in these 174 cases, all treated by salicylate of soda, except a few by salicin, and in nearly the same doses, there were more or less toxic effects in no fewer than 111. Of these, deafness was present in sixty-one cases, delirium in thirty-three; headache, vomiting, and singing in the ears in about the same number, a slow or irregular pulse in thirteen, epistaxis in eleven, and hæmaturia in the two cases above quoted.

Lastly, it has been supposed that the salicylic treatment causes anæmia, and leaves the patient exhausted and feeble, so that he regains health and strength more slowly than if the disease had been left to run its natural course. There can be no doubt that the stay in hospital of cases treated with salicylic acid is little if at all shorter than it used to be before the remedy was used. But it must be remembered that for a long time after active symptoms have ceased one keeps the patient on low diet and confines him strictly to bed, for fear of the occurrence of a relapse.

It remains to consider the facts brought forward by Dr Greenhow, which led him to express, on the whole, an opinion unfavourable to the treatment of rheumatism by salicyl-compounds. On throwing his sixty cases (ten treated by salicin and fifty by salicylate of soda) into a form to be compared with the preceding table, we obtain the following results (Table IV).

These results do not seem to be unsatisfactory. In most of the cases which are set down as of indefinite duration, it is doubtful whether the value of the medicine was fully tested: either too small a dose was given

TABLE IV.—*Dr Greenhow's Results with Salicylic Treatment.*

Period at which Patient became Free, both from Pyrexia and Joint Pains, dating from the commencement of Treatment.	Number of Cases.	Number of Cases in which Relapses occurred.
1 day	5	2 (in 1, two relapses; in 1, three relapses).
2 days	11	3 (in 1, two relapses).
3 days	14	6 (in 2, two relapses; in 2, four relapses).
4 days	5	3 (in 1, two relapses).
5 days	1	1 (two relapses).
6 days	1	—
7 days	1	—
8 days	2	—
12 days	1	—
14 days	1	1
18 days	2	1
20 days	1	—
22 days	1	—
Of indefinite duration	13 (Cases 1, 2, 4, 5, 6, 7, 9, 17, 29, 31, 38, 40, and the last of the salicin series)	3
Affording no evidence as to treatment	2 (Cases 26 and 32)	—
Total	60	21

(Cases 2, 4, 6, 9, 38, 40) or the persistence of pyrexia may be fairly attributed to the presence of pericarditis (Cases 1, 5, 7, and the last of the salicin series). Of the remaining two cases (29, 31) it is to be noted that in one the joint-pains, and in the other the pyrexia rapidly disappeared.

Dr Greenhow, however, had laid down the rule that "no patient should be put on the treatment with salicin or with salicylic acid until he had been from twenty-four to thirty-six hours in the wards, and then only if it seemed clear that the illness was running an acute course." His object in giving these instructions was to exclude such cases as would, without any medicine, "improve rapidly after admission into the hospital, and become convalescent in three or four days." His plan, however, was not always strictly carried out, and the consequence is that he dismisses as valueless no fewer than twelve cases in which the subsidence of symptoms under treatment was most rapid. But surely there is no evidence that so large a proportion as this (in addition to an indefinite number of other cases withdrawn from treatment on the ground of their mildness) would get well in from one to four days without medicine. Probably, therefore, Dr Greenhow did in reality obtain good results from salicylic acid and salicin, but in his anxiety to weigh strictly the therapeutical claims of these drugs he did them less than justice. That the sixty cases which he submitted to treatment were of more than average severity, is supported by the fact that in about twenty-five of them signs of pericarditis were discovered on admission, or within a day or two afterwards.

Treatment of hyperpyrexia. — The discovery that an excessively high temperature is the real cause of the supervention of dangerous cerebral

symptoms in rheumatism was soon followed by the employment of active antipyretic treatment. The administration of salicylic acid or of quinine fails in serious cases; and the newer drugs, as antifebrin and antipyrin, described in vol. i, p. 205, are too transient in their effects and too depressing in their action on the heart, to be either efficient or safe. The only satisfactory treatment is by the direct application of cold.

The first instance in which this treatment brought about recovery from hyperpyrexia in rheumatism seems to have been recorded by Dr Meding in the 'Arch. f. Heilkunde' for 1870; the temperature was 108·6°; the means employed were cold affusion and enemata of iced water. In the following year Dr Wilson Fox published two cases which drew the attention of the whole profession in this country. The first occurred in a woman, aged forty-nine, who was in the fourteenth day of her illness, and who had been five days in University College Hospital, when her temperature began to rise quickly. At 3 p.m. it was 105°; at 6, 106·4°; at 8.5, 107·1°; at 9.15, 108·4°; at 9.50, 109·1°. She was then completely unconscious, her pulse was imperceptible, her face cyanotic, and she appeared to be drawing the few last gasping respirations which commonly precede death. There had been delay in preparing a bath, into which, at a temperature of 96°, Dr Fox had intended to put her when her temperature reached 107°. However, she was lifted into it at 9.50, and five minutes later the temperature in the rectum was found to be 110°. With admirable courage, Dr Fox sent for some ice; two large lumps were placed, one on her chest, and the other on her abdomen; a bag filled with ice was tied down the length of her spine; two assistants baled the warmer water out of the bath, and two others poured iced water over her as fast as the pails could be filled. The temperature in the rectum gradually fell until at 10.25 it was 106·2°. The pulse now became perceptible, and some slight signs of consciousness were manifested. At 10.35 the temperature in the rectum was 103·6°, and she was removed from the bath. At 10.55 the temperature in the rectum was 100·6°; and she was able to speak. The bath had to be repeated on the following morning, but she finally recovered.

Dr Fox's second case was that of a man, aged thirty-six, in whom on the sixth day after his admission (the seventeenth of his disease) the temperature rose to 107°, having before been always below 104·5°. He showed signs of pericardial effusion, and also of inflammation at the bases of both lungs; he coughed and expectorated thin mucus stained with blood. This did not prevent Dr Fox from having him placed in a bath at 89° for twenty-five minutes, during which time it was cooled down to 86°. The rectal temperature fell from 107·3° to 103·1°; and after removal from the bath became normal. He ultimately got well, after eight baths in all.

During the time which has passed since Dr Fox recorded his cases, the treatment of hyperpyrexia by cold has been repeatedly adopted, and with very satisfactory results. A striking instance occurred in the person of one of the pupils of Guy's Hospital, who in 1875 had a very severe attack of acute rheumatism, during the course of which his temperature on twenty-six occasions, from the ninth to the twenty-fifth day of March, rose to a point between 105° and 107·2°, and was each time brought down by immersion in an iced bath. He recovered, and is now engaged in medical practice. Full particulars of this case may be found in a paper in the 'Liverpool Medical Reports' for 1876, by Mr F. T. Paul, who was house

physician at Guy's Hospital at the time, and who carried out the treatment ordered with unwearied patience and determination.

Unfortunately, even when the bath is perfectly successful in lowering the patient's temperature, it does not always restore his consciousness, still less save his life. Thus Mr Paul records the case of a man aged thirty-two, one of the porters in Guy's Hospital, who after a week's illness with rheumatic fever, became extremely delirious and then comatose, and was found at 9 p.m. with a temperature of 108·8° in the axilla. As he lived out of the hospital, there was a delay of at least an hour before a bath could be procured. When he was put into it his temperature was 110·9°, and he was violently purged. The bath was at 90°, and he was kept in it for thirty-five minutes, during which time it was reduced to 66° by cold water. His temperature on removal was 106·6°; he was still perfectly insensible, with contracted pupils and with noisy and rapid breathing. Half an hour later the temperature in the rectum was 101·3°, and an hour after the bath it was 99·3°. Subsequently it rose slightly, but it never reached 103°. He died in the afternoon of the following day, the only change being that the contracted state of the pupils changed to wide dilatation.

Another instance, which may also be found in Mr Paul's paper, is that of a woman who died after having had twelve baths during a period of nine days. For the last two or three days mucous râles were audible widely over the chest; but at the autopsy nothing was found, except a little broncho-pneumonia at the bases of the lungs, and some mucus in the tubes. We have had five other cases at Guy's Hospital, which ended fatally, notwithstanding that the hyperpyrexia had been overcome by baths; in only one of them did the *post-mortem* examination reveal an adequate cause of death in severe pleurisy with pericarditis. Such patients seem generally to sink by failure of the circulation. Indeed, in both of Dr Wilson Fox's successful cases it was deemed necessary to give large quantities of brandy after the baths, and also to apply hot bottles to the feet and warmth to the back; his first patient took six ounces of brandy within an hour.

In all probability the best method of averting collapse after hyperpyrexia is to have recourse to a bath early, before the heart and the tissues generally have been too much damaged by the heat. When the bath is too long delayed there is always some risk of death during immersion, a mishap which has twice occurred at Guy's Hospital, once in 1874, and again in 1877. When the temperature is rising for the first time to a dangerous height, it should not be allowed to reach 106° before the bath is used. The necessary preparations should be made as soon as the thermometer indicates 104·5°, and if convulsions occur the patient should be immersed at once. Subsequently, the bath should be repeated as often as the temperature rises to 105·5°. It is best to let the water have a temperature of 90° at the time when the patient is immersed; if it be much colder than this he is likely to shiver and complain; whereas when it is from 90° to 100° he often finds it exceedingly pleasant, so that he will afterwards beg to have the bath repeated. He should be lowered into the water upon a sheet. As soon as this has been done, the temperature of the bath should be reduced to 75°, or even to a still lower point, by the addition of ice, which is more convenient than cold water, because it occupies less space, so that no baling out is required. The patient should not be left in the water after the temperature in his rectum has reached 102°, as it will continue to fall after his removal. When he has been

lifted back upon the bed, he should be lightly covered with a blanket, and allowed to sleep.

The milder methods of employing cold, which were described in the treatment of enteric fever (vol. i, p. 204), sponging, wet packing, &c., are insufficient to meet the unusual danger of excessive temperature in rheumatism. The only efficient substitute for the cold bath, if circumstances make its administration impossible, is rubbing the patient's body with ice.

Treatment during convalescence.—The treatment of rheumatism, apart from the special necessities of hyperpyrexia or other complications, is much simplified by the good effect of the salicyl treatment. Relief is so soon obtained that in most cases we need only guard our patient from a relapse.

With this object, our duty is, first, to keep the patient in bed for many days after the fever has left him and his joints are free from pain; and secondly, to feed him still on strictly low diet, and only when his temperature is normal on farinaceous food. Many physicians believe beef-tea to be injurious, and undoubtedly no meat nor even fish should be allowed for a week, or, better, a fortnight after convalescence is established. Nor should the patient be on any account allowed to leave his bed until the same period has elapsed. This rigorous system is often difficult to carry out, but one has rarely been tempted to relax it by the entreaties of the patient without regretting one's compliance. Moreover, in order to prevent a relapse, salicylates in less frequent doses should be continued for three weeks or even longer. When convalescence is well established, and before the specific drug is left off, it is usually well to administer tincture of steel to combat the anæmia which usually follows rheumatism, but it is probably injurious if given before a week or ten days of freedom from pain and fever have elapsed.

The only exception to the above rules which should be allowed is in the case of patients who have suffered repeatedly from the disease, and whose hearts are already damaged beyond hope of repair. The pyrexia in these cases is often moderate, and the pain not severe; but relapses are frequent, and the cardiac symptoms are more important than those of the rheumatism. For such patients strong soups, eggs, and wine or brandy are often harmless and useful, while steel and digitalis may be given with benefit to the heart and with no apparent ill-effect on the rheumatism.

GONORRHŒAL SYNOVITIS*

Distinction from rheumatism—Sex and age—Relation to gonorrhœa—Distribution —Symptoms—Sclerotitis—Prognosis—Diagnosis—Pathology—Treatment.

Sir Astley Cooper's 'Lectures on Surgery,' published in 1824, seem to contain the earliest notice of the fact that gonorrhœa may be followed by a painful disease of the joints.

This is commonly called "gonorrhœal rheumatism," but the editor, in a paper in the 'Guy's Hospital Reports' for 1874, proposed to term it *gonorrhœal synovitis*. Whether that name or gonorrhœal arthritis be preferred, we shall see that it differs in origin, course, prognosis, and treatment from true rheumatism described in the preceding chapter. The relation between the affection of the joints and that of the urethra is observed far too frequently to be merely accidental. Moreover, we shall see that the clinical course, concomitants, and sequelæ of the disease are quite characteristic and distinct from those of any other articular lesion.

Ætiology.—The present writer collected twenty-nine cases from the medical records of the hospital in 1870–72; and during that time many others must have presented themselves in the surgical out-patient room.

All these cases occurred in men; Mr Brodhurst, however, in 'Reynolds' System,' says that he has met with a few instances in women, and Mr Davies-Colley and Dr Church have since observed the same. Senator, in 'Ziemssen's Cyclopædia,' suggests that the toughness and thickness of the vaginal mucous membrane may afford an explanation of the rarity of gonorrhœal synovitis in the female sex. It would be interesting to know whether urethritis is always present in the exceptional cases when it occurs in women; in some it undoubtedly is.

Since the twenty-nine cases above mentioned were published, more than eighty others, sufficiently reported to be available, have been admitted into our medical wards, and they have been abstracted for the editor by Mr T. F. Ricketts. Of these, seven occurred in women (two being open to some doubt), and seventy-six in men. Adding the twenty-nine earlier cases, we have a total of 105 male and seven female patients.†

These 112 patients were, with few exceptions, in early adult life; 11 were between sixteen and twenty; 66, or more than half, were between twenty and thirty; 30 were between thirty and forty; 4 were between forty-one and forty-seven; and one was fifty-two.

A urethral discharge was present in every case, but it was often not recognised until looked for to explain the other symptoms, when a gleet was

* *Synonyms.*—Gonorrhœal Rheumatism, Arthritis, or Syndesmitis.—*Fr.* Rhumatisme blenorrhagique.—*Germ.* Tripper-rheumatismus, Trippergicht.

† Mr Burghard, the Surgical Registrar at Guy's Hospital, has abstracted the reports of a considerable number of cases of articular disease, combined with urethral or vaginal discharges, of which twenty-one are sufficiently complete to make it tolerably certain that they were examples of this disease, and not of accidental coincidence of gonorrhœa with inflammation of a single joint. Of these twenty-one surgical patients, seventeen were men and four women.

always found. The ætiological relation of the synovitis was again and again proved by the patient having previously suffered from "rheumatism" after gonorrhœa, sometimes three and even four times. Among our 112 patients, eighteen gave a history of a similar painful affection of the joints with a previous attack of gonorrhœa; fourteen had twice, and one had three times, suffered in the same way before.

Sir Benjamin Brodie recorded an instance in which, after two attacks of synovitis from gonorrhœa, two later ones were attributed to irritation of the urethral canal by the use of a bougie.

According to Mr Brodhurst, exposure to cold and wet may be an exciting cause. He mentions the case of an officer serving in a tropical climate, who while suffering from gonorrhœa slept out of doors until after sunset, and woke in such pain that he could with difficulty be removed to bed.

In fourteen of our 112 cases the patient had previously experienced what was called rheumatic fever; but in two of them this was pretty clearly the same complaint as that from which he was then suffering, and in all but one there was no sign of cardiac disease—an improbable exemption after rheumatism at an early age. With a disease so common as rheumatism in children and young adults, its occasional occurrence previously to gonorrhœal synovitis is surely a mere accident.

In a considerable number of cases there was a history of "rheumatism" or "gout" in the parents or brothers and sisters of the patient. In thirteen they were "rheumatic" or "subject to rheumatism," in ten the father was "gouty," or had "rheumatic gout," and in seven cases a near relation had suffered from "rheumatic fever" or "acute rheumatism." Whether this shows any predisposition to inflammation of the joints generally—an "arthritic diathesis"—is very dubious.

The length of time which elapses between the commencement of the gonorrhœa and the development of the synovitis is put by Mr Brodhurst at from ten days to three weeks; but we found it to range from three or four days to six months, most often falling between one week and three months. Sometimes the urethral discharge continues to be profuse after the appearance of the joint-affection, but in most instances there remains only a slight gleet. Indeed, the patient is very apt to omit all mention of its presence, or even to deny that he has had any venereal complaint.

Locality.—Writers generally say that the knee is the most frequent seat of gonorrhœal synovitis. But at Guy's Hospital it has long been taught that the feet are the parts most apt to suffer, and this was borne out by a careful analysis of the first twenty-nine cases above mentioned; for in more than twenty of them the ankle, sole, heel, or instep was attacked, while the knee was affected in only fourteen, the wrist in six, the shoulder in three, the hip and the elbow in one each. This pain in the heels and soles of the feet has to be inquired after, for it is not attended by swelling or other signs of inflammation. Hence it is often overlooked in reports, while stress is laid on the more obvious changes in the knee and elbow. The "plantar fascia" is the traditional seat of these pains, which are almost always worse at night, and of an aching, grinding character; but it seems probable that the local lesion here, as elsewhere, is in the synovial membrane and ligaments of the joints.

However this may be, the much larger number of cases collected between 1873 and 1887, including the twenty-one from the surgical wards, give results more in accordance with general belief, so far as the pre-eminence of the knee

joint is concerned. For among 102 patients, one or both knees were affected eighty-two times, the ankle fifty-five times, the sole, heel, instep, or metatarsus thirty-seven times, and the hip twenty-six times. Of the joints of the upper extremity, one or both shoulders were affected thirty-four times, the elbow twenty-two, the wrist twenty-six, the metacarpus (and occasionally the fingers) twenty-five, and the sterno-clavicular joint twice. In six cases the cervical vertebræ suffered, and in two the temporo-mandibular joint.

A healthy, well-built young policeman aged twenty-one, who was in Philip Ward for nearly three months in 1885, had almost every joint in the body successively affected: first the ankles, heels, and instep, then one knee, then the elbow, shoulder, temporo-maxillary, atlanto-axial, and probably the cervical articulations.

The great toe is never affected alone; but there is often pain referred to the metatarsus generally and to the smaller toes, which usually escape in gout. The joints of the fingers are very seldom attacked, a point of distinction from subacute cases of osteo-arthritis in young subjects.

There is no rapid "metastasis" from joint to joint, as in true rheumatism; each suffers for many days, or even weeks. There is less local tenderness than might be expected from the pain, and but little redness or œdema; but there is almost always effusion to be detected in the larger and more superficial joints, particularly in the knee.

In a certain number of cases one joint only is attacked, but this is certainly rare; when it does occur, one knee or one ankle seems usually to suffer. But often, after the other joints are well, one remains as bad as ever, and this appears to be most frequently the elbow or the shoulder.

Symptoms.—The *pain* of gonorrhœal synovitis is usually worse at night, and is always described as of a dull, constant, aching character.

The *pyrexia* is almost always moderate, and in some instances the temperature remains normal. Hyperpyrexia is unknown.

This disease differs much from true rheumatism in the fact that even in young subjects it shows little or no tendency to produce *cardiac inflammation*—pericardial or endocardial. Among the 112 cases on which this chapter is based, there was in two a slight systolic basic murmur heard for a time, probably pulmonary and anæmic, in three there was a systolic apex murmur (described as doubtful in one case), and in one only a diastolic, presumably aortic, bruit, which, however, disappeared in a day or two. In no case does it appear that permanent cardiac lesions have resulted from gonorrhœal synovitis, however young the patient and however protracted the disease.

It is, however, frequently accompanied by *catarrhal ophthalmia*. Thus, what first drew Sir Astley Cooper's attention to the subject seems to have been the case of an American gentleman, who came to him on account of a gonorrhœa, and said that two previous attacks had each led to inflammation of the eyes, and a few days later to swelling of the joints. Precisely the same sequence occurred on the third occasion, under the observation of Sir Astley himself. The writer has observed this affection in eleven out of nineteen cases under his own care, and it was noted twenty-one times in the eighty-three cases collected by Mr Ricketts. It usually attacks first one eye and then the other. It is commonly attended with injection of the small radiating vessels of the sclerotic which surround the cornea, and sometimes with marked iritis. It usually subsides in a few days, under treatment by cold bathing, covering from light, and applying atropine drops; but in more than one instance it returned, after having disappeared. This *sclerotitis*, as

it may be distinctively called, is quite separate from the gonorrhœal ophthalmia which results from inoculation with gonorrhœal pus, and also from syphilitic iritis and choroiditis.

Events.—Gonorrhœal synovitis commonly runs a tedious and protracted course, lasting for several weeks, and even for months, notwithstanding treatment. In hospital practice the obstinacy of a supposed "subacute rheumatism" has often led to the discovery of the fact that the patients had gleet.

Recovery seems to have occurred in each of our 126 cases, although some of the patients left the wards before they had entirely lost the pains and stiffness of their joints.

But Mr Brodhurst mentions one instance in which the hips, the knees, and the jaw were all ankylosed, and another in which in the course of five years the whole skeleton became fixed, including even the articulations of the vertebræ and those between the atlas and the occiput, so that the head itself could not be moved. That patient had had three several attacks of gonorrhœa, each followed by "rheumatism." About six months after the last attack, and when he was only just able to walk about, he was unfortunate enough to marry a woman who had an occluded vagina, so that attempts to consummate the marriage were unsuccessful. Very soon afterwards, although there was no urethral discharge, the articular inflammation recurred; and on this occasion it led to the terrible results already described.

Though peculiarly tedious and obstinate, gonorrhœal synovitis when once cured does not return—another difference from true rheumatism. Numerous instances, however, prove that a fresh urethritis will produce a fresh synovitis, and in this way the same patient may suffer twice, thrice, or even oftener from "gonorrhœal rheumatism."

Fibrous ankylosis is not unfrequent, especially in the shoulder and elbow; but when the adhesions have been broken down under chloroform, the joint generally regains its usefulness. In the more protracted cases it is not very uncommon for eburnation and the other changes of osteo-arthritis to supervene, but the deformity is only slight.

Diagnosis.—The distinction from *gout* depends on the age of the patient, the absence of tophi, the distribution of the articular lesions and the freedom from return, except from a fresh urethral excitation.

It is more difficult to distinguish gonorrhœal synovitis from *rheumatism*, with which it is still commonly confounded. The ætiology, the different relation to sex and age, the more continuous instead of transient affection of the same joints, the much longer course, the freedom from relapses or spontaneous recurrence, the affection of the eyes and the immunity of the heart—are amply sufficient grounds for the pathological distinction between the two diseases, and with care and attention are almost always sufficient for a right diagnosis in practice. The difference in prognosis from both rheumatism and gout, and the no less important difference in treatment, make the discrimination between these diseases of great importance.*

The absence of pyrexia and the normal state of the other organs, together with the fact that the joints do not suppurate, are distinctions from *pyæmia* no less than from rheumatism. There is perhaps more chance of gonorrhœal synovitis being confounded with the much more chronic and persistent affection to be described as *arthritis deformans* in the next chapter.

* See an excellent paper by Dr Thomas Bond published (before the editor's paper in the 'Guy's Reports') in the 'Lancet,' March 23rd, 1872.

It is a question whether we ought to distinguish the cases above described from the much more severe and localised inflammations of a single joint following gonorrhœa, which come under the notice of the surgeon rather than the physician. They have been described by Duplay and Brun in France ('Arch. Générales de Médecine,' 1881), and by Mr Davies-Colley in England ('Guy's Hosp. Rep.,' 1882). In both articles they are accounted as belonging to gonorrhœal synovitis, but Mr Colley proposes to distinguish them as acute gonorrhœal arthritis or syndesmitis. The joint is very prone to ankylosis.

However this may be decided by further experience, there is no question that we must exclude from the disease now under consideration the rare cases of true pyæmia arising from gonorrhœa, with suppuration of the affected joints, of which an example was recorded in the 'Pathological Transactions' for 1885, by Mr Pollard.

Pathology.—This is quite unknown. Gonorrhœal synovitis, whether in its milder or in its more severe forms, is certainly not a modified pyæmia, a process from which it differs in every point. Nor is it a "hybrid" or "modification" of gout or rheumatism. It is conceivable that it may be in some way connected with the abundant nervous distribution over the prostatic part of the urethra, and so related on the one hand to the rigors which sometimes follow the passage of a catheter, and to the arthropathies of tabes on the other. But this is mere speculation. The micrococcus of gonorrhœa has been found in the effused serum in the knees.*

Treatment.—Some authors recommend iodide of potassium in full doses, up to thirty or forty grains daily, but it frequently fails even in larger amount. The patient must be kept in bed and upon light diet during the first week or two. Mr Brodhurst insists upon the importance of placing the affected joints upon splints, and says that leeches sometimes do harm rather than good. He has seen the Turkish bath very useful, and instances a patient who was lodged in a house attached to one of these baths, so that he could be carried down into the hot chamber every day; when profuse perspiration was obtained, the pain, which was very acute, left him for the time. Opium may be given in sufficient doses to relieve the pain and procure sleep. It probably is useful in other ways also. No other drug, and notably neither colchicum nor salicin, is of much value. Good feeding, bark and nitric acid, quinine, and a moderate use of alcohol, particularly porter, are necessary as soon as the first severity has subsided. It is most important that every effort should be made to cure the urethral disorder.

In chronic cases, blisters may be applied with advantage, or liniment of iodine, or mercurial ointment. When one or more of the joints has become fixed, it is often advisable to give chloroform and to break down the adhesions by force.

* Petrone and Kammerer, quoted by Eichhorst

OSTEO-ARTHRITIS *

History and nomenclature—Pathology and relation to gout and to rheumatism—Anatomy of the joints—Symptoms and course—Acute cases—Resulting deformity—Involution and recovery—Ætiology—Prognosis—Treatment by drugs and external applications, by diet and climate.

Charcot's arthropathie ataxique, its relation to osteo-arthritis and tabes dorsalis.

THIS remarkable disease appears to have been first recognised by Sydenham. He says that rheumatism, when free from fever, is often called arthritis (*i. e.* gout), though really distinct from it :—"Unde forsan petenda est ratio cur tam sicco illum pede transiverint scriptores medici : nisi forsan arbitremur hanc morbi speciem ad reliquam malorum Iliada de novo accessisse." He goes on to describe the chronic course of the disease, with its remissions and exacerbations, and concludes his description as follows :—"Potest fieri ut æger omni membrorum motu ad mortem usque privetur, digitorum articulis quasi inversis, et protuberantiis, ut in arthritide, nodosis, in interna magis quam externa digitorum parte se prodentibus : stomacho nihilominus valeat, et cætera sanus vitam toleret."† Since that time the affection has been described by Heberden in 1782 as "the chronical rheumatism," and by Haygarth in 1805 as "nodosity of the joints," by Cruveilhier as "arthrite avec usure des cartilages articulaires," by German pathologists as "arthritis deformans," by many English authors as "rheumatic gout," by Adams in his beautifully illustrated monograph as "chronic rheumatic arthritis," and by Garrod as "rheumatoid arthritis." It is also supposed to correspond to the "poor man's gout" (*arthritis pauperum*) of older writers; but true gout is far from uncommon among the poorer classes of this country, so that the name was probably never very applicable. In popular language this disease is generally called "rheumatic gout." Objection may be made to all the above names, and it is perhaps best to adopt the term *osteo-arthritis*, which is used in the "nomenclature of diseases" of the College of Physicians, and in the Registrar-General's Reports. The course is most often "chronic," but sometimes subacute.

Pathology.—There are still some pathologists who maintain that all these joint-diseases, including even acute rheumatism, are closely related to one another, and in common depend upon what is termed an *arthritic diathesis*. This view is held by most French writers, and in this country by Mr Hutchinson, who thinks that he can identify an "arthritic" iritis, and even an "arthritic" pneumonia, by their recurring again and again in the same individual. He brings forward instances in which different members of the same family, belonging to successive generations, are said to have suffered from different "arthritic" affections; but one cannot attach exact signifi-

* *Synonyms.*—Arthritis deformans, Nodi digitorum, Malum articulorum senile—Chronic rheumatic arthritis, Rheumatoid arthritis, Rheumatic gout, Irish gout, Chronic rheumatism (in part).—*Fr.* Rhumatisme noueux, Arthrite sèche.—*Germ.* Arthritis nodosa sive deformans, Deformirende Gelenkentzündung.

† 'Obs. Med.,' sec. vi, cap. 5 (p. 256 of Syd. Soc. Ed.).

cance to a statement that a mother, or an uncle, or a grandfather had "rheumatic gout," or "chronic rheumatism," or even "gout," unless we have a more detailed account of the case than can generally be obtained.

It is, indeed, certain that the lesions characteristic of gout and those which belong to arthritis deformans are sometimes found in different joints of the same person, and still more rarely, even in the same joint; but, occasionally, this might be expected as a mere coincidence. Between 1874 and 1879 four examples of this association were met with in the autopsies at Guy's Hospital. Three of the patients were men, aged fifty-two, fifty-four, and sixty-two respectively. The fourth was a woman, aged thirty-six; and it is an interesting fact that she was said to have had rheumatic fever at the age of twelve, which in all probability was really the case, for she died of mitral stenosis. In each instance the great toe-joints contained urate of soda, showing that gout had been present, while the lesions indicative of arthritis deformans were found in the knees or in the hip-joints; once the left knee showed both kinds of change, the right that of arthritis deformans alone, and the two great toes that of gout alone. In a fifth instance the editor observed the edges of the patella thickened, and other signs of osteo-arthritis present, together with patches of urate of soda.

Again, there is no question that osteo-arthritis is sometimes preceded and apparently caused by a single attack or more often by a series of attacks of rheumatic fever. In the vast majority of cases true rheumatism leaves no deformity behind, and this is a most remarkable and distinctive point in its natural history. But that the synovitis of rheumatism, when unusually protracted, or when frequently repeated, should produce no structural change would be most improbable.

The same results are less rarely produced by gouty arthritis, and still more frequently by gonorrhœal arthritis, because in both there is a far more persistent inflammation than in true rheumatism. They are also produced by traumatic synovitis or by consecutive irritation, as in a case reported by Mr Hutchinson in the 'Med. Times and Gaz.' for 1881, of a young woman whose thigh was amputated by Mr McCarthy for a myeloid growth in the tibia, when bony lips were observed on the edges of the condyles of the femur, doubtless as the result of irritation caused by the proximity of the tumour. So that if we define osteo-arthritis by the peculiar anatomical condition of the joints to be presently described, it may be the consequence of rheumatic, gonorrhœal, gouty, or traumatic arthritis.

Nevertheless, it remains true that in the great majority of cases this characteristic multiple articular lesion does not follow either rheumatism or gout, that in fact such a sequence is exceptional and rare, and that, on the contrary, it far more often appears independently of any other disease. Sometimes it may be traced to hard usage of the affected joints; more frequently it depends upon the ordinary wear and tear of laborious work during many years; and often it appears in young subjects who have never worked hard and have never suffered from any form of synovitis.

Moreover, the primary or idiopathic anatomical change in the joints is associated with a definite and constant pathological process, and with characteristic clinical symptoms. It has its own course, prognosis, and treatment, and therefore is, on every ground, practical and theoretical, entitled to be considered as an independent "disease."

Anatomy.—At the commencement of arthritis deformans the morbid process is often limited to a very small area of one or both of the carti

lages of a joint. Sometimes it begins at the centre of the cartilage, sometimes (as in several specimens exhibited to the Pathological Society by Mr Hutchinson in 1872) it begins round the margin, and spreads irregularly inwards.

The cartilage first becomes soft and velvet-like in appearance; afterwards it ulcerates, so that there is formed in it a cavity, with a more or less sharp edge, in the floor of which the bone may be exposed. Histologically the change consists in a proliferation of the cartilage-cells, so that each becomes replaced by from eight to twenty large corpuscles; the matrix at the same time splits into fibres in a direction perpendicular to the articular surface. Presently the enlarged capsules rupture into the cavity of the joint; the fibres then remain for a time as shaggy projections, until ultimately they too disappear.

The denuded bone is said occasionally to exhibit an open cancellous tissue, but as a rule it is converted into a very hard, compact substance, or, to employ the usual term, it undergoes *eburnation*. When, after the removal of the whole of the articular cartilages, the osseous surfaces everywhere come into apposition, with no soft material between them, they become scored and fluted with parallel grooves and ridges, corresponding in direction with some particular line of movement, to which they are henceforth restricted. The texture of the subjacent part of the bone becomes wasted, filled with oil drops and so gradually absorbed. Thus the neck of a femur, for example, may be gradually shortened, until what represents the head lies in a hollow between the two trochanters.

Intra-articular fibro-cartilages, as in the temporo-maxillary joints, resist the disease no better than the cartilages which cover bones. So also the ligamentum teres becomes lost when the hip is affected, and in the shoulder the long head of the biceps disappears; it seems to undergo fusion with the capsular ligament of the articulation, so that it cannot be traced upwards beyond a certain point, and yet no definite free end can be found.

Formative changes are generally, though not always, associated with the destructive process from a very early period. From the edges of the articular cartilages there arise a series of nodulated outgrowths—or "ecchondroses," as they are termed—which form a raised lip or border. These are at first small, but they afterwards increase in size; and soon lime salts are deposited in them, so that they become converted into bony plates or masses (*osteophytes*), which not infrequently grow into the capsular ligament, or into the tendons round the joint, and unite to form a complete osseous shell around the joint. In like manner, outgrowths from the edges of the bodies of the vertebræ often cohere together across the intervertebral discs, so as to constitute a number of bony splints, which may immoveably fix a large part of the spinal column.

The synovial membrane of the affected joints is more or less thickened, especially near its lines of attachment to the bones. The folds, which project into the joint-cavity, are greatly enlarged and very vascular; they often form long villous processes, with numerous bodies like melon-seeds hanging from them: these bodies may ossify, become detached, and lie as "loose cartilages" in the joint. They cause fresh accession of pain, and are named *Mäuser* by the Germans, from their slipping away from the touch.

A modification of the bony outgrowth of arthritis deformans was long ago described by Heberden under the name of *digitorum nodi*, which, as he says, consist of little hard knobs, about the size of a small pea, situated

upon the fingers, particularly a little below the top, near the joint; they have no connection with gout, continue for life, are hardly ever attended with pain, and are rather unsightly than inconvenient ('Comment.,' cap. 28).

These various changes are attended with a gradual loss of mobility, and at length the joints become perfectly fixed by the bony splints around, although when these are removed after death the articular surfaces are found perfectly free and smooth. Neither bony union nor suppuration ever occurs, but occasionally fibrous ankylosis is established.

At an advanced stage of arthritis deformans the deformity produced by it is generally very characteristic. The joints, as a rule, are fixed in a bent position. But the wrists are commonly extended. The fingers almost always lie at an angle with the rest of the hand, and are deflected to the ulnar side, so that the knuckle of the forefinger projects towards the thumb. Some of the phalangeal joints are usually over-extended, so as to be concave on the dorsal aspect; this feature of the disease was noted by Sydenham. The degree of enlargement is very variable. Sometimes each articulation forms a bulbous swelling; sometimes the ends of the bones are almost of normal size.

Distribution.—The fingers and wrists are most commonly first attacked (in about two thirds of the number of cases), then the knees. The hip may be affected alone, but only in the later periods of life (*morbus coxæ senile*). Next to the hands and knees, the elbows and shoulders are most often obviously deformed, the feet less frequently. The vertebræ are, however, very often found united by osteo-arthritis after death, and this is one cause of the stooping back and stiff neck of old people in the workhouse. Adams figures the disease well marked in the temporo-maxillary articulation, and it is stated to affect the bones of the ear and the ossified laryngeal cartilages of old age.

Symptoms.—The early stages of arthritis deformans may be attended with no subjective symptoms whatever. This fact is well illustrated by one of Mr Hutchinson's cases in the 'Pathological Transactions.' A man, aged forty-one, had his thigh amputated for destructive inflammation of the knee, it not being known that any other joints were affected; but the cartilage was found eaten away in every articulation of the foot, except the distal joints of three toes. The same writer speaks of having frequently been able to detect the presence of a projecting lip round the articular cartilage of the lower end of the femur, in persons who are not aware that the knee had ever suffered. He places himself in front of the patient, puts the finger ends of one hand flat upon one condyle, and those of the other hand upon the other condyle, and then directs him to bend and extend the joint slowly several times in succession. In this way, he says, the edges can be easily found, and the degree of their elevation estimated. He admits, however, that practice is required to prevent one mistaking for a morbid condition a ridge which normally exists at the same spot in many healthy persons.

Dr Spender, of Bath ('Brit. Med. Journ.,' 1888, vol. i, p. 781), who has seen large numbers of patients in the early as well as in the chronic stages of osteo-arthritis, believes that it is marked at or even before its development in the joints by a rapid and incompressible pulse (90 to 110 or 120), with cold hands and feet and no rise of temperature. He has also noticed as an early symptom pigmentation of the skin, particularly on the forehead

and face and on the fingers. Maculæ occur later over the legs, and freckles on the face. A constant dampness of the hands from perspiration, and liability to fits of neuralgic pain in the affected limbs, are other early symptoms on which Dr Spender lays stress.

It is probable that when a patient begins to complain of pain and stiffness in one or more of his joints, the disease has often been already present for a considerable time. The pain is not generally constant; it "comes and goes," sometimes without obvious cause, sometimes in apparent relation to changes of the weather. In certain cases it has a shooting character, so as to resemble neuralgia. With many patients it is worse when the limbs are warm, in others when they are cold; in some by night, in others by day. It is very apt to be brought on by the use of the part; in the hip, for example, by walking even a short distance; in the shoulder, by carrying anything, however small, in the hand. The stiffness, however, is apt to be more noticeable after rest, as when the patient first attempts to get out of bed in the morning. A sense of weakness and of distressing fatigue may be as marked a symptom as pain; and there is often far more wasting of the muscles than seems to be accounted for by mere disuse of the limbs; the thenar and the hypothenar eminences, for example, may be so hollowed that the case looks like one of progressive muscular atrophy.*

Another very characteristic symptom, which, however, is not present during the early stage of the disease, is creaking or grating, which accompanies the movements of the affected joints; it can be plainly felt or even heard by the patient, and is easily recognised by grasping the joint with one's hand.

In some cases osteo-arthritis sets in from the first with well-marked symptoms; it produces swelling, heat, and even perhaps redness of the affected joints, and is attended with more or less pyrexia; so that Garrod describes an *acute variety* of the disease. which, he says, very closely resembles ordinary acute rheumatism, differing only in the greater length of the paroxysm, in the absence of profuse sweating, and in its having no tendency to attack the heart;† and Mr Hutchinson declares that it sometimes gives rise to paroxysms as short and as definite as those of gout itself. In one of Dr Ord's cases the temperature one evening rose to 102·8°.

There is no doubt that osteo-arthritis is very often attended with effusion of fluid into the affected joints, and that the designation of *arthritis sicca* is altogether inappropriate. Many cases of so-called *hydrops articuli* really belong to this disease. Analyses of the effused fluid from the hip-joint in arthritis deformans have been made by Hoppe-Seyler, and recorded in 'Virchow's Archiv' for 1872; he found a proportion of mucin which greatly exceeded that in normal synovia.

In other cases hæmorrhage takes place into some of the affected joints. Thus in 1875 Dr Goodhart, in examining the body of a man aged fifty, who had been admitted for arthritis deformans, but had died of hernia, found that each ankle contained several drachms of liquid blood,

* "The chronical species of rheumatism equally partakes of the palsy, for there is always a trembling, weakness and numbness left for some time in the limb affected, and the use has at last in many been wholly taken away" (Heberden, 'Comment.,' cap. 79).

† Trousseau states that in four out of nine autopsies of "nodular rheumatism" made at the Salpêtrière by Cornil, pericarditis was present. It is, however, incredible that this could be the result of a disease which rarely, if ever, produces cardiac lesions; and on looking into the cases it is at least probable that chronic cirrhosis of the kidneys was the real cause of the pericarditis which ended some of these long-standing cases of arthritis.

and that there was also blood in both knees, the synovial membrane being greatly swollen, with rounded vascular œdematous fingers; all these joints, as well as the hips, which contained no blood, showed the characteristic changes of arthritis deformans (see also 'Path. Trans.,' vol. xxvi, p. 162).

Among the less common symptoms of arthritis deformans must be mentioned the presence of fibrous nodules at a distance from joints, as, for instance, among the muscles of the arms or of the forearms. These are distinct from the nodules of true rheumatism mentioned above (p. 815).*

Age and sex.—Osteo-arthritis is decidedly more common in women than in men. Of 75 well-marked cases which have come under the writer's personal observation, 48 occurred in women and 27 in men. Of Adams' 21 patients, 16 were men and only 5 women. Among 500 cases, Dr Archibald Garrod found 411 in women and only 89 in men ('Med.-Chir. Proc.,' Nov., 1887).

It is very rare in children, and not commonly met with under thirty. Of the 75 cases quoted above, 3 occurred before the patient was twenty-one years old—one in a girl of sixteen—and 7 between twenty and forty; 3 patients were above seventy years old, 7 between sixty and seventy, and all the others were between forty and sixty. In some cases, as Haygarth remarked, it first develops itself in women at the climacteric age, and it has been supposed to depend on uterine disturbances. It is, however, by no means confined to any one period of life. Garrod says that he has seen it in children of ten or twelve years, and he has also met with instances in which it began in people above seventy years of age.

Two types of this disease may be recognised, for the characters of arthritis deformans differ somewhat according to the sex and age of the patient:—the cases in which it begins in the hands and feet, subsequently spreading to the larger joints, almost always occur in women; men are more liable to have it in the hip or in the shoulder, before any other parts are affected. The latter variety of the disease is especially frequent at an advanced period of life. As Mr Hutchinson points out, the change in the affected joints is not quite the same at different ages; in persons under middle age the outgrowths of bone, which constitute a striking feature of the disease in old people, are rare; and if present they are usually small.

Ætiology.—Osteo-arthritis is the disease commonly called chronic rheumatism, which cripples the joints of old men in the country who have been exposed to wet and cold during a lifetime of hard work and often scanty food. These men have never suffered from true rheumatism; the heart is unaffected (unless from chronic atheroma), and they live to an advanced age; but they suffer much from these crazy joints, and not infrequently are bedridden from the same cause during the last years of their lives.

So far as the causes of arthritis deformans are known, they appear to differ altogether from those of gout, and to some extent also from those of acute rheumatism. Garrod was unable, after looking over a large number of cases, to find much evidence of its being transmitted by inherit-

* I once saw an old lady aged seventy-one, who for about three months had complained of a curious affection of the tongue and cheeks, which perhaps belonged to this disease, inasmuch as she also had hydrarthrosis of each shoulder-joint, and a less marked affection of her knees. The tongue was uniformly enlarged, and had a peculiar firm, fleshy consistency, without being at all indurated; its surface was rather smoother than natural. At each corner of the mouth there was a button-like mass, which extended outwards for some distance into the substance of the cheek, and on which the mucous membrane adhered more closely than elsewhere to the subjacent tissues.—C. H. F.

ance ; one member of a large family not infrequently suffers severely from it, while the rest remain free. Again, it is not produced by indulgence in rich food or in alcoholic stimulants. It is apt to occur in weakly, ill-fed women, who are exhausted by repeated child-bearing, by menorrhagia or prolonged lactation, by grief or mental anxiety.

Dr Ord, in the 'Transactions of the Clinical Society' for 1877, has brought out, more prominently than other observers, the relation of osteo-arthritis to dysmenorrhœa and "ovario-uterine provocation." In one of his patients it was regularly developed paroxysmally just before, throughout, and for a short time after each menstrual period, and underwent no less regular remissions in the intervals. He noticed three cases in which the joint-affection was limited to, or began and remained excessive in, one side of the body, while the ovary on the same side was painful and tender.

Garrod believes that tubercular subjects are especially liable to be affected by osteo-arthritis ; as also are "individuals of weak frame, whose circulation is languid, and whose extremities are habitually cold." Among thirty-four cases examined by the editor there were three in which phthisis was also present.

Connection with diseases of the spinal cord has often been suggested. A point on which Senator insists is that the parts most apt to be attacked are such as have been most used ; as, for instance, the fingers and the wrists of watchmakers, and of women who have worked hard with the needle, or at knitting. The effect of different kinds of labour in producing pressure on certain joints with "usure des cartilages," eburnation of the articular surface and prominence of its edges, has been ingeniously analysed by Mr W. A. Lane in several interesting papers published in the 'Guy's Hospital Reports,' the 'Pathological Transactions' (1886), and the 'Journal of Anatomy' (vol. xxi).

But the effects of pressure and irritation in producing the anatomical deformity of osteo-arthritis are not confined to the human race. It is interesting to find that a similar affection occurs in the feet of horses, probably as the result of overwork.*

With regard to the geographical distribution of arthritis deformans in man, no extended observations have yet been made. But Trousseau, after stating that it is "a disease of rare occurrence" (in Paris), remarks that in "certain damp countries it is so common as to be almost endemic." A circumstance of some interest is that signs of it have been detected by Delle Chiaje in bones taken from Pompeii, and by Lebert in bones from the catacombs of Paris, so that the disease is clearly not of modern origin, although it has only been distinguished of late years. For an account of a specimen from a Roman tomb by Dr Norman Moore, see 'Path. Trans.,' vol. xxxiv.

There is no question that arthritis deformans is more common in Ireland than in England or Scotland, or that it is more frequent among agricultural labourers than in towns.

Prognosis.—The patient often becomes completely crippled, unable to dress or to undress, to carry food to the mouth, or even to hold a paper in the hand.

The disease seems to have little or no tendency to shorten his life, its duration from first to last being perhaps ten, twenty, or even thirty years. Sometimes exhaustion by pain seems to be partly concerned in bringing his sufferings to a close ; but more often death, when it arrives, is directly attributable to some intercurrent affection.

In ten years at Guy's Hospital (1875 to 1884) we had nine deaths with,

* This may perhaps be the affection described by Aristotle as ἡ τῶν ἵππων ποδάγρα.

but none of them from, osteo-arthritis. One patient, who was only thirty-five, died from adherent pericardium, the result of several attacks of rheumatic fever; the changes in the joints were slight, and certainly secondary to true rheumatic synovitis.

Of the rest, the youngest was fifty and the oldest seventy-four. Two died of strangulated hernia, three of cancer, one of cerebral hæmorrhage, and the rest of chronic interstitial nephritis and its effects.

It must not, however, be supposed that the prognosis is always unfavourable, nor that all patients who become the subjects of arthritis deformans, at least in its slighter forms, necessarily continue to suffer from it for the rest of their lives. Among the author's friends was an old gentleman who, some years ago, when he must have been nearly seventy, was unable during one winter to move the right shoulder, so that he had to be shaved by his servant, and required help in many other ways. During the following summer he became free from the complaint, and it never returned. Dr Ord speaks of more than one of his patients having regained a fair state of health when menstruation became normal; but he does not say that joints which had been enlarged ever returned to their natural size.

Treatment.—The medicines most serviceable in arthritis deformans are arsenic, guaiacum, and cod-liver oil. Trousseau, following Lasègue, recommended the tincture of iodine in doses of ♏x up to xxx. Iodide of potassium is more often prescribed in England; and it is most likely to be useful when warmth augments the pain. Garrod speaks highly of the syr. ferri iodidi. Dr Ringer says that actæa (cimicifuga) racemosa has yielded him very satisfactory results, and that it is most useful when the pain is worse at night, and when the disease is traceable to uterine derangement. Given in full doses, and continued for a long time, arsenic is the most efficient drug in this disease, although no doubt cases occur in which it is useless: patients have said that as soon as their eyes begin to itch they know the pains and stiffness will be relieved. Next to arsenic, steel and bark are useful drugs. But more important than either is cod-liver oil (first introduced by Dr Laycock for "rheumatism") with porter and a generous diet. When the pain is severe, there is no objection to the administration of opium in doses sufficient to subdue it.

Hot douches, hot sand poured over the joints affected, hot baths, and blisters or other counter-irritation are all valuable. Warmth and flannel and powdered sulphur do good, cold and damp are harmful. Passive movement is necessary, and after the hot bath or douche the joints should be well shampooed. Moreover, the patient should be encouraged to persevere in exercising the affected joints, and to play the piano, or make any other exertion of the fingers.

Lastly, removal from Ireland, and if possible from England, to a warm, dry, and equable climate is invaluable. Perhaps most cases might thus be cured if the treatment could be early adopted.

A weak continuous galvanic current is sometimes applied with advantage. A narrow terminal, connected with the positive pole, may be placed over or below each of the affected joints in turn; a sponge, connected with the negative pole, higher up the limb or nearer the spine.

Baths and douches (including even cold douches) are recommended by most writers. Tincture of iodine may be painted over the swollen articulations; or they may be strapped up with plaster; or the belladonna liniment may be applied to them; or the linimentum cantharidis, which Garrod says

is more convenient than an ordinary blister. Trousseau recommends that the affected parts should be buried in hot sand, at a temperature as high as can be borne, three times a day for an hour or two at a time. In certain cases stimulating applications, such as the cajeput oil, do good. As to the extent to which the patient should use the joints Garrod suggests the rule that only such an amount of movement should be allowed as will not cause them to be more painful on the following day. The best climates are such as are warm, but dry and bracing. Drinking alkaline waters does harm; but the chalybeate springs of Tunbridge Wells or Schwalbach may sometimes be useful. The warm baths of Bath and Buxton, with internal use of the waters, are certainly often valuable means of relief and sometimes perhaps of cure.

CHARCOT'S JOINT-DISEASE.*—It is to the acumen of Professor Charcot (1868) that we owe the recognition of a remarkable chronic affection of the joints which is liable to occur in the earlier stage of tabes dorsalis (locomotor ataxia). It is sometimes abrupt in its origin, but chronic in its course and remarkably wanting in local signs of inflammation. It was first described by Charcot in 1853 as a variety of osteo-arthritis, under the title "rhumatisme noueux d'origine nerveuse."

The joint affected, after a period of swelling probably produced by intracapsular effusion, gradually becomes completely disorganised. The synovial membrane and cartilage disappear, and the articular ends of the bone undergo singular atrophy. The whole shaft of the bone becomes porous and brittle, not only the ends, as in ordinary osteo-arthritis. In the knee, which is most often affected, the tuberosities of the tibia and the condyles of the femur are absorbed, or, when the hip is attacked, its entire head and neck. With this there is little or no hypertrophy. At last the ligaments are so relaxed and the ends of the bones so altered that the joint swings in all directions like a flail. The shoulder and elbow are also liable to attack, but not, it appears, the joints of the fingers or of the vertebræ.

Dr Buzzard has called attention to the frequency of the gastric attacks of tabes when this articular lesion is present. He found them present twelve times in twenty-six cases.

The remarkable affection known as perforating ulcer of the foot has sometimes been met with in cases of Charcot's disease.

Whether this affection is a variety of osteo-arthritis deformans, and what is its true pathology, are still disputed points.

Charcot's atrophic arthritis, as we may call it, was briefly alluded to in the account of locomotor ataxia in the first volume of this work (p. 536). A discussion of its nature by Sir James Paget, Mr Hulke, Mr Hutchinson, Mr M. Baker, Dr Duckworth, Mr Lucas, and other pathologists, in which many cases were recounted and specimens shown, will be found in the eighteenth volume of the 'Clinical Society's Transactions,' and an earlier paper by Dr Buzzard ('Path. Trans.,' vol. xxxi, pp. 193, 202) should also be consulted. A good case was published by Mr Keetling ('Clin. Trans.,' 1882), two by Dr Charles Atkin in the Manchester 'Medical Chronicle,' April, 1885, and six by Dr Sydney Roberts in the Philadelphia 'Medical News,' Feb. 14th, 1885.

* *Synonyms.*—Arthropathie ataxique—Maladie de Charcot—Arthritis tabidorum atrophica—Chronic atrophic arthritis.

In Germany this condition has attracted little attention. The excellent text-book by Prof. Eichhorst, of Zurich, merely reproduces the statements of Charcot and his pupils.

In this country it is certainly remarkably rare, for it is not a late sequel of tabes, which might only be found in sick asylums and workhouse infirmaries; it belongs to the earlier stages of the disease. It is believed to be a trophic neurosis, dependent on wasting of the cord, yet it appears to be nearly as common in the upper as in the lower extremity, whereas ataxy of the arms is very rare compared with that of the legs.

The chief distinctions between Charcot's arthropathy and osteo-arthritis are the absence of pain, the rapidity of the destructive process, and the excess of atrophy over hypertrophy—"incapability of repair," as Mr Baker puts it. Whether we may regard it as osteo-arthritis modified by occurring in a tabid subject, it is difficult to say. That joints corresponding to Charcot's description may be met with in England is certain, and not less so that cases are seen of articular disease clinically combined with tabes dorsalis. But all affections of the joints which do not end in suppuration or pulpy degeneration tend towards the same result. We have seen that gout, true rheumatism when frequently repeated, gonorrhœal arthritis and traumatic synovitis, together with the mere wear and tear of long-continued labour, may each and all produce an anatomical condition of joints, with thickened lips and atrophied cartilage, which is indistinguishable from what may be called primary or idiopathic osteo-arthritis. It may be that tabes is only another cause of what is essentially the same result. But if so, this is itself an important clinical and pathological discovery.

GENERAL DISEASES AFFECTING THE BONES

RICKETS.—*History—Anatomy of the rachitic limbs—of the thorax—pelvis—skull—craniotabes—Histology—Course and event—The spleen in rickets—Symptoms—Ætiology: age, sex, climate, food—Relation to tubercle and syphilis—Pathology—Diagnosis and prognosis—Treatment, preventive and curative.*

MOLLITIES OSSIUM.—*History—Ætiology—Symptoms and events—Diagnosis—Histological and chemical changes—Pathology.*

RICKETS.*—In the middle of the seventeenth century the famous anatomist and physician, Francis Glisson, drew attention to a disease affecting the bones of children, which he supposed to have recently sprung up, where it was first observed, in the counties of Devon and Somerset. It had already become known as *the rickets*. In a thesis by Whistler (said to have been published at Leyden in 1645) the complaint was spoken of as *morbus puerilis Anglorum*, and probably this led foreign writers to call it *morbus Anglicus*. It was hence supposed to have spread from England to the Continent; but there is little doubt that it had really existed among children from time immemorial on both sides of the Channel, and it is as common in Holland and Germany at the present day as in England. Only few and doubtful allusions to the disease are to be found in older works, but there is an antique statue of Æsop mentioned by Stiebel which is said to exhibit the deformities characteristic of rickets.†

In Grant's 'Observations on the Bills of Mortality" (1662), it is noted that "the rickets" first appeared in the returns for London in the year

* This word has been derived from a verb in use in Dorsetshire: "rucket" (= to breathe laboriously), or from "rick" (= elevation or hump as "hayrick"), or, according to Trousseau, from a Norman word "riquets," applied to deformed persons, itself said to be derived from Alberiquet, dim. of Alberic, a dwarf in Gothic mythology (see Koch's paper, 'Arch. f. Gynæcologie," 1885, and Virchow's criticism in the same year, 'Archiv f. Path.,' vol. cii). But the true derivation, according to Skeats, is from *wrikken*, to "wrest," to twist "awry:" cf. a "ricked" ankle, a "rickety" chair.

The term *Rachitis* (or Rhachitis) was first proposed by Glisson, who chose that name on account of its similarity in sound, although in his work on this disease (the second edition is dated 1650) he offers to his readers a Greek root (ῥάχις), on the ground that the dorsal spine is one of the first parts to be attacked.

He says that "the rickets" is the word common for the disease in the West of England, in London, and in the Southern and Midland Counties, for in the North it is hardly known. In framing a Latin word he bore in mind the following excellent rules: "(1) Ut nomen morbi notabilem aliquam ejus conditionem comprehenderet; (2) Ut id satis esset distinctum ab aliorum morborum et symptomatum nominibus; (3) ut esset satis familiare, pronunciatur facile, memoriæ quoque accommodatum, non nimis longum, neque operosius decompositum." And the word ῥαχίτις, *morbus spinalis*, having been suggested by a friend, he accepts it as expressive of the affection of the spine, as distinctive, and as near to the vernacular, either by accident or possibly by corruption of a physician's term.

The vernacular term in French is *chastre* (castrum), and in German *doppelte Glieder*.

† A cast of this statue from the Villa Albani at Rome has been lately added to the collection of antique casts in the South Kensington Museum (No. 229). It represents rather the effects of extreme angular curvature of the spine than of true rickets. Two of the children in Glisson's frontispiece also have cyphosis not obviously rachitic.

1634, when fourteen deaths were ascribed to it. In 1658 the number was 476 (for the disease was better recognised), and in 1659 it was 441.

Rickets may be defined as a defective and perverted development of growing bones, attended with an enlargement of certain parts, and leading to a distortion of their shape. In its extreme form it affects the whole skeleton of the child, but it often begins in some particular region, and it may remain limited to the chest, or to the head, or to some of the limbs, at least so far as its more obvious manifestations are concerned.

Anatomy.—In the arms and legs the earliest sign of the disease is an increase in size of the ends of the long bones. This is particularly marked at the wrist; the radius and ulna form a flattened pear-shaped prominence which contrasts with the small hand. Something similar may be seen in the case of the ankle. The joint of the knee appears as a hollow or depression between the projections of the articular extremities of the femur and of the tibia, and hence the expression "doubling of the joints," which has sometimes been used as synonymous with rickets. If the affection advances the limbs become curved. In the forearm the bones almost always bend, so that they are convex towards the extensor surface; in the upper arm the character of the distortion is less uniform. In the thigh the rule is that the femur is arched, with its convexity looking forwards and outwards. The knees may thus be thrown far apart, and the patient becomes bow-legged. The shape assumed by the tibia and fibula varies in different cases. They often carry downwards and inwards the curve formed by the femur on each side, so that the ankles meet one another, although the knees do not; but in other instances they are themselves bent with the convexity outwards or inwards; so that in the latter case the feet are widely separated. Sometimes each leg presents a sharp angle, projecting forwards at the junction of the middle and the lower thirds, and producing what is called the sabre-shaped tibia.

The chief cause of these various deformities appears to be the yielding of the bones to the traction and pressure to which they are subjected. They are, in fact, so soft that very little force is required to bend them, as can easily be demonstrated after death. Thus the curves in the forearms and upper arms are probably due to efforts made by the child to raise itself by laying hold of fixed objects with its hands, and to other like movements: there is often a marked angle at the insertion of the deltoid into the humerus. The more common distortions of the bones of the thighs and legs seem to be caused by the weight of the body in the erect posture, but the angular bend which is found just above the ankles is probably due to pressure transmitted to the tibiæ from the insteps and feet in crawling about upon the floor, a favourite mode of progression among these little patients.*

Another effect of the softening of the bones is that a very slight accident suffices to partially break them. Such "green-stick" fractures, as they are called, may be caused by abrupt movements; sometimes several of them are seen in the same child. Their effects, of course, complicate and alter in various ways the more regular distortions resulting from the disease, and all the more because, interfering but little with the movements of the affected part, and giving rise to no marked increase of pain, they are very apt to escape notice until a large quantity of callus has been thrown out.

* It has been objected that in stillborn fœtuses, believed to be rachitic, similar changes have been observed in the shape of the limbs. But surely they may, while within the uterus, undergo compression powerful enough to deform them; and there is no evidence that the distortions are identical with those of ordinary rickets.

Of far more importance as regards the patient's health, though perhaps less conspicuous to the eye of an untrained observer, are the changes produced by rickets in the shape of the *chest*. Here, again, the first indication of the disease is an enlargement of the growing ends of the bones; namely, of the ribs just where they join their cartilages. The consequence is the formation of a series of little nodules, which can be easily felt, and may even be seen through the integuments, and which are arranged in a vertical line, slanting outwards as it passes downwards on each side of the sternum. This "beading" of the ribs, as it is termed, is sometimes the only discoverable sign of rickets which a child may present; it must therefore be carefully looked for whenever the existence of this disease is suspected. But, further, there is in most cases a more or less considerable alteration in the form of the thorax, by which its capacity may be greatly reduced. If one watches a healthy child who is suffering from extreme dyspnœa dependent on obstruction of the larynx or trachea, one may observe that at each inspiration the middle parts of the ribs are forcibly dragged inwards. This is especially the case with those ribs which lie towards the base of the chest on each side, and cover the lungs; for the effect is not to be seen where they overlie the solid organs, the heart, and the liver. The cause of this is that the ribs are unable to resist the atmospheric pressure when they are no longer supported by the counter-pressure of air entering the lungs freely from the throat. Now, in rickets, it would seem that the mere elasticity of the lungs is sufficient to turn the scale and to prevent the lateral portions of the softened ribs from moving outwards when the child draws its breath; or it may be that this result is brought about by trifling and transitory affections of the bronchial tubes. In either case the effect is not transitory, as it would be under normal circumstances; but there arises a persistent flattening, or even a depression, of the chest walls. This generally runs, as a vertical broad and shallow groove, downwards and outwards from just below the fold of the axilla on each side; as it approaches the margins of the costal cartilages it forms an angle and slopes away to each side, now lying almost parallel with the diaphragm. Or one may describe two sulci—the one nearly perpendicular, the other horizontal—meeting at an obtuse angle near the base of the xiphoid cartilage. The vertical groove is generally said to be formed by the ribs themselves, outside their beaded ends. But Dr Gee ('St Bart. Hosp. Rep.,' vol. iv) has pointed out that the beads sometimes occupy the bottom of the groove, and that in exceptional cases they may lie to its outer side, so that, in fact, it corresponds with the cartilages only and not at all with the bones. One result of this depression of the ribs is that the higher abdominal viscera are pushed out from below the ribs; the liver projects beyond the costal margins more than in a healthy child; and, as the intestines are commonly full of gas, the belly becomes protuberant and contrasts strongly with the narrow chest. Another effect, according to Sir William Jenner, is the production of a white friction-patch on the surface of the heart, just above the apex of the left ventricle, where the fifth rib presses on it (see his well-known Lectures in the 'Medical Times' for 1860). Yet another is an increase in the antero-posterior diameter of the thorax; the sternum is pushed forwards, and the dorsal vertebræ form a rounded curve.

These changes together constitute what is commonly called the *pigeon-breast*. They are almost always associated with the presence of emphysema in the anterior edges of the lungs, beneath the projecting sternum; while

in correspondence with the flattened ribs, one may often notice a collapsed condition of the inferior edges of the lungs, and even of parts of their lateral surfaces. Another feature of the disease is that the clavicles are much more bent than in the normal state, and carry the shoulders further backwards, with the effect of increasing the apparent prominence and narrowness of the upper part of the chest.

The *spinal column* in a rachitic child becomes gibbous, curved with the convexity backward. In contrast with this *cyphosis* of the dorsal spine, the cervical and the lower lumbar vertebræ have their natural forward curves exaggerated (*lordosis*). The part especially affected is, as Mr Lane has shown in the 'Guy's Hospital Reports' (vol. xlii, p. 319), the junction of the thoracic and lumbar vertebræ, the eleventh and twelfth dorsal, and the first and second lumbar. The sacrum also becomes more flexed on the iliac bones and its promontory moves downwards and forwards.

In the *pelvis* various deformities occur, but these are not obvious during the acute stage of the disease, and are only important because in females they may permanently narrow the cavity and obstruct parturition. In most cases the brim assumes an hour-glass or oval shape, the pubes being approximated to the sacrum, but the opening may be triangular, and the symphysis pubis rostrate.

The growth of the body generally is retarded in rickets; a child two years of age may be taken for not more than six months old; a boy of twelve may be no taller than he ought to have been at three. Among forty-two cases in which Ritter von Rittershain (1863) made careful measurements at ages between four months and three years, there was only one in which the length of the body was not from one and a quarter to two and a half inches below the mean length in healthy children at the same ages. Rickety infants, however, are not infrequently fat, and sometimes excessively so.

The face is peculiarly backward in its development: the jaws remain narrow, and *dentition* is late and irregular. It is not uncommon for a rachitic infant a year old to have cut none of its teeth; and when two or more of the incisors have appeared before the commencement of the disease they are sometimes without successors for several months. The teeth themselves are imperfectly formed; their enamel is defective; in a year or two they turn black and break off, or fall out. Dr Gee has pointed out that the second dentition is also delayed.

In marked contrast with all other parts of the skeleton is the appearance of the *skull*. This is disproportionately large, so much so that until recently it was generally believed to be larger than in healthy children of the same age. Ritter von Rittershain has, however, shown by accurate comparative measurements that the enlargement is generally only relative.

With regard to the state of the brain we find some discrepancies of statement among different writers. Trousseau maintains that the softness of the cranium allows of the more easy development of the hemispheres, and so accounts for the possession by rachitic children of intellectual faculties in advance of their age—a fact, if it be a fact, which it would be more reasonable to attribute to the habits induced by their unfitness for exertion and their habitual association with adults. Dr Gee thinks that the growth of the brain is really dwarfed, like the rest of the body, and that fluid is commonly effused into the ventricles to fill up the empty space within the skull. But it seems to be certain that, in many cases in which the head appears to be increased in size, there is no excess of fluid. On the other hand, hydrocephalus is of frequent occurrence as a complication of

rickets, and the affection known as hypertrophy of the cerebral substance is sometimes met with (vol. i, p. 647).

The form of the cranium is also altered. It has been described as elongated; but in reality it is more often square, and flattened on the top, in consequence of the fact that the fontanelles fail to close at the proper time. Clinically, this is the most important of all the symptoms of the disease, except the beading of the ribs. The principal fontanelle not uncommonly remains open up to the age of three years or even longer; moreover, there is often separation of the bones, where they meet to form sutures; their margins, being the growing parts, are generally more or less thickened; so that sometimes one can feel a distinct ridge along the vertex and even down the front of the forehead.

There is often an irregular thinning of the occipital bone—a condition first described by Elsässer, in 1843, as *craniotabes* (cf. p. 859, and vol. i, p. 336). The way to detect it is to grasp the head with the two hands, and to make very gentle but firm pressure with the tips of the forefingers over all parts of the surface of the bone in succession. One may then find that certain small spots, generally near the lambdoidal suture, yield and become indented, just as though the osseous tissue were replaced by a piece of cardboard.

Histology.—The microscopical changes in rickets are very interesting. If with a strong knife one cuts through a rib and its cartilages, across the plane of the union between them—or if one divides the end of a long bone, so as to expose on the face of the incision the junction between its shaft and one of its epiphyses—certain deviations from the normal appearances are at once obvious, even to the naked eye. The "zone of proliferation" of the cartilage ought to be a well-defined, straight, narrow, bluish-white line, perhaps one sixteenth of an inch in width; and the yellow "ossifying zone" beneath it ought to be still narrower. Instead of this, in a rachitic bone the zone of proliferation is considerably thickened, reddened, and of a soft spongy texture. Moreover, the meeting line between them is most irregular and sinuous, with promontories and islands of bone and medullary spaces projecting far into the cartilage. Rindfleisch aptly sums up these changes by saying that the processes which prepare the way for the conversion of cartilage into bone are morbidly accelerated, without the actual ossification keeping pace with them. So, again, beneath the periosteum, instead of an almost inappreciable quantity of embryonic tissue, there is in rickets a soft, red, vascular layer, perhaps one twelfth of an inch thick, which has been compared with the substance of the splenic pulp. It sends processes into the superficial vascular canals, and often has embedded in it numerous minute osseous processes, which tear away with it from the shaft, leaving the latter rough. The whole of the interior of the bone also, including the medullary cavity, is unnaturally red and vascular.

In thin sections, and with the aid of a microscope, the exact nature of the affection can be traced more minutely. The broad, bluish-white zone contains long columns of proliferated cartilage-cells, thirty or forty deep. But, unlike what occurs in the normal process of ossification, these cells can easily be seen to be directly transformed into stellate bone-cells, each of which, however, remains surrounded by a delicate ring, corresponding with the former cartilage capsule. Rindfleisch says that the homogeneous chalky appearance of this "cartilage-bone" enables one to recognise it with the naked eye, even when it is embedded in normal osseous tissue.

The *chemical* composition of the bones in rickets has been several times

investigated, and the proportion of inorganic to organic matter has been found much below what is normal. The analysis of Friedleben, however, published in 1860, made the percentage of earthy salts from 33 to 52, which is considerably higher than that given by earlier inquirers, although much less than the percentage of 63 to 65 obtained from the bones of healthy children.

Course.—Rickets generally runs a somewhat chronic course, but subsides, under favourable circumstances, at the end of a year or two. Some writers, however, have described an acute form of the disease. Senator records the case of a child, four months old, who became feverish, and in whom the epiphyses of several of the long bones of the limbs were swollen and very tender, but without redness; the affection subsided entirely in about six weeks. Fürst is said to have recorded instances of this kind in 1882; but on referring to them, it appears that his were simply cases of rapidly fatal multiple abscesses of joints occurring in infants.*

Event.—When recovery from rickets takes place, the bones lose their soft spongy appearance, and become denser and harder than natural. The articular ends are no longer enlarged, but perhaps this is not due to their absorption, but rather to their being overtaken in their growth by other parts, so that the normal proportions are restored. Many of the deformities which are so conspicuous in young children seem slowly to disappear, at least when they do not exceed certain ill-defined limits. It used, to be traditionally taught at the Hospital for Children in the Waterloo Road that although tibiæ which were laterally curved might become straight in the course of time, a similar change never occurred when they were sharply bent with the convexity forwards near the ankles, in the manner which is attributable to crawling on the floor (p. 852). The pigeon-breast is very generally permanent; and in too many cases the limbs, as well as the trunk, remain distorted for the rest of life. Even when there are no very striking alterations in the shape of the bones, one can often recognise the fact that a person was rickety in childhood by his short stature, his square thick-set frame, and his large protuberant head.† Such persons are often erroneously supposed to have suffered from hydrocephalus.

Most writers express doubts as to whether death is ever caused by rickets alone, apart from complications. Dr Eustace Smith, however, says that he has seen it directly fatal, with extreme dyspnœa and lividity. As a rule, if the child succumbs, it is to diarrhœa, or bronchitis, or laryngismus stridulus; or perhaps to croup or pneumonia, or to one of the exanthems.

Visceral changes.—Following Sir William Jenner, some English observers attach considerable importance to a change in the liver and spleen and lymphatic glands, which he described as an "albuminoid infiltration." Dr Dickinson has investigated the microscopical characters of this affection, which he finds to be an overgrowth of the fibrous tissue in the portal canals of the liver and in the trabeculæ of the spleen respectively, with some excess of cellular elements

* Lately, however, a remarkable form of what may be called "acute rickets associated with purpura" (or scurvy ?) has been described by several authors: Dr Cheadle ('Lancet 1878), Dr Gee (who called it "osteal or periosteal cachexia," 'St Bart.'s Hosp. Rep.,' vol. xvii), Mr Thomas Smith ('Path. Trans.' vol. xxvii), Dr Goodhart ('Dis. of Children,' p. 556) and Dr Barlow, whose excellent account of eleven cases with two autopsies will be found in the 'Med.-Chir. Trans.,' vol. lxvi, p. 159. See also Dr Eustace Smith's remarks on the point in his 'Disease in Children,' p. 255 (cf. *supra*, p. 787).

† This is the type described by Victor Hugo in Quasimodo, the hunchback of Notre Dame, who must certainly have had rickets in childhood.

also. The organs, he says, feel hard, dense, and elastic; the liver shows yellowish acini, each surrounded by a thin pinkish or grey line; the spleen, which may be so large as to extend below the umbilicus, is of a deep red or purple colour, besprinkled with smooth, white spots, or mottled into a pale buff. The lymph-glands are moderately increased in size, tough, white, and opaque. Dr Gee states that in the majority of rickety children who die with an enlarged spleen, its appearance differs in no respect from that of the spleen of ague, or of inherited syphilis, or of cachexia due to unknown causes. He thinks that the affection is really a result not of the rickets, but of the general state of ill-health which caused the rickets; and this seems probable. Dr Dickinson connects it with the occurrence of emaciation and anæmia, others with congenital lues. It is certainly a rare complication, and, when present, usually subsides under treatment.

General symptoms.—The rachitic process in the bones is sometimes ushered in by prodroma, as sickness, diarrhœa, and tumefaction of the abdomen, with languor, drowsiness, loss of appetite, and febrile disturbance. But these supposed early signs of rickets ought to be regarded either as independent effects of the same defective nutrition, or as themselves accessory causes. At any rate, it is a point of great practical importance to remember when called to a case of croup or of bronchopneumonia that one often has to deal at the same time with an advanced state of rickets, although the mother may have thought the child in good health up to the beginning of the attack.

Some minor symptoms often lend considerable aid in diagnosis. One is a peculiar *restlessness* at night, which causes the child, even in cold weather, to kick off the bedclothes, or to throw its naked legs out upon the counterpane, as often as it is covered over by the nurse. Another is a tendency for profuse *perspiration* to break out upon the head and neck and the upper part of the chest, especially during sleep. Elsässer laid much stress on this in connection with his craniotabes. Dr Gee has shown that rickets in itself causes no elevation of *temperature*. Another sign is a *sensitiveness* of the body and limbs, so that the child lies motionless, and dislikes being touched, or moved, or handled; while, if lifted by the armpits, and tossed up and down, it at once begins to cry. The tenderness appears to be partly in the bones and periosteum, but Sir William Jenner and Dr Gee have pointed out that gentle pressure upon the muscles of the loins or abdomen is sometimes no less painful. The muscles are commonly soft and flabby, and more or less wasted. In severe cases the child is almost always unable to walk or even to stand, even though he may have been on his feet for some time before he became rickety, so that Dr Gee speaks of "pseudo-paraplegia" under such circumstances. Jenner relates the case of a girl, six years old, who could neither change her position in bed without assistance nor lift her arm an inch from the surface on which it lay; even at a later period, when she had greatly improved, she was obliged to be tied into a chair with a pillow at its back to support her head; and if the head fell forward the nurse had to raise it for her. She afterwards recovered so as to walk without assistance.

Rachitic children are apt to develop laryngismus stridulus, tetany, the slighter form of convulsion called carpo-pedal contraction, and more severe infantile eclampsia—bronchitis, and bronchopneumonia with collapse of the lungs—and lastly, an obstinate form of intestinal catarrh with severe diarrhœa.

Age.—That rickets is generally a disease of early childhood is admitted by

all observers, but they are not agreed as to the exact limits of the period within which it is most apt to arise. The common belief is that this period is from the sixth month to the eighteenth, or the end of the second year; corresponding, in fact, with the first dentition. And when one inquires closely into cases which are said to have begun later than this, one usually finds grounds for suspecting that a slight form of the affection had existed for some time previously, although it may recently have undergone a more rapid increase. Dr Gee, who collected 635 cases, is disposed to agree with von Rittershain in thinking that in reality its commencement does not often date after the end of the first year. The latest case that he had himself observed was one which seemed to have begun at twenty months. The child, who showed considerable beading of the ribs, had cut the first tooth at six months, and at twelve months it had been weaned and had walked; six weeks before it came under Dr Gee's notice it had begun to get weak in the legs and loins, and during the last three weeks it had sweated much. Even in that instance the absence of the disease at an earlier period was merely a matter of inference; and there can be no doubt that the statistical tables which have been drawn up by different writers are so largely open to the same objection that they have little value.

Some have supposed that rickets may develop itself in young adults, but this idea appears to have been based upon erroneous views as to the nature of certain cases of spinal curvature and of articular disease. Indeed, it seems scarcely possible for the morbid process described above to arise when the process of ossification has in the main been completed; and in future no such case of "late or retarded rickets" would be accepted without full details as to its histology.*

On the other hand, it is certain that rickets may occasionally be seen before the sixth month. Dr Gee speaks of unquestionable beading of the ribs in infants only three or four weeks old. In such cases it seems probable enough that the starting-point of the disease was in intra-uterine life; but whether it can ever be recognised at the time of birth is very doubtful. A few supposed instances of such an occurrence in the stillborn fœtus were recorded by Jules Guérin in his 'Mémoires sur les caractères de Rachitisme' as early as 1839, but Urtel (1873) and Eberth (1878) have each found that the histology of the affection described under that name is altogether different; for the process by which the epiphysial cartilages normally undergo conversion into bone was arrested at a much earlier period, before the cartilage-cells had begun to proliferate and to arrange themselves in vertical columns. The most conspicuous character of such cases is the extremely stunted form of the limbs. Cases of so-called "fœtal rickets" have been published by Dr Thomas Barlow and Mr Shattock ('Path. Trans.,' 1881, pp. 364, 369, and *ibid.*, 1884). The condition is probably allied to cretinism.

Sex.—With regard to the relative liability of boys and girls to the disease, writers have made opposite statements; the only conclusion seems to be that it is equally common in the two sexes.

Climate.—The frequency of rickets varies in different countries, being greater where the climate is damp and cold than in the Mediterranean regions or the tropics. The statistical results hitherto collected are not exactly comparable with one another.

* These conditions seem to have been fulfilled by a case of rickets in a boy of eleven shown by Dr Drewitt ('Path. Trans.,' xxxii, 385), and examined after death by Drs Abercrombie and Barlow.

Dr Gee found rickets in no less than 30·3 per cent. of all children under two years old brought to him at the hospital in Great Ormond Street in 1867. It is believed to be far less common among those who live in the country than among the inhabitants of crowded cities, where children are apt to get too little light or air.

Efficient cause.—Many attempts have been made to find a definite exciting cause for the disease. One observer endeavoured to trace it to too prolonged lactation, another to premature weaning; Guérin is said to have given support to the latter view by some experiments on young puppies, which he deprived of their mother's milk, and fed with meat; the more recent investigations of Tripier, however, have shown that although animals treated in this way become sickly and die, they are not really affected with rickets. Another suggestion has been that the malady is due to a deficiency of lime and of phosphoric acid in the food. Chossat and Alphonse Milne Edwards succeeded in producing curvature of the bones in pigeons and dogs by cutting off the supply of their nutritive salts; but Friedleben has since found that even when atrophy of the osseous tissue is thus induced, the changes characteristic of rickets are wanting. The effect of insufficient and indigestible food in producing rickets is well shown by Dr Norman Moore in his thesis on the subject (1876); but clinical observation points to the conclusion that although defective or improper food is a frequent and important cause of rickets, it acts indirectly, by producing general weakness.

The same may be said of other *conditions in the parents* to which the disease has been attributed, such as syphilis, phthisis, emaciation, anæmia, and even old age. Sir William Jenner thinks it very doubtful whether impairment of a father's health has any influence in producing rickets in his children. Von Rittershain thought that he traced the disease to the presence of some chronic tuberculous disease in the father more often than in the mother. But the truth is that among the poor it is impossible to isolate causes of this kind; a husband's illness may deprive the wife of nourishment, throw much heavy work upon her, and in many different ways render her likely to bear weakly infants. So, again, even when the parents of a rickety child have, one or both of them, had rickets in early life, it is very doubtful whether the disease is really transmitted. A point of great importance, on which Jenner has laid stress, is that the first child of a family, or even the first two or three, may be found free from rickets, where later ones are affected by it; and again, that if once a woman has borne a rickety infant, those that follow are almost sure to have the same disease. This is due not only to the progressive enfeeblement of the mother's health by repeated child-bearing, but also, among the poor, to the overcrowding and deficiency of food and clothing which are implied by a large family; and perhaps among the middle classes to the way in which children are sometimes kept indoors, when there is but one nursemaid for several of them.

The relation of rickets to *tuberculosis* requires further investigation by pathologists untrammelled with previously formed opinions. Jenner, although he contrasts the two "diatheses," adds that rickets does not by any means exclude tubercle. Dr Eustace Smith says that rickets never occurs in children in whom "the tubercular disposition" is well marked; but this statement is a matter of course, because the features supposed to indicate that disposition are of themselves a disproof of rickets. The statements of von Rittershain have already been quoted as to the inheritance of rickets from tuberculous fathers, but Jenner refers to a

table made for him by Dr Edwards, which appeared to show that phthisical parents are actually less likely than others to have rickety children.

The relation of rickets, and particularly of craniotabes, to *congenital syphilis* was discussed at the International Medical Congress of 1881 ('Trans.,' vol. iv, p. 35) by MM. Parrot, Guérin, and Bouchut, of Paris, by Dr Rehn, of Frankfort, and by other pathologists. See also on this point an admirable paper with tables, by Drs Lees and Barlow, in the 'Path. Trans.,' vol. xxxii, p. 323.

Pathology.—It was long ago suggested that the immediate cause of the changes in the bones in rickets was the action of lactic acid, dissolving out the lime salts from their substance. The acid was said to have been detected not only in the bones themselves, but in the urine; it was supposed to be formed in excessive quantity in the alimentary canal from milk and other articles of food. Some chemists also stated that more than the normal amount of phosphate of lime was excreted by the kidneys. The modern investigation as to the histology of the disease would obviously have rendered such a theory untenable, even if it had otherwise had a chance of acceptance. Recently, however, another view has been promulgated, in which the acid plays a different part. In 1871 Dr Wegner, of Berlin, in the course of some experiments upon young animals with minute doses of phosphorus, found that if, while administering the poison, he withheld lime salts from the food, there arose an affection of the bones precisely like rickets, as it is seen in the human subject. He supposed that the phosphorus was a stimulant to the osseous tissue. Now, Heitzmann has since stated that lactic acid is capable of acting in the same way. The hypothesis, therefore, as given by Senator, is that the disease is the combined result of the irritant influence of that acid upon the growing bones, and of a deficiency of phosphate of lime, consequent either on there being too little of it in the food, or on its being carried away through the bowels by diarrhœa. But at present this theory seems to rest upon too slender a foundation of facts, almost as slender as that which supports the theory of lactic acid as the cause of rheumatic fever. The two theories seem to be mutually destructive.

Diagnosis.—The recognition of rickets is very easy when it is fully developed. As a source of fallacy in regard to the craniotabes of Elsässer, may be mentioned here the case of cerebellar tumour (referred to in vol. i, p. 620), in which a somewhat similar thinning of the occipital bone was observed. The only real difficulty is as regards early cases in young children. The author must confess that he has sometimes been in doubt as to what constitutes "beading" of the ribs, as distinguished from the slight roundness of their ends which is normal.

The mistake usually made is not to look for the signs of rickets. This should never be omitted in cases of infantile diarrhœa, of laryngismus stridulus, and of bronchitis in young children.

The *prognosis* of the affection, if left unmodified by treatment, is said to be more grave in proportion as the child is younger at the time of its commencement. It is therefore extremely important to be on the look-out for it whenever an infant at the time of the first dentition begins to fail in health, or suffers from any trifling disorder, such as relaxation of the bowels. For, as follows from what has been stated in regard to its ætiology, rickets is eminently a disease that can be prevented. It is also one that can be cured.

Treatment.—The chief thing of all is to attend to the food. When an infant is suckled, the breast should during the first six weeks be given every two

hours, except from 11 p.m. to 5 a.m., during which interval the mother or the wet nurse should be allowed to sleep; at a later period every three hours, or still less frequently. It is very wrong to let a child lie asleep with the nipple in its mouth, although nothing is more common than for it to be kept at the breast all the night while the mother herself is in a sound slumber. If a baby does not thrive, one cause for it may be that its mother's milk is insufficient in quantity or too poor. According to Dr Eustace Smith, it is a sign that this is the case when the infant falls asleep while sucking; or one may notice that it sucks at its thumbs until they become raw. If something in addition to the breast-milk is required, one may employ cows' milk or asses' milk, sweetened a little, and perhaps diluted with water, according to the age. Neither biscuit powder nor any other farinaceous food should be administered to very young infants. The secretion of saliva appears not to be established, under normal conditions, before the third month; and it is believed that up to that time all starchy matters pass through the intestine unaltered, and are discharged with the fæces. But it must be confessed that some well-grown children are seen whose parents had been ignorant of this rule, and had brought them up in direct opposition to it. Liebig's or Mellin's malted food may safely be used mixed with milk, even a week or two after birth. Some infants, however, will not take it; and there is no doubt that what suits one perfectly may not do for another. Swiss milk and the other condensed preparations of milk are often given to young children. They undoubtedly fatten rapidly; but the more important tissues seem not to be well sustained, and infants so brought up are apt to succumb if attacked by diarrhœa or by other ailments. Probably these effects are attributable to the quantity of sugar contained in it; but one also feels a natural prejudice against the attempt to preserve artificially a substance so liable to decomposition as milk; and the same doubt would apply to Nestlé's compound.

After six months it is advisable that the mother's milk should be supplemented either by farinaceous food or by Liebig's maltine. At eight months, a little mutton- or chicken-broth, or beef-tea, may be given with advantage. At ten or twelve months the child should be weaned.

When a feeding-bottle is used, the most extreme care is required to keep it and its tube clean, so that it may not turn the milk which is put into it sour. It should be scalded out every time it is employed; and the tube should be always kept in water.

No one who had not witnessed it would believe how utterly wrong is in most cases the management of young children belonging to the lower classes. At a very early age they are allowed to have bacon, fried fish, pickles, potatoes, and beer. If brought up by hand they perhaps are given cornflour, or some other substance which is little better than pure starch; and it is often made up with water instead of with milk. If suckled, they are not weaned until they are eighteen months or two years old; but long before this they sit with their parents at meals, and share what they eat.

Even at a later age, a child affected with rickets requires certain precautions to be taken, which are not equally necessary for those who are healthy. Not only must its food be nutritious and digestible; it must also be easy of mastication, if the teeth are few in number or decayed. Thus one often has to direct that all the meat should be finely powdered in a mortar, and that the potatoes should be mashed, and all the lumps be carefully picked out.

When indoors a rachitic child should be kept lying down, and not be

encouraged to attempt to walk, so long as the bones are soft. Splints projecting below the feet may be used for the purpose of rendering such attempts impossible. But mechanical appliances seem to be of little service in straightening the spine or the limbs. Jenner, however, has found, where the ribs were inclined to yield, that a well-adjusted bandage round the abdomen was useful by restraining the descent of the diaphragm.

At night the child should sleep on a hair mattress, and if its head is tender and inclined to perspire, it should have a horsehair pillow made with a hole in the centre so as to remove all pressure from the occiput. Dr West has seen this give quiet sleep for the first time for weeks. A thin linen nightcap may also be worn, which can be changed two or three times if necessary.

Bathing is of considerable importance. The child should be sponged with warm soap and water once or twice a day, or, according to the season, with tepid or even cold water, in which sea-salt may be dissolved. Dr West recommends tan baths, made by adding to the water a decoction of oak bark; the receipt is to take three handfuls of the bruised bark, and boil it in a linen bag in three quarts of water for half an hour.

Drugs.—Among medicines the most valuable is cod-liver oil. It should be given even when the bowels are relaxed, unless it causes an increase of diarrhœa, which is not often the case. Steel wine and quinine are also useful, and the *liquor ferri perchloridi* is often prescribed with advantage. Dr Eustace Smith has seen marked benefit result from Alison's prescription of tannic acid in doses of half a grain to a grain twice or thrice daily. If an occasional aperient is necessary, a little castor-oil or a powder of rhubarb and soda, or the syrup of senna, may be given. Lime-water should not be used as a matter of routine, nor unless there is proof that the milk which the child takes becomes curdled in its stomach.

Sir William Jenner forbids the administration of repeated doses of mercury to rickety children; and he says that when they are attacked with acute diseases leeches should never be applied, nor antimony be given.*

Mollities Ossium.†—In the middle of the last century instances in which extreme deformities had been produced by softening of the bones were recorded, almost in the same year, by three observers, Duverney, Morand, and Pringle; and the names of two of the patients—the Marquise d'Armagnac and Madame Supiot—have become historical. Similar cases have since been met with from time to time, but very rarely, except in certain districts bordering upon the Rhine, where, according to Senator's statement (in 'Ziemssen's Handbuch') they have been somewhat less infrequent.

Sex and age.—Mollities ossium is far more common in women than in men; among 145 cases quoted by Mr Durham, in the 'Guy's Hosp. Reports' for 1864, thirteen only occurred in males; and one may suspect that the disproportion would have been still more marked if they could have been sifted very critically.

The *age* at which it is least uncommon is between twenty-five and thirty-five; a few of the patients are said to have been under twenty years old, and a few over fifty. In a well-marked case which the editor saw with

* Beside the discussion on rickets in the International Congress of 1881, referred to above, an instructive debate will be found in the 'Pathological Transactions' for the same year (vol. xxxii, pp. 312—404). It was introduced by the author of this work and continued by Dr Norman Moore, Mr Haward, Dr Dickinson, Mr Parker, Sir William Jenner, Mr Hutchinson, Mr Lucas, the late Dr Baxter, Mr S. Watson, and Dr Goodhart.

† *Synonyms.*—Malacosteon—Osteomalacia—Le rachitis des adultes.

Dr E. O. Day, the disease began about sixteen, and Dr Rehn has recorded one in an infant ('Internat. Med. Congr., 1881,' vol. iv, p. 59); so also has Dr Bury, of Manchester ('Brit. Med. Journ.,' 1884, vol. i, p. 213).*

Ætiology.—A definite cause can seldom be assigned. Habitual exposure to cold and wet, as from living in a damp house, has sometimes been supposed to give rise to it, but to many instances such an explanation could not be applied. The patients have often been well fed and in easy circumstances. Sometimes it has been noted that they had been affected with rickets in childhood, but in all probability this was a mere coincidence. The occurrence of utero-gestation appears to play an important part in the ætiology of mollities ossium; in ninety-one of Litzmann's and Durham's 132 cases it began during pregnancy, or shortly after childbirth.

A case of extreme distortion of the limbs, thorax, and pelvis, recorded by Dr W. J. Webb, of Chicago, in the 'New York Medical Journal' for March 21st, 1885, occurred in a man some of whose brothers and sisters had symptoms more or less like his own.

Symptoms and course.—As a rule, the earliest symptom of the disease consists of pains in the trunk or in the limbs, which seem to vary in character in different cases, and which may appear to wander or fly about from part to part, so that they are usually supposed to be "rheumatic." The next thing may be that one of the bones breaks without cause, or during some slight effort, as in getting out of bed. Or a progressive change in the figure may be noticed, the body becoming short or stunted, the back rounded and distorted laterally, the neck stooping so that the chin may be brought close to the sternum. There is extreme lassitude with disinclination for any kind of muscular exertion. The patient waddles in walking, and has to help herself with sticks or crutches. Presently she is obliged to take to her bed. She now becomes a most pitiable object. Her bones may show numerous fractures, and these remain unrepaired, the broken ends being merely surrounded by a soft callus, and forming so many false joints. Her limbs also become bent in the strangest way, perhaps one leg outwards and the other inwards, according to the pressure to which they have been subjected while she is lying or half-sitting, propped up with pillows, and "all in a heap." Towards the last the softening of the bones may be so extreme, that one can bend them backwards and forwards with but little force, and without injuring them; bringing, for instance, the foot round so as to touch the back or the head. The more superficial bones, even the cheek-bones, can often be indented by the finger, or they may feel like egg-shells, as if they had merely a thin layer of osseous material on their exterior.

For a time the general functions of the body may appear to be but little interfered with; the appetite and the digestion may be good; and menstruation may go on naturally. Pyrexia may be occasionally present, but not to any marked extent.

Event.—Ultimately, however, the disease ends fatally, either by exhaustion or more commonly from inability to breathe, the ribs being dragged inwards at each inspiratory effort, so that scarcely any air enters the lungs. Another frequent cause of death is the obstruction to parturition caused by distortion of the pelvis. Usually the brim acquires what is termed a *rostrate* character, the pelvic symphysis forming a sharp angle between two

* See moreover the report on Dr Goodhart's case ('Path. Trans.,' vol. xxxiv, p. 201), and Mr Davies-Colley's (*ibid.*, vol. xxxv, pp. 285, 292); also Mr Thos. Jones's 'Diseases of the Bones' (1888).

prominences due to the pushing upwards of the acetabula and the parts adjacent. Cæsarean section has often been necessary, and has often ended fatally for the mother. It is a point of great importance that mollities ossium seems to advance step by step during successive pregnancies, the patient in the intervals regaining strength and being sometimes able to get about to work. The duration of the disease is usually from four to six years; but sometimes it has lasted eight, ten, or thirteen. Once, in a case recorded by C. Schmidt, it is said to have ended fatally in three months. In very exceptional instances it has ended in recovery.

Diagnosis.—Mollities ossium is very easy to recognise at an advanced stage of the disease, but it must be borne in mind that mere brittleness of the bones is not sufficient to determine it. In old people, and in persons who have been long bedridden, the ribs and some other parts of the skeleton are apt to undergo atrophy, so that one can very readily snap them with the fingers (*atrophia ossium senilis*). In the inmates of lunatic asylums this change seems to be particularly frequent. But even in young persons a somewhat similar state of *fragilitas ossium* is sometimes met with. The author once saw a young man who was dying of bronchitis, and in whom a large number of the ribs were found to be broken, as the result of muscular efforts in coughing. He had at different periods of his life had fractures of many of his bones from very slight injuries. Another affection which might be mistaken for mollities ossium is sarcoma, developing itself in a large number of the bones at once, and causing their spontaneous fracture.

Anatomy.—The fundamental distinction between all other morbid states and malacosteon is that in it the bones are not only fragile but *soft*. After death they are found to be readily cut with a knife, and they feel like india rubber or even like cheese. On section, the compact osseous tissue may have entirely disappeared, or it may be reduced to a very thin lamina beneath the periosteum, within which there may be nothing but a soft pulpy material. Or the cancellous tissue, instead of undergoing uniform absorption, may be hollowed out here and there into rounded or oval cavities. In the case which occurred at Guy's Hospital in 1864 this change in the bodies of the vertebræ was so striking that at first it almost seemed as though masses of a soft myeloid growth had eaten away the bone. The calvaria was considerably increased in thickness, and had a homogeneous texture, which was compared by Mr Durham to softened pasteboard.

On chemical analysis the composition of the bones is found to be greatly altered. Different observers give different figures, but it may be roughly stated that the proportion of inorganic constituents is reduced to about 30 per cent. The carbonate of lime is said to be diminished in quantity even more than the phosphate, and the character of the latter salt to be changed, the amount of lime being deficient in relation to the acids, so that it no longer forms the ordinary basic compound. C. Schmidt has also stated that gelatin is often absent. It must be remembered that the percentage given above represents not the definite actual constitution of osseous tissue affected with mollities ossium, but an average derived from some parts in which the change is extreme, and from others which perhaps deviate but little from the normal.

As a rule, the morbid process seems to be most advanced in the interior of the bone, and least so towards the circumference. The microscope shows that there may be wide differences even within a single Haversian system, the lamellæ nearest the central vessel being completely decalcified, while

the outer ones still retain their inorganic constituents. The orifices of the Haversian canals on the surface of the bone are widened, and a viscid fluid may exude from them when the periosteum is stripped off, which itself is very thickened and unduly vascular.

The substance which fills the interior of the bones in mollities ossium seems not to differ essentially from the normal medulla. It varies in appearance, being sometimes of a deep red colour, and spotted with ecchymoses, sometimes opaque, yellow, and fatty, sometimes mucoid and semi-translucent. These variations probably correspond with those which naturally occur in the same tissue. In the red material there are numerous cells, some of which may contain two or sometimes several nuclei. At least this is what Virchow and other histologists have generally stated. Rindfleisch maintains, on the contrary, that exceptionally few young elements can be seen.

The pathology of mollities ossium is as yet unknown. The decalcification of the bones has been supposed to be due to the action of some acid, and lactic acid is said to have been actually detected in the osseous tissue by several of those who have made analysis of it, and also in urine passed during life. In one case which ended in recovery Moers and Mück found that the acid gradually disappeared from the urine as the disease was subsiding ('Deutsches Archiv,' 1869).

Rindfleisch, on the other hand, suggests that the solvent is carbonic acid. Recent observers have failed to find in the urine an excess of phosphate and of carbonate of lime, which was at one time said to be present, and, according to Mr Solly, in one case reached four times the normal quantity, such phosphatic deposits occurring in urine having an acid reaction (?). Phosphatic calculi seem, however, to have been found in the kidneys or in the bladder in certain cases, and abundant deposits of phosphate of lime were constant in a case of a woman who was under the care of Dr Oldham in Guy's Hospital about 1866.

It is a point of some interest that osteo-arthritis deformans has sometimes been noticed in connection with mollities ossium. Moreover, in the muscles changes have been found which cannot easily be referred to mere disuse: not only do they become flabby, wasted, and fatty, but Friedreich has recently shown that the nuclei of their fibres multiply, and that there are other histological appearances identical with those which occur in progressive muscular atrophy. Trousseau and Lasègue observed in some cases that gently touching or stroking the surface of the limbs was capable of exciting painful contractions of the muscles beneath. It may therefore be that mollities ossium is, after all, something more than a mere disease of the bones.

Treatment.—No certain means of checking the progress of this remarkable disease has been ascertained. It is most important for women who show signs of the disease to avoid pregnancy, since we have seen that this condition aggravates the evil each time that it recurs. Phosphate and carbonate of lime have been given with a view of supplying the deficiency of earthy salts in the bones, but probably without benefit.

A more efficient treatment is the administration of cod-liver oil. Trousseau (who regarded the disease as essentially the same as rickets) narrates two striking cases (one published by Beylard in his thesis (1852)), in which complete recovery of health followed the administration of this remedy.

DISEASES OF THE SKIN

INTRODUCTORY CHAPTER

General pathology — Classification — Nomenclature — Historical sketch — The elementary anatomical lesions—The local distribution of cutaneous affections: in depth (bathymetric) and over the surface (regional)—The circumstances: age, occupation, and habits of the patient, season, history of the malady, subjective symptoms, disturbance of other organs, effect of previous treatment.

It is now well known that diseases of the skin differ in their origin and significance no less than diseases of the tongue, the eye, or the bladder. Some of them are symptomatic of specific febrile diseases, of which they form a part, and are therefore important chiefly for the sake of diagnosis. Others are produced by certain articles of food, by poisons, or by drugs. Some are examples of various pathological processes which are familiar in other organs, as cancer, hypertrophy, atrophy, hæmorrhage, pigmentation. But the majority are examples of the widespread morbid process called inflammation.

Dermatitis is sometimes the result of definite local irritation, but is often independent of such obvious cause, and therefore must at present be called primary or idiopathic. In this and in other respects it is analogous to the inflammations which affect the bronchial mucous membrane, the stomach, the intestines, and the urinary tract. Closely resembling these structures in its origin and development, its general anatomy, its vascular and nervous relations and its glandular apparatus, the skin has also a pathological alliance with the mucous membranes.

Diseases of the skin are best studied as examples of general morbid processes, modified by the peculiarities of the affected tissue.

Classification.—Any arrangement of diseases is valuable so far as it helps the memory to retain useful facts; any arrangement is useless or mischievous if it pretend to be a universal or "natural" or "scientific" system. Diseases are not natural objects; they are physiological states, which we sometimes define by their cause, as plumbism and scabies, sometimes by their histology, as sclerosis of the cord and epithelial cancer of the lip, sometimes by their constancy in transmission, as measles and typhus, and sometimes by more or less constant concurrence of symptoms, as chorea and epilepsy.*

Nomenclature.—We must abandon the binominal terminology which once extended to the whole of medicine, as founded upon a misleading analogy between natural organisms, like plants and animals, and heterogeneous objects like diseases. Next to a false pathology and fruitless attempts at classifica-

* This subject was dealt with by the present writer in the twenty-second volume of the third series of 'Guy's Hospital Reports' (1877), where examples of classification of diseases of the skin are given on the several bases of pathology, ætiology, anatomical distribution, prevalence at certain ages, and reactions to remedies (pp. 174—177).

tion nothing has been more injurious to the rational study of this group of diseases than a cumbrous, pedantic, and often barbarous nomenclature.

A good name should have the following characters :—(1) It must consist of a single word. (2) It must be distinctive, and easily recognised both by the eye and the ear. (3) It must be capable of forming an adjective. (4) Less essential points are that it should be short, if possible familiar, of Latin or Greek origin, or capable of easy reproduction in the former tongue, and as classical and euphonious as may be. It should be unmeaning, or at least arbitrary and conventional in its application ; or if descriptive, should apply only to some obvious and constant feature of the malady.

Historical sketch.—The fact that many eruptions of the skin are closely attendant upon febrile and other general disorders early attracted notice ; and the humoral pathology which pervaded medicine from classical times until almost the present day afforded a ready explanation of their occurrence. Hence cutaneous diseases were long regarded as mere symptoms of some hypothetical "dyscrasia" of the four Galenical humours : the blood which took its origin in the liver, the phlegm secreted by the pituitary gland, the bile by the gall, and the black bile by the spleen. From due mixture of these humours arose the four natural temperaments : sanguine, in which the blood was predominant ; phlegmatic or pituitous ; bilious or choleric ; atrabilious or melancholic ; and from their ill-mixture resulted dyscrasiæ, such as scurvy, scrofula, and gout.

When the doctrine of the four humours was given up, they were thought to be due to disorders of the blood ; then, when better knowledge of the chemistry and morphology of the blood began to stand in the way of so easy an explanation, they were ascribed to "diatheses," or tendencies, of which the eruption was at once the evidence and the effect. Explanation by assumed causes still took the place of inquiry into anatomical and clinical facts.

During the eighteenth century the causes and indications for treatment of an eruption were readily determined by learned physicians to be due to a strumous cachexia, or a scorbutic state of the blood, or vitiated humours from obstruction of the primæ viæ, or lues venerea, but the actual physical conditions of the skin were scarcely noticed. It is the great merit of Willan (and in some degree of his predecessor Plenck, of Vienna) that he accurately described the *anatomy* of the morbid skin. His "orders," the *elementary lesions* of later dermatologists, are the alphabet of the subject, and correspond with the "physical signs" of the diseases of the lungs introduced by Auenbrugger and Laennec.

From the English or anatomical school of Willan and Bateman sprang the French school of dermatology, which may be generally described as *ætiological* in aim. Biett, its founder, was a pupil of Willan, and introduced his system into France. He had the great merit of perceiving that syphilis does not merely act along with other predisposing causes in producing diseases of the skin, but that it has as its direct consequence definite, constant, and recognisable lesions, the knowledge of which is all-important for diagnosis and for cure. He was succeeded by Cazenave (1843), Devergie (1854), and other systematic writers, who continued the work of clinical investigation and accurate description. Unfortunately Biett's success in tracing certain cutaneous affections to syphilis led to the formation of similar groups of "scrofulides" and "maladies dartreuses."

The attempt to define cutaneous diseases by their true *nature and cause*

instead of by anatomical lesions had been already made by Alibert, a contemporary of Biett. His eloquence and power of picturesque description had much influence, which was increased by the publication of a magnificent atlas of plates, illustrating his 'Arbre des Dermatoses.' His pathology, however, was erroneous, his description superficial, his nomenclature inaccurate, capricious, and unclassical. The same attempt to explain rather than to investigate, and to supply the nature and causes of disease by hypothesis when proof is absent, pervades the voluminous writings of Bazin (1853–70), who carried the hypothesis of "diathesis" to its extreme limit. The same principles were illustrated in the interesting and clinically useful lectures of Professor Hardy (1858–64), so long connected with the great hospital of St Louis.*

Meanwhile, another school had arisen at Vienna which was guided by the pathological doctrines of Rokitansky. Its founder was Ferdinand Hebra, whose writings have done more than those of any other man to put the study of dermatology on a sound basis and to extend its limits.†

The *pathological* school of Vienna represented by Hebra, the *diathetic* school of Paris represented by Hardy, and the *anatomical* and *therapeutical* school of England represented by Erasmus Wilson (1847–67) have all changed during the last twenty years. *Histological* investigation by the improved methods of the last twenty years have thrown much light upon the morbid processes of the skin, and the newest school of dermatology, that of America, is making important contributions to the subject.

Before entering upon the description of the several diseases to which the skin is subject, it will be well to say a word on the most important points in their description and diagnosis.

I. The Anatomical Lesions.—The following are the most characteristic and important:

1. *Hyperæmia* or *Congestion.*—(*a*) Mere overfulness of the vessels from paralysis of the vaso-motor nerves with redness and heat, but without the exudation and tissue changes which accompany inflammation. This hyperæmic blush, readily produced in the physiological laboratory, is rarely seen as an uncomplicated morbid condition (*e. g.* Trousseau's *tache cérébrale*, see vol. i, p. 683).

(*b*) *Active, or arterial inflammatory hyperæmia*, varying in colour from brilliant scarlet to rose pink, and combined with heat, tingling, or other sensations. Such an early stage of inflammation is often called "erythema." The local swelling and the subjective symptoms distinguish it from the non-inflammatory hyperæmia just described.

(*c*) *Passive* venous or congestive *hyperæmia*, dependent upon retarded circulation and distended venules. The colour is purple, bluish, or livid, the surface is cold, and there are no painful sensations. This passive congestion, frequently seen as the result of thrombosis, and also in long-standing

* Since these lines were written, M. Hardy has returned to his dermatological studies and has published a systematic treatise on the subject.

† His 'Acute Exantheme und Hautkrankheiten' was the third volume of the series of textbooks of 'Pathology and Therapeutics' superintended by Virchow, and was published in parts between 1860 and 1874. Much of the latter part is written by Hebra's son-in-law Moritz Kohn, now better known as Kaposi. The translation into English for the New Sydenham Society (1866–75), begun by Dr Fagge and the writer, was completed by Mr Waren Tay.

affections of the heart and lungs, is often associated with the more chronic forms of inflammation in which œdema is present, and connective-tissue overgrowth is apt to result. The best example of this condition is in the congestive erythema of a chilblain.

2. *Pimple or Papule*.—A solid, small elevation of the skin. Under this name more than one pathological lesion is included.

(*a*) The inflammatory papule, more or less pointed, bright red, small, and very early seen with a lens to contain a minute drop of exudation. It is either abortive or ends in a vesicle or pustule.

(*b*) Chronic large inflammatory papules, never showing liquid exudation, but apt to become covered with minute scales. Sometimes, as in psoriasis, these papules increase so as to form a raised patch, and then become covered with scales, and sometimes they coalesce with the same result as in lichen planus. More often the papules remain discrete and without scales, as in prurigo.

(*c*) A solid non-inflammatory papule formed by true hypertrophy of the normal papillæ of the cutis. Such papillary growths produce the minute multiple warts which occasionally occur in immense numbers over wide surfaces of the body; large local warts and condylomata are much more common.

(*d*) Solid elevations of the skin, which may be called false papules; such as the heaped-up scales at the orifice of a hair-sac which form the so-called "lichen or pityriasis pilaris," and a sebaceous gland occluded by its own secretion, which is called a "comedo."

3. *Vesicle*.—A visible cavity in the skin filled with transparent liquid. In almost all cases the vesicle is inflammatory, and the liquid is exuded plasma, consisting of water, salts, albumen, and a few leucocytes with only a trace of fibrinogen. Where the epidermis is thin the vesicles rupture almost as soon as they form, but where it is thick, as in the palm and sole, they grow and coalesce into large bladders. Broad and flat vesicles, as those of zona, are usually distinguished from the smaller and more closely-packed vesicles of eczema. The vesicles of smallpox are remarkable not only for their size and depth, but for the exudation being so effused into the meshes of the papillæ and Malpighian layer that the cavity is "pocketed," and shows a central depression or *umbilicus*.

Non-inflammatory vesicles consist of retained excretion either of sweat-glands (sudamina) or of mucous glands. The latter are practically the only vesicles seen on the mucous membranes, for under the moisture and friction of the mouth, though inflammatory vesicles form, they are scarcely ever seen before they burst.

4. *Pustule*.—A cutaneous abscess, that is a cavity in the skin containing inflammatory exudation, water, salts, albumen, and abundance of dead leucocytes in a state of fatty degeneration, with usually only traces of fibrinogen. The distinction between a vesicle and a pustule is therefore often one of time only, and rests upon the abundance of the corpuscular element in the exudation; but while most vesicles become pustules the exudation remains serous in many cases of eczema. Again, in contagious impetigo, in furunculi, and in some other cases, the first visible exudation is opaque, yellow, and purulent.

5. *Bulla* or *Bleb* is the name given to a very large vesicle. It is, as a rule, inflammatory and of essentially the same pathology as a vesicle or pustule. It contains at first transparent serum, but this usually becomes more or less completely purulent. There are also almost always shreds of fibrin to be

seen. The anatomical distinction asserted between ordinary inflammatory bullæ and those of pemphigus will be referred to under that heading.

6. *Scab* or *Crust.*—A dried-up concretion of the contents of a vesicle, pustule, or bleb. Its form depends upon the inflammatory process ceasing, otherwise fresh exudation succeeds, and no dried-up mass is allowed to form. The size of a scab will always depend upon that of a pustule or bleb which formed it, its thickness upon the amount of fibrin and leucocytes in the exudation. Its colour is often characteristic ; light brown or yellow when the exudation is serous, deeper yellow (compared by the elder anatomists to honey, whence the term *Porrigo favosa*), greenish yellow in some cases when the pus is thick, red or almost black when the exudation contains red blood-discs.

7. *Scale* (*squama*).—A dry flake of epidermic cells. When scales form in moderate amount and of small size as the result of inflammation which has passed, they are described as *furfuraceous ;* when large, adherent, imbricated, and glistening silver-white from the refractive power of air enclosed in the spaces, scales have the characteristic appearance seen in psoriasis. Large, thin, and very abundant scales, which have been compared with dry hop leaves, and are sometimes termed *squames*, are almost characteristic of pityriasis-rubra. Beside the true epidermic scales desquamation often consists of dried-up sebum or of dried exudation mixed with epidermis. The microscope distinguishes the amorphous fatty material of the former and the leucocytes of the latter from the flat horny cells of true scales.

8. *Wheal* (*pomphos*).—A flat solid elevation of the skin much larger than a papule, and of ephemeral duration. Such wheals may be either traumatic or idiopathic ; they are the characteristic effects of the poison of the stinging-nettle and of the form of erythema, hence called urticaria. They are formed by acute œdema of the skin producing local anæmia from pressure.

9. *Scratch-mark.*—An injury to the skin of linear form and curved outline, usually marked by dried-up blood, and having a definite relation to the range of the patient's hands. They are of diagnostic value as proofs of pruritus.

10. *Raw.*—A surface which has lost its horny layer of epidermis so that the moist and living Malpighian layer is exposed, from which more or less exudation oozes. Such a raw weeping surface is characteristically seen after the blister formed by cantharides has been broken. It also results from the rapid rupture of a number of vesicles as in the kind of dermatitis called eczema madidans.

11. *Chap* (*rima*).—A crack or fissure which goes through the epidermis to its Malpighian layer or to the vascular papillæ beneath. These rimæ or rhagades sometimes extend very deeply, are apt to bleed, and are always extremely painful.

12. *Sore* (*ulcus*).—The result of destruction by inflammation which has reached below the Malpighian layer and has destroyed the papillæ ; characterised by the absence of any trace of epidermis, by the granulations which cover its floor, and the pus in which they are bathed.

13. *Scar* (*cicatrix*).—The result of the healing process after an injury or disease which has been deep enough to destroy the papillæ of the part. Accordingly the presence of a cicatrix, however superficial and slight, shows that the preceding process affected the deep layer of the cutis.

14. *Nodule.*—A solid elevation of the skin larger than a papule, and seated in its deep layer. The nodule was formerly called a tubercle, but the

word "tubercle" should never be applied except with its present pathological meaning. A *node* is a large nodule, and there is no reason for restricting the term to syphilitic nodes or *gummata*.

15. *Stain* (*macula*).—A patch of increased pigmentation of the skin, either the result of long-continued preceding hyperæmia or occurring independently as a primary increase of pigment.

16. *Hæmorrhage* (*ecchymosis*).—When a blood-vessel of the cutis vera gives way, a dark red or purple mark is produced, which (like the macula) does not disappear on pressure. When small, the punctiform spots resemble flea-bites, and are hence called *petechiæ*; larger extravasations, particularly when elongated in form, resemble the bruises caused by a stick, and are termed *vibices*.

II. Distribution.—After determining the morbid anatomy of a disease of the skin the next step is to notice its distribution.

(A) *In depth.*—In its pathology the skin does not follow the anatomical and embryological division into epidermis and cutis vera. It may be physiologically divided into three layers:

(1) The *horny layer of epidermis* or *cuticle*, dead scales, the only affections of which are increased growth, atrophy, dryness, desquamation, and other results which really depend upon perverted growth in the living layer of cells which lies immediately beneath it.

(2) The *living Malpighian layer* of the epidermis, together with the *papillary layer* of the cutis. These two tissues are constantly and inseparably united in their pathology. Their inflammation constitutes the enormous group of diseases which come under the head of superficial dermatitis. Affections confined to this part never leave scars.

(3) The *deep layer of cutis* with the *subcutaneous connective tissue*. Inflammation or new growths beginning below the papillæ are prone to spread to the subcutaneous tissue and not to stop until they reach subjacent muscle or bone or deep fascia. The deep affections of the skin which lie in this region are less numerous, but more severe, than those of the superficial layer, and are always marked by cicatrices.

(4) Lastly come the cutaneous affections which particularly affect the *sweat-glands*, the *sebaceous glands*, the *hairs* and hair-sacs, or the *nails*.

(B) In their distribution over the *surface* of the body the diseases of the skin differ greatly. The earliest attempts at classification were between affections of the scalp and of the trunk. In Willan and Bateman's system this character did not receive due consideration; but it has met with still less at the hands of French and German dermatologists. Even in the best descriptions of Hebra and his successors it is sometimes impossible to learn what part of the body is affected by a particular disorder. But the fact is that very few diseases of the skin are indiscriminate in their extent, while many are at least as definitely and exclusively fixed to certain localities as the lesions of enteric fever in the intestine, of tubercle in the lung, or of tabes in the spinal cord. The skin is not uniform in its structure, the relative thickness of its layers, its vascularity, its nervous supply or the distribution of its glands. Its differents parts are variously protected both by natural and artificial coverings, they are variously exposed to injuries, to irritants, and to moisture. It is therefore not surprising that their diseases differ so greatly. Psoriasis of a flexor surface and scabies of the face should be regarded, like carcinoma of connective tissue or phthisis of the base of the

lung, as altogether exceptional. It must be remembered that in childhood the several regions of the skin are not yet differentiated, and hence the local distribution of its diseases is less strictly adhered to than in adults. We find precisely the same rule in the distribution of pneumonia, of malignant disease, and of tubercle, in children.

The several regions will be fully described in the following chapter on eczema and dermatitis generally.

III. Circumstances.—The third group of characters includes the natural history and surroundings of a case of cutaneous disease. They help in diagnosis, they throw light upon pathology and causation, and they frequently supply hints for treatment. We have to consider:

1. The *age* and *sex* of the patient. Some affections, like prurigo pedicularis, are scarcely seen except in the aged skin; others, like impetigo, are extremely common before puberty and extremely rare afterwards; others, like true acne, are with rare exceptions confined to the period of adolescence.

2. *General health*, and particularly the state of the stomach and bowels, the urine, and the temperature.

3. *Occupation*, particularly when parts of the skin are exposed to cold, to great heat, to wet, or to chemical or mechanical irritation. Also intercourse with other persons affected with a similar disease.

4. *Clothing.*—Some cutaneous maladies are caused by the irritation of under-garments being saturated with sweat, or of deleterious dyes or friction.

5. *Cleanliness.*—Pediculi and tinea versicolor are the result of dirt, and the same is true of some forms of eczema, intertrigo, and acne. But as a rule it is remarkable how little local mischief is done by uncleanly habits and neglect of the skin.

6. *Climate*, season, temperature, moisture or dryness of the air, sun, frost and wind. Many diseases of the skin, as of other organs, which were formerly supposed to be endemic, are not really so; but some are peculiar to hot countries, and others, notably leprosy, have, within historical times, become notably restricted in their range.

7. *History* of the malady, its duration, the manner of its onset, and particularly, when obtainable, a knowledge of the primary lesion. The fact of recurrence is also of great importance.

8. *Subjective symptoms;* pain or discomfort, itching, burning, smarting, tenderness, or neuralgic pains, alleviation by exposure to the air, or by covering, by heat or cold, by the application of water or oil, by pressure or by friction.

Diseases of the skin should be classified (as they should be named) like diseases of other organs: *i. e.* for convenience, either alphabetically or otherwise. The order followed in the present volume is to begin with the most common affections, then superficial forms of dermatitis, eczema and its allies, psoriasis and its allies, erythema and its allies. Then will follow affections of the hair-sacs and cutaneous glands, including ringworm. Next comes a chapter on the deep inflammations and the hypertrophic conditions which result therefrom. Closely allied to the deep chronic forms of dermatitis are the important and well-defined diseases known as lupus and leprosy, and the chapters on treatment of them are naturally followed by a shorter one on tumours and new growths. Then comes a short section on abnormalities of the cutaneous pigment and of cutaneous innervation, and the subject concludes with a chapter on the classification and partial diagnosis of diseases of the skin in general.

ECZEMA*

AND COMMON SUPERFICIAL DERMATITIS

Definition—Willan's—Hebra's—Its distinction from other forms of dermatitis—Histology—Anatomical lesions—Course—Distribution and local varieties—General symptoms—Ætiology—Diagnosis—Prognosis—General treatment—Local applications—Diet and regimen, baths, &c.—Internal remedies—Special treatment of local varieties.

Impetigo.—*Its relation to eczema—to pediculi—to contagion—Its treatment.*

By far the most important of diseases of the skin, and perhaps the most important of all diseases which do not shorten life, from its frequency, its obstinacy and the misery it occasions, is the affection now universally known as eczema (ἔκζεμα), the "outbreak" or "eruption," as the Greek physicians called it. In its commonest form it is familiar to the profession and the public, and cannot escape instant recognition, but under many circumstances it is difficult to diagnose, and opinions have differed widely as to its pathology, its definition, and the extent to which dermatoses bearing other names are allied to or identical with it.

Definition.—Willan classed eczema among *vesicular* diseases, and this is a proof of his acumen and judgment; for, although the vesicles of eczema are so small and numerous, so short-lived and speedily supplanted by pustules or weeping surfaces or scales that one may see hundreds of cases before the vesicular stage can be demonstrated, yet there is no doubt that vesicles are characteristic and, if not a constant, the most nearly constant anatomical lesion of eczema.

The most important step in the pathology of this disease was Hebra's statement that eczema can be produced at will, for it is in fact identical with the *common superficial dermatitis* which is the result of ordinary irritants. As a result of this important statement, Hebra not only described under eczema much of erythema, intertrigo, the pustular form of dermatitis known as impetigo, and most cases of papular dermatitis previously classed under various species of lichen and strophulus, but he boldly included scabies itself as also a common inflammation of the skin and therefore a true eczema. All succeeding dermatologists have more or less followed Hebra in extending the bounds of eczema far beyond the definitions of Willan and Bateman.

But invaluable as was the new doctrine of Hebra, it has become clear that for clinical purposes we must seek again to narrow the definition of the word eczema. Inflammation, the reaction of the living tissues to injury, is the key-note of pathology. If to the doctrine of inflammation we add that of degeneration and new growths, of parasites and of contagia, almost the whole range of modern pathology is covered. It is quite true that the vast

* *Synonyms.*—Moist Tetter—Common idiopathic superficial dermatitis.—*Fr.* Eczème.—*Germ.* Ekzem—Die nässende Flechte.

majority of diseases of the skin, like those of the rest of the body, are inflammatory, but for prognosis and cure we need much more than this elementary fact. Hebra himself had too much sagacity and practical sense to be led far astray by his own reform.

(1) Syphilitic diseases are most of them undoubtedly inflammations of the skin, but however closely they might approach in symptoms and appearances to some forms of eczema he separated them very widely. Scarlatina is a dermatitis not unlike some stages of eczema. Variola and varicella often approach impetigo still more closely in appearance, but neither Hebra nor any of his disciples have classed the exanthemata of Willan with eczema. These diseases are all separated by our knowledge of their ætiology, by their combination with definite symptoms in other organs than the skin, by their course, and by the practical measures for which they call.

(2) Scabies, again, is distinguished from all other forms of dermatitis, not by the pathological process, but by the peculiarity of the irritating agent, by the consequent characteristic distribution, and by the special mode of treatment.

(3) Lastly, we must separate from true eczema diseases like psoriasis, which, though undoubtedly inflammatory, are special in their characters, in their anatomy, in their chemical products, in their results, and (above all) which cannot be produced or even simulated by an external irritation. In other words, they are not "common superficial dermatitis" such as results from the natural reaction of the healthy skin against a common mechanical, thermal, or chemical irritant.

But now comes a more fundamental definition of the term, which is absolutely necessary for those practical objects which are the end and justification of all refinements of nomenclature. If we call eczema common superficial dermatitis and assert with Hebra that we can produce eczema at will by rubbing in an irritant ointment or by exposure to the sun, we run the danger of forgetting what is after all a most important character of the disease which we agree to call eczema, whatever else may be included under the name. Undoubtedly "wet tetter" is in the majority of cases *not* the direct and immediate result of local irritation. It is therefore preferable to say that a scorching sun or a mustard plaster will often produce a common superficial weeping dermatitis which is histologically and chemically absolutely identical with eczema, which may, if we please, be called artificial or traumatic eczema, but which yet differs from the true disease by the very fact that it is the physiological reaction of the healthy skin to a definite known irritant; that it further differs in its course, in its distribution, in its whole natural history, from idiopathic, typical, true eczema, and demands as a consequence a different prognosis and different therapeutics. The distinction, however subtle in theory or difficult to draw in practice, is of direct and paramount importance.

For instance, the surgeon of a gaol is shown an eruption on a prisoner's arm which, by every anatomical character, is a "common superficial dermatitis"—is what might have been made by Hebra's eczema-producing liniment. He diagnoses eczema, prophesies the course it will take, its obstinacy and its probable recurrence, and prescribes what he calls appropriate treatment. But whether verbally correct or not, he has made as great a practical blunder as is possible. The common superficial dermatitis is traumatic; the eczema is not *like* that produced by an irritant, but was actually and designedly so produced; the subject of it is not a patient with

a disease, but a skilful impostor who has inflicted injury upon his skin; the course of the eruption will not be guided by the natural history of eczema but by the will of the patient, it will not recur except by his wish, and will not be cured by "appropriate treatment." Since, therefore, the name which follows a diagnosis should connote as much knowledge as possible in brief, it is much better not to name factitious dermatitis "eczema."

In the same way we should exclude all common superficial dermatitis which is the direct and immediate result of local irritation, for what is remarkable is not that the skin should inflame when irritated, but that the skin of many people is liable to undergo the exact pathological changes produced by irritation *without* any demonstrable irritant.

Eczema, therefore, may be defined as "idiopathic, common, superficial dermatitis." We must, however, fully admit the difficulty or impossibility of drawing a line in every case. We can only classify diseases as they more or less naturally are connected with certain typical forms. At one end of the scale we have purely traumatic dermatitis produced by a demonstrable external irritant, limited to its immediate effects and disappearing not to return when the cause is once removed; at the other end we have dermatitis appearing on parts of the skin which are not exposed to any known irritation, following a distribution which is independent of irritants, recurring without external cause after it has once disappeared, and only curable by measures other than those addressed to the local irritation. But in every case of dermatitis, however idiopathic, there is no doubt an *irritans* if we could only recognise it, and in every case, however traumatic, there is an *irritabile* in the patient's tissues. Inflammation can never be truly "idiopathic," that is uncaused; for like every other event it depends upon antecedents. No heat of the sun, no activity of cantharides or of croton oil can produce a pustule or a bleb upon the skin of a corpse. All eczema is common superficial dermatitis, but every common superficial dermatitis has not the characters in its origin, its distribution, and its course, in fact in its whole natural history, which entitles it to the name of eczema. In order practically to identify eczema we must therefore look for the clinical characters to be presently described.

Eczema is dermatitis at the stage of *exudation:* it is well called "moist tetter." Cases of dry eczema no doubt occur, but they are either abortive or residual. When we use the term eczema, we imply that the eruption is moist, or will be moist, or has been moist; or that at least it occurs in a person who has previously been, or will hereafter be, subject to another outbreak of the same thing, when exudation will be apparent.

Slight degrees of inflammation, when the result of irritants, fall under the minor degrees of superficial traumatic dermatitis. Slight degrees of idiopathic inflammation which do not reach the stage of exudation—hyperæmia, roseola, erythema of the skin—when not shown to be abortive eczema by their locality and course, belong to very different pathological groups. They may be, first, symptomatic rashes like those of measles and scarlatina which are true dermatitis with all the characters of inflammation, and followed by desquamation. Under the same head of exanthemata should be included, secondly, the roseolar, erythematous or papular rashes of enterica, cholera, and syphilis. Again, there are superficial forms of dermatitis which never assume the characteristic exanthematous aspect, which differ entirely in locality, in the persons they are most prone to affect, in their local and general symptoms, and in their constantly subacute character.

These superficial dermatites, of which erythema nodosum is perhaps the best type, are clinically and pathologically to be separated from eczema, and will be treated of in a separate chapter.

Histology.—The pathology of eczema is that of inflammation generally. Its signs are the four Galenical characters of pain, heat, redness, and swelling, to which we now add a fifth, pyrexia or febrile reaction. Of its cause we know no more than of inflammation in other parts. Traumatic inflammation follows injury or local death of a tissue; idiopathic inflammation we assume must have some corresponding lesion, but of its nature we are ignorant. The order of events is vaso-motor paralysis, dilatation of the small arteries and capillaries, stagnation of the blood-stream, diapedesis of leucocytes through the stomata of the capillaries, and exudation of the plasma or liquor sanguinis.

If a section of eczematous skin be made the cuticle is found unaffected, the Malpighian layer swollen, the papillæ œdematous with dilated blood-vessels, and multitudes of leucocytes clustered round them; the deep layer of the cutis and the subcutaneous tissues are unaffected. Looking at the living skin we see, so soon as a sense of slight irritation with some pain of a tingling or smarting character has drawn the patient's attention to the spot, that there is already an inflammatory blush. This usually has from the beginning a brighter, more arterial hue than the rose-coloured tint of true erythema. A more important distinction is that the erythematous blush is diffused and fades off at the edge. It is scarcely ever disposed in blotches, circumscribed or mottled patches or figures of definite outline. The swelling from œdema is very slight. On close inspection, particularly if a lens be used, one can see that the apparently uniform redness is produced by a number of isolated deeper-coloured points. In this and in other respects the early stage of eczema resembles scarlatina as true erythema resembles measles.

Before long, but never without a precedent stage of hyperæmia, there appear minute vesicles. Frequently, however, they are preceded by little red elevations, which for some time show no bright transparent spot of fluid, and such inflammatory papules may appear early and continue for a long time before becoming vesicular. Such papular forms of eczema must be regarded as abortive, and very seldom will a careful scrutiny fail to discover the evidence of liquid exudation at one period or another of the case. Soon after the vesicles have formed the remarkably thin roof of the cuticle ruptures and they run together, forming a raw weeping surface, eczema madidans, or they may previously have sunk somewhat deeper and acquired more or less purulent contents before their thicker roof bursts. Such pustular forms of eczema usually produce, not weeping surfaces, but more or less extensive scabs, though intermediate stages are very frequent. In the most typical form of eczema the weeping stage continues until a great abundance of clear watery exudation is poured out. It consists chiefly of serum, to which the salines give its irritating property and the albumen its characteristic effect in stiffening linen. On the raw weeping surface it is easy to distinguish more injected points which mark the seat of ruptured vesicles. This *état ponctué*, as the French writers call it, is very characteristic and may be sometimes seen before and even after the moist stage. The involution of eczema is accomplished by the exudation diminishing, and at last drying up, the weeping ceases, or scabs take the place of pustules. Finally, the cuticle again covers the abraded surface and a branny desquamation, formerly described as psoriasis or pityriasis diffusa

and also as eczema squamosum, covers the lately inflamed parts. The itching still continues and is sometimes troublesome up to the very last.

In chronic eczema the skin becomes exceedingly thickened, a result which is readily appreciated on pinching up a fold. It is constantly covered with branny desquamation, acquires a deep red instead of a brilliant scarlet colour, and in certain parts is marked by deep fissures or rhagades, which often penetrate to the true skin and give rise to bleeding and excessive pain. This eczema rimosum is most frequent in the palms or the soles and in its hæmorrhagic form on the nipples and the lips.

Course.—The course of eczema is very rarely acute in origin, development, and recovery; even in what appear to be the most acute cases it will be found that the patient has been subject to previous attacks, and that in the intervals small patches of the disease linger behind the ear or on the face or hands or some other isolated part, and when the acuteness of the attack has passed off it is rare for the whole of the skin to return to its normal condition. Fresh smaller outbreaks occur again and again, so that with scarcely an exception, however acutely the first attack of eczema may sometimes begin, its course is a chronic one.

Another peculiarity also very characteristic of eczema is its strong tendency to *recurrence*. It is extremely rare for a person to suffer from a single attack in the course of his life. Again and again when the disease appears in a quiescent condition a fresh acute outbreak will occur, or one attack will scarcely have passed off when another supervenes. Happily the majority of cases are not lifelong in duration, but they usually extend over several years, and it is not unusual for recurrence to take place even after long intervals of complete freedom.

Distribution.—One of the distinctions between eczema or idiopathic dermatitis and that which is traumatic in origin is that while the latter corresponds more or less exactly to the irritant, typical eczema has its own peculiar laws of local distribution. Speaking generally, it is a disease of the thinner parts of the skin, it is a disease of the flexures and joints, it is a disease of the head and limbs rather than of the trunk, and, lastly, it is a symmetrical disease.

We must here stop for a moment to explain the meaning of the word *symmetry* in pathology. It is nothing to the point to call universal eruptions like that of scarlatina symmetrical; they are so only because the human body is itself bilaterally symmetrical. Nor is it enough that the same diseases should be found in both right and left member as an acute rheumatism. Symmetrical distribution means that exactly the corresponding parts on the right and left side are simultaneously affected, both ears, both elbows, the back of each hand, the under surface of each wrist, and the popliteal space on each thigh, the sole of each foot. This is bilateral symmetry, but we also see examples of serial symmetry in pathology, where the same condition is seen on the elbow and the knee, the wrist and the ankle, the palm and the sole. Eczema is an extremely symmetrical disease, as thus defined more so than any other affection of the skin excepting psoriasis.

The most characteristic locality for eczema is behind each *auricle*, not only because it is so frequently seen here when it affects other parts, but also because this spot is but little liable to other diseases. The *face* is more frequently affected with the pustular form of eczema (which we shall presently describe as impetigo) than any other part of the body, and this particularly applies to the lips, nostrils, and cheeks. In adults the face is less frequently

the seat of ordinary eczema, coming next in frequency to the *limbs*. The same remarks apply still more strongly to the *scalp*, where impetigo is the commonest affection in childhood, but where ordinary eczema is comparatively rare except on the bald scalp of infancy or age. Eczema does not frequently affect the skin which is covered by the *beard*, and when it does is not usually remarkable for obstinacy, but sometimes the inflammation can be unmistakeably seen to penetrate the hair-sacs and there become a deep instead of a superficial dermatitis. Its clinical features and treatment are then so different that it is properly known as a spreading disease, sycosis. It may in like manner spread to the sacs of the *eyelashes* and become localised as what used to be called tinea tarsi.

The *neck* is very frequently the seat of eczema, especially the front and sides. On the *trunk* the shoulders, back, and loins are but rarely the seat of the disease ; and the same statement applies to the gluteal region, which is so frequently the seat of isolated pustules, not only in scabies, but in the impetigo and ecthyma of children. The flanks, though covered with soft and delicate skin, are not often affected with eczema, which when present has usually spread from the axillæ or from the neck ; and the same applies to the chest. In women, however, eczema of the *breast* is common either as eczema intertrigo beginning in the fold under the mammæ or as eczema of the nipple. The *abdomen* is more often the seat of eczema than other parts of the trunk, especially of that variety which begins at the *navel*. The *genital organs* by the thinness of the skin and mutual contact are readily disposed to dermatitis, but are certainly less often affected with ordinary eczema than either the face or the limbs. Either the inflammation begins as intertrigo of the scrotum and thigh, especially common in infants, or it is an acute weeping eczema which extends to the abdomen, thighs, and other parts as well, or it is a chronic and extremely pruriginous eczema of the vulva or scrotum. The last form even more frequently affects the neighbourhood of the *anus*, particularly in elderly persons ; and the cleft of the nates is extremely liable to eczema intertrigo, both in infants and in fat persons, under the irritation of long walking and free perspiration. In many cases eczema of the anus, perinæum, genital organs, and thighs forms a well-marked local variety, which must be carefully distinguished from the so-called "eczema marginatum" of the same regions, to be afterwards described as a form of tinea.

On the *arms*, eczema is scarcely ever seen over the deltoid, and though common in the axilla, and particularly its anterior fold, is much less so than at the elbow. The bend of the elbow is probably, next to the face and ears, the most frequent seat of ordinary eczema, and if we were to exclude cases occurring under puberty even that exception need not be made. The skin covering the biceps cubiti and the flexors of the front of the forearm usually participates in eczema of the elbow, and this local form of eczema is one of the most constantly and accurately symmetrical. The disease scarcely ever affects the olecranon, but there is a form of eczema which, though relatively uncommon, is seen quite often enough to deserve special mention. It is an ordinary weeping eczema occurring in adults and affecting the outer side of both forearms from an inch or more below the point of the elbow down to the wrist. It is extremely symmetrical, and often affects the skin of the upper arm which covers the triceps, though without spreading to either shoulder or elbow. The *wrist* and back of the hand are comparatively seldom the seat of eczema, though the affection will sometimes spread from the arm as far as the knuckles, and this region is not unfrequently the seat

of the dry chronic circumscribed dermatitis which will be described as "single patch eczema."

The *fingers*, especially their clefts, are often the seat of eczema, but in most cases this can be traced to a traumatic origin. The *palm* of the hand might from its thickness seem little adapted to eczematous inflammation, but it is very frequently the seat of a characteristic chronic dermatitis, painful, disabling, symmetrical, and obstinate; which, from the absence of vesicles and the presence of deep fissures dependent upon the thickness of the epidermis, has received the name of eczema rimosum. This is, however, either of local traumatic origin, or at least is usually unassociated with eczema of other parts, and is curable by local applications alone. Eczema of the matrix of the *nails* is almost always part of eczema manuum. It proves a long period of dermatitis, and its presence is therefore a point of diagnosis in distinguishing eczema from scabies. The consequent malformation of the nail is generally marked by longitudinal grooves, and by less thickening than in the far more rare psoriasis unguium.

In the *lower extremities* eczema of the groin and inner part of the thigh is very common in adults, and is either associated with eczema of the arms or with eczema of the abdomen and genitals. The outer side of the thigh is not often the seat of ordinary eczema, and the patella, like the olecranon, is practically exempt, but the popliteal space is almost as favourite a seat of the disease as the bend of the elbow, and the inflammation spreads thence more or less extensively over the thighs and legs. Below the knee, however, eczema is on the whole less frequent than below the elbow, and most often appears in one of two forms. (1) Eczema of the calf and peroneal region of an ordinary weeping and irritable type frequently accompanies the corresponding affection just described of the outer side of the forearms, and, like it, is almost always symmetrical. (2) Varicose eczema, a local dermatitis often unconnected with eczema of other parts, is obviously the result of ordinary irritation acting upon a skin congested by varicose veins. It affects the inner side of the leg from the internal malleolus upward, that is to say from the point where, as Hilton showed, the least considerable anastomosis takes place, between the internal saphenous and the posterior tibial veins. This form of dermatitis is known by its purplish tint and frequent association with ulcers as well as by its locality.

The *foot* is less often affected with eczema than the hand, but follows its serial homologue very closely. When not of traumatic origin and not due to scabies or to intertrigo, that is to say, when dermatitis of the foot is true eczema, it either affects the dorsum in association with eczema of the outer side of the leg, or the soles as a chronic eczema rimosum much resembling that of the hand, or it is an eczema-intertrigo-digitorum sometimes leading to deep clefts between the toes. This last form is as common as eczema of the fingers, if not more so. It is rarely associated with the disease in other localities, and must be treated entirely by topical measures.

Chronic eczema of the soles sometimes assumes the characters of hypertrophic dermatitis, with accumulation of horny epithelium a quarter or even half an inch thick, very painful, and of a sickening caseous odour. This is quite distinct from the similar condition occasionally produced by syphilis.

Symptoms and natural history.—Locally, eczema provokes extreme itching, more so perhaps than any other cutaneous disease except prurigo and scabies. Indeed, although unlike these two there are many cases of eczema which are almost free from irritation, yet in others the pruritus seems to be

at least as intense, constant, and obstinate a symptom as in the worst of any other disease. Itching is usually less in the weeping and acute than in the dry, papular, or chronic and scaly conditions, and it is very rarely marked in the pustular form of eczema. It is most intense in ordinary eczema of children, and in that of old persons, and of all local varieties is most constant and most severe in eczema ani et vulvæ.

Smarting, tingling, and some amount of local tenderness are common symptoms of the more acute and ordinary forms of eczema, and are associated with a peculiar sense of burning and tension. There is slight febrile movement at the onset of the disease and particularly when large surfaces are invaded at once, but even when the thermometer shows no appreciable elevation of temperature there is thirst, loss of appetite, and a slightly furred tongue. The mucous membranes are unaffected; there is no foundation for such names as eczematous gastritis, enteritis or bronchitis. The pathology of the digestive, pulmonary, and urinary mucous tracts is quite different from that of the skin. We have no right to assume an eczema of membranes which we cannot see when we cannot demonstrate eczema of those which we can. The writer has never seen eczema of the lips spread to the mouth or tongue, eczema of the anus to the rectum, eczema of the eyelids to the conjunctiva, or eczema of the penis to the urethra and bladder.

Except for anorexia produced by the slight pyrexia of the onset of eczema, the appetite and digestion are as a rule unaffected in this disease. Dyspepsia is so common that many persons suffering from eczema are also dyspeptic, but there is no reason to regard the latter condition as the result of the former, nor are the bowels either constipated or relaxed. One would expect beforehand the urine to be affected when large surfaces of the skin are inflamed, but there does not seem to be satisfactory evidence of any constant change. The fact that in the large proportion of cases of eczema the patients are not confined to bed, or even to the house, makes it the more difficult to obtain observations of the amount or average condition of the urine in this disease. Any deviation from health observed may be explained from causes unconnected with the eczema, so that, as far as we know at present, the urine must be stated to be unaffected in eczema.

Ætiology.—I have already stated what appears to me to be the true relation between traumatic dermatitis and idiopathic eczema. When the inflammation directly follows an irritant, is not prolonged after its cessation, does not spread to other than the irritated region, does not recur without fresh irritation, and does not follow the local distribution of eczema, then it is best called common superficial dermatitis of traumatic origin. But some skins, whether by natural stability or by habit, are insensible to sun and friction and sweat, and the other irritants which in ordinary persons produce inflammation. In others a hot day or bathing in sea-water, or an east wind, or a long walk, will produce eczema solare or eczema intertrigo or some other form of local traumatic dermatitis—which when once established becomes chronic, persists long after its original cause has ceased to act, and localises itself in the bends of the joints, and in the symmetrical positions which have been above described as characteristic of eczema. Admitting, therefore, local irritation of various kinds as an exciting cause of eczema, we must also admit a certain proneness of the skin to inflammation, and in more than half our cases this predisposition causes the disease without our being able to fix upon any probable irritant.

It has been widely supposed that we are to seek the predisposing, and

in most cases the efficient cause of eczema in a diathesis or disposition of the whole body, which can be recognised independently of the presence of the eczema, and which produces other recognisable diseases. This diathesis has been called dartrous, arthritic, and gouty, while the ever-ready explanation of a strumous disposition has been invoked when the other failed. The reader is referred to the works of Hardy and Bazin, of Gigot-Suard, and other French writers, for their exposition of the dartrous doctrine; while all that can be said for the more particularly gouty relations of eczema will be found in the writings of Erasmus Wilson, Hutchinson, and the late Dr Tilbury Fox. The present writer can only express unbelief in the whole doctrine of temperaments and diatheses, which appears to him to be the residuum of the exploded physiology of Galen, and seriously to impede the advance of medicine. It appears that although persons with gout are often subject to very irritable and obstinate eczema, in the vast majority of cases of eczema there is not the faintest reason for the belief that gout, that is, the deposit of uric acid in the joints, has been present in the patient or his immediate relatives; that there is no pathological connection between gout and true rheumatism, arthritis deformans, or gonorrhœal arthritis, and that none of the latter forms of multiple arthritis have any demonstrable connection with eczema; that eczema rarely co-exists with psoriasis, pemphigus, or other supposed manifestations of the dartrous diathesis; that no one can give an intelligible account of the characters by which this predisposition can be recognised; that there is no evidence that eczema has more than accidental connection with diseases of other parts of the body, or that it is anything but a common superficial dermatitis; and lastly, that the diathetic hypothesis is practically misleading in prognosis and treatment, no less than scientifically unsound.*

The efficient cause of eczema, apart from local irritation, is as much, and as little unknown, as the efficient cause of bronchitis or of cystitis. All we can say is that in some persons the skin is naturally sensitive, or delicate, or irritable. Such persons are, in other respects, like their neighbours, and the predisposition of their skin to inflammation can only be prophesied after the event.

General eczema is sometimes set up by an accidental and very local irritation, and this is probably the explanation of the undoubted though rare occurrence of vaccination leading to eczema: much more frequently their relation is purely accidental. It appears to be waste of time to discuss the vague speculations, at once unscientific and unpractical, which ascribe eczema to such common disorders as dyspepsia, or to such *idola theatri* as constitutional predisposition, assimilative debility, nervous debility, perverted innervation, renal inadequacy, strumous cachexia, scurvy in the blood, acidity of the *primæ viæ*, or *la diathèse dartreuse*.

There is no doubt that eczema is in some cases very decidedly *hereditary*, but it is certainly much less so than psoriasis, and in the great majority of cases there is no reason to admit hereditary predisposition. The exudation of eczema is not contagious so long as it is transparent. When purulent it probably shares in the infective characters more or less common to all pus, and occasionally the pustular eczema which we shall presently describe as impetigo capitis is most markedly contagious.

Eczema affects both *sexes* indifferently. It is common at all *ages*, but differs in its most frequent characters. In the infant it is of the ordinary papular

* The writer may be allowed to refer to papers on this subject in the 'British and Foreign Medical Review' for January, 1874, and in the 'Guy's Hospital Reports' for 1880.

and vesicular kind. The same form is seen in older children, but much more frequent in them is impetigo, or pustular eczema, which is comparatively rare before the first dentition and after puberty. In adults the commonest form is ordinary weeping eczema of the limbs and face. In old age, the dry, very chronic, and extremely pruriginous forms are the most characteristic.

Eczema appears to be universal over the globe. It is certainly not more frequent where gout is prevalent, as in London, than where it is almost unknown, as in Vienna and New York.

The traumatic forms of eczema naturally occur in those *occupations* where the hands are exposed to constant irritation. Hence, we have the eczema of the hands which has long been recognised as frequent in washerwomen, grocers, and other hand workers.

It is a popular opinion that skin diseases generally, and particularly eczema, as the most common of them, are most prone to occur at certain *seasons* —the spring and fall. Like most popular beliefs, this was not founded upon experience, but chiefly upon theory, and partly perhaps upon analogy. The period of change in the seasons seems "naturally" to be the most likely period for change in the human economy, and changes are proverbially dangerous. It is possible also that the insalubrity of southern Europe in the autumn, from the prevalence of malaria, led to a belief in the same result in northern countries, while even in England malaria was far from uncommon until quite recent times. However this may be, one meets with eczema of the ordinary irritative and inflammatory kind more often in the spring than at other times, and this may be fairly attributed to the dry east winds which then prevail.

Diagnosis.—Keeping to the definition of eczema as above stated, the only difficulty is on the one hand to distinguish between idiopathic and traumatic dermatitis, or rather to detect the decided and efficient prevalence of a traumatic cause, and secondly, to draw the line between eczema and certain other forms of superficial dermatitis, the distinctive characters of which, and the justification for their separation, will be considered under each head. The distinction between eczema and intertrigo, eczema and impetigo, eczema and some forms of lichen, and even eczema and scabies, depends ultimately, as all distinctions in medicine should, upon practical utility. What it appears to me we must recognise is that all superficial inflammations of the skin may be grouped around certain types, and that the most common and important of these, which we call eczema, is characterised by being *common*, that is to say, the same as is produced by ordinary mechanical or chemical irritants, *idiopathic*, that is to say, not directly co-extensive with irritation, *moist* from visible inflammatory exudence, *symmetrical*, *selecting* certain favourite parts of the skin, and *prone to recur* after disappearance.

It may, however, be well briefly to point out the following characters. From *scarlatina* and other rashes, eczema differs in being never truly universal in its moisture and in being unaccompanied by marked febrile symptoms. From *erysipelas* it is distinguished by the colour, the minute vesicles, the locality, and the absence of a defined margin, of œdema,* and, again, of fever. *Erythema* exudativum is more rosy in tint, and though it may form papules or even bullæ, never shows the small vesicles or the weeping surface of eczema: its distribution is different, and it is never chronic in its course. Eczema has no resemblance to *psoriasis* except in very old cases of the latter disease, when the scales have disappeared and the locality is obscured.

* An exception occasionally occurs when eczema affects the eyelids.

Prognosis.—Eczema is extremely amenable to treatment, that is to say, we scarcely ever see a case in which no improvement can be produced, and still more rarely one which finally resists all therapeutical measures. Moreover, it is scarcely ever dangerous to life. There are, however, exceptions to each of these statements. In the outbreak of acute vesicular and weeping eczema, whether primary, or, as far more often happens, occurring in a chronic or nearly cured attack, we can do little or nothing to stop its violence. Abortive treatment is unfortunately rarely successful in any acute disease. Again, in some cases eczema, though treated until very little remains, cannot be driven entirely away but remains in a quiescent state here and there, to burst forth again after a longer or shorter interval.

Lastly, though it is remarkable how little the general health is affected even by very extensive, troublesome, and long-continued eczema, yet occasionally, in infants, or in aged persons, broken rest and loss of appetite cause wasting and muscular weakness, which may at last end fatally. The only cases of the kind which have occurred in the writer's practice were, first, an infant which became emaciated, pale, and unable to take the breast, and secondly, an old gentleman considerably over seventy, who after being much relieved by constant tepid baths and other treatment from an almost universal and extremely irritable eczema, sank rapidly and died without any evidence of organic disease. On the other hand, everyone must have seen scores of little children who appeared worn to a skeleton and almost moribund through severe eczema, who nevertheless, by treatment or by time, completely recovered. Very general eczema in a person over seventy, especially if complicated with gout or with chronic Bright's disease, should, I think, always suggest a guarded prognosis.

Treatment.—In the first place, it is our duty to treat and if possible to cure every case of eczema as quickly, safely, and pleasantly as we can. The supposed danger of driving in eczematous eruptions upon internal organs appears to be without any foundation. It arose partly from theoretical views of the sympathy of organs, partly from the well-known fact that cutaneous hyperæmia diminishes or disappears during acute febrile affections, partly from observing the benefit of counter-irritation of the skin in synovitis or bronchitis, and possibly, as Hebra unkindly suggests, from the difficulty of curing some cases of eczema. In little children, however ill, one may again and again observe that as soon as cutaneous exudation is checked and the irritation subsides, their general health begins to mend. The only caution I would give is to be very careful to ascertain the condition of the heart, the arteries, and the kidneys in aged persons suffering from eczema, lest the treatment or cure of their cutaneous disease should be credited with the fatal result which is really the consequence of degenerated viscera.

Prophylaxis and general protective treatment.—The irritants which excite or keep up and renew eczema are chiefly mechanical, thermal, and contact with water. Of mechanical irritants the most important are rough clothing, friction against adjacent parts of skin (intertrigo), prolonged contact with decomposing sweat, and also with dirt and various chemical irritants which are incidental to certain trades. But the most difficult mechanical irritant of all to get rid of is that which is the result of the disease itself. Eczema always itches, and itching is sure to produce scratching. Hence our first attempt is to prevent this by persuasion of an adult patient, by muffling the hands of infants, and by such local applications as will at least relieve the intolerable irritation.

With the same object, we forbid cold bathing, we forbid the application of either cold or heat, the latter for its immediate, and the former for its consecutive, effects on the circulation of the part. Contact with air is, in many cases, a decided stimulant, and one important use of the various ointments with which the eczematous skin is smeared is to protect it from air. With weeping eczema, we obtain the same end by covering it with wetted rags, or, as is often more efficient, by dusting it with absorbent powders.

But even more important as a cause of irritation is moisture. It is not that mere contact with water is an irritant; probably a slightly alkaline and weak saline solution with a little colloid material, such as gum or size, or the albuminous part of oatmeal, is the least irritating medium with which an inflamed and excoriated skin can come in contact. A continuous bath, even of ordinary water, is a most useful and perfectly safe means of treatment in some cases of very general eczema, with profuse exudation and great irritability. To Hebra is due the merit of proving that patients can be kept continuously in a bath of suitable temperature, not only for hours, but for days—indeed, for an indefinite period, without leaving it for any purpose whatever. The writer saw this plan carried out at Vienna, and has more than once adopted it himself. The practical difficulties are obvious, and it is fortunately not often that we need resort to it.

Though *continuous* contact with water is by no means irritating, bathing and washing mean *intermittent* wetting of the skin. The change from dry to wet, from higher to lower temperature, and the reverse change on withdrawing the eczematous surface from the bath, the necessary friction of the towel, the saline constituents of most waters, and, most of all, the evaporation which even great care cannot entirely prevent, and which, after careless washing, go on abundantly from the half-dried surface—these form altogether a most efficient series of irritations.

It is even possible that the frequent and systematic cleansing of the skin from dead epidermis and from sebaceous secretion, which is the result of the artificial condition of extreme cleanliness to which modern civilised society more and more tends, may itself render the skin more susceptible to slight irritants, and certainly with tender skins the use of soaps, of nail brushes, of rough towels, and of flesh gloves, may sometimes aid in exciting dermatitis—a small set-off against the advantage to general health of mind and body to which a clean and active skin undoubtedly conduces.* Important rules of treatment in eczema, therefore, are that the inflamed parts must not be washed with soap, must not be washed with either hot or very cold water, must not be washed frequently, and must be very carefully dried after washing with soft, dry, and warm towels. In order to prevent the "chapping" of the hands which is so common in children during the winter, it is important to take care that they are thoroughly dried on towels which are not already damp. In large schools for poor children, a good plan is to make them dry their hands by dipping and rubbing them in a tub full of bran, instead of upon towels, which are sure to be wet for all but the first comers. They should also be given olive oil, or any other neutral fatty compound, to rub into the hands after washing, when there is the least appearance of dermatitis. With severe and chronic eczema rimosum of the hands, it is necessary absolutely to forbid washing, and to protect from

* See an amusing article by Hebra on the dangerous consequences of being overmuch clean, translated in the 'London Medical Record,' March 15th, 1877.

contact with air or moisture by ointment and a well-fitting kid glove. The best plan in chronic eczema is to advise that once a week, or more frequently as may be thought safe, a complete warm bath and thorough cleansing of the whole surface should be used, taking care to keep the skin immersed from the time of its being first wetted, and to dry it thoroughly when the washing is over. Washing, with unscented, pure yellow soap and lukewarm water, if done seldom and followed by careful drying and inunction, is less injurious to an eczematous skin than more frequent and careless ablution with no soap at all. For the exceedingly irritable skin of the face and hands, it is sometimes desirable, however, absolutely to forbid all contact with water, and cleansing can then be accomplished by friction with dry and stale bread crumbs. When there is eczema of the scalp the best cleansing agent is white of egg diluted with water.

Poultices are almost always injurious, and scarcely less so is the modified form which is the usual result of the application of water dressing, a piece of lint dipped in water or lotion, and closely covered with oiled silk or gutta percha. The impermeable covering soon raises the temperature, and the result is the combined warmth and moisture of a poultice, most valuable for relaxing tension, promoting suppuration, and relieving deep-seated inflammation, but most injurious in superficial dermatitis.

Eczema is common dermatitis and must be treated like other inflammations. Cold, however, is not often practically applicable, powerful as is its influence on inflammation; the surface affected is too extensive, the difficulty of continuously applying adequate cold too great, and the ill-effects of considerable depression of the temperature of the surface too serious, for us to attempt treatment by ice or by cold baths. Moreover, intermittent application of cold by the reaction which ensues proves worse than useless. Eczema in the majority of cases which come before us has passed its acute or subacute stage, and irritation rather than heat is the common symptom. It is, however, always well for patients with eczema to avoid the heat of the sun or exposure to fires or to the heated atmosphere of crowded rooms. The affected parts should not be covered with thick woollen garments, and the patient should lie lightly covered at night; the bedroom should be well ventilated, the temperature kept somewhat low, and much relief is experienced by keeping the feet or arms uncovered except with a thin rag dipped in lotion.

Local medicinal treatment.—We next come to the treatment of eczema by chemical applications. Our object is, first to diminish the hyperæmia and exudation by *astringents;* secondly, to diminish irritability and to prevent scratching by *sedatives;* thirdly, to substitute for a chronic and interminable process of inflammation a more directly traumatic, acute, and self-limiting process, or else, it may be, by less stimulus to produce an effect short of this but serving to quicken the natural process of physiological repair. Such agents have received the vague title of *alteratives.*

The most powerful chemical astringent which can conveniently be used is probably lead. (See a paper by Dr Payne in the 'St Thomas's Hospital Reports' for 1878.) Salts of copper, zinc, and iron, nitrate of silver, boracic acid and borax are also efficient astringents. So are galls, tannin, and similar vegetable preparations, though these are less applicable to the skin than to mucous membranes. As local sedatives we may use belladonna, opium, chloroform, hydrocyanic acid, but these are generally unsuitable to eczema on account of its extent and raw, denuded surface, both which characters

make absorption too probable for these narcotics to be safe. More efficient as remedies against itching, and free from any but local action, are preparations of zinc, which combine antiphlogistic and antipruriginous qualities. Dilute solutions of carbolic acid, 2 per cent. in water, 1 in 20 in oil, are very useful. Weak tarry preparations are also efficacious, especially in the drier forms of eczema; as diluted oil of cade (juniper tar) or liquor carbonis detergens with vaseline in the proportion of two drachms to an ounce.

For chronic and no longer very irritable eczema more stimulant applications are necessary; sometimes stronger tarry preparations, unguentum picis liquidæ or pyrogallic acid. These are most useful in limited patches of scaly and very chronic dermatitis with much thickening of the skin; those, in fact, which approach most nearly in appearance and pathology to psoriasis. The colour and smell are objectionable, but the staining of both skin and clothes by chrysarobin ointment is much more unpleasant.

A still more energetic method, introduced by Dr Anderson, of Glasgow, is painting the eczematous surface with liquor potassæ. This must be done with much caution, for it gives rise to considerable pain, though in many cases this is less complained of than might be expected; but the writer can bear witness to the efficacy and safety of the treatment when applied tentatively on limited surfaces of old and obstinate eczema, especially of the dry kind. With moist secreting surfaces of unusual obstinacy, one finds more useful the application of a solution of nitrate of silver varying from a scruple to as much as a drachm to the ounce. It must be occasionally painted on, not kept in constant contact, and often proves most efficient as an astringent and a sedative, as well as an alterative.

But more often there is too much active inflammation for us to venture on such treatment, and more generally applicable alteratives are the various preparations of mercury, corrosive sublimate in solution, white precipitate ointment, red oxide ointment, and dilute nitrate of mercury ointment. Mercury in some form is particularly adapted to pustular forms of eczema, and is seldom suitable to those which profusely secrete serum.

Most often, however, the cases of eczema which come before us combine the characters of inflammation, itching, and chronicity, so that for perhaps the majority of cases, at least if we include those of impetigo of the scalp, there is no more useful preparation than such a combination of zinc, lead, and mercury as forms the unguentum metallorum of the Guy's Pharmacopœia.* This may be varied by substituting the red oxide for the nitrate of mercury ointment, and by varying the proportion of the three constituents; often again lead and zinc act better without mercury, or the carbonate better than the alkaline acetate of lead.

Whatever be the chemical application used it is important to decide whether the vehicle should be watery or oleaginous. An excellent general rule was that of the late Dr Hughes Bennett, of Edinburgh; for dry affections of the skin, use ointments; for moist, use lotions. If an ointment is applied to a profusely secreting eczema the drug and its vehicle are washed away by free exudation and never reach the subjacent skin. Lotions, on the other hand, have but little power of penetrating the epidermis, and if carefully watched will be seen to run from the surface, which is greasy by its natural sebaceous secretion. With raw surfaces which do not secrete profusely, either lotions or ointments may be appropriately used. Practical

* ℞ Ung. zinci, Ung. plumbi acet., Ung. hyd. nitr. āā partes æquales: Misce.

considerations teach us that lotions are better suited to diseases of exposed parts like the face and hands, that they are readily applied to young children, that they are more efficiently used by persons confined to bed or by women living indoors than by those who are engaged in active work, that they are more cleanly and pleasant to most people, but that they also give more trouble and demand more time in their application, and lastly, that in the summer, when the skin is frequently covered with sweat, they are particularly grateful and efficient. We must remember that lotions should in most cases be used with exposed skin or with the surface only covered by a thin rag into which the lotion has soaked. If applied in the morning and covered up till night they speedily become water dressings, and probably in less than an hour mere applications of rag with no further therapeutical power. On the whole, therefore, notwithstanding the rule quoted above and the fact that eczema is pre-eminently a moist tetter, it will be found that with the majority of our out-patients, whether private or at the hospital, ointments are practically the more eligible vehicle. It is important to make sure that the lard or other oleaginous material is not in the least rancid, and that it is free from salt. The addition of benzoic acid as now ordered in the British Pharmacopœia makes as good a vehicle in most cases as can be wished. The mineral oils have the advantage of not decomposing, and for some reason ointments made up of vaseline suit certain cases of eczema better than those prepared with animal fats.

Unmedicated oily applications, vaseline, cold cream, olive oil, have in themselves the good effect of protecting from air and of softening rough, harsh skin, inspissated sebum and dried secretions. Lanolin is better fitted for psoriasis and other affections in which it is desired to rub the oily vehicle thoroughly into the skin. Glycerine, from its strong affinity with water, is well known to be a direct stimulus to a nerve-trunk. It is, except when dilute, a decided irritant in eczema, and has far from the same soothing effects as cold cream or zinc ointment, in cases of intertrigo, chilblains, and eczema solare. In very small quantities, however, it may be added to lotions with the view of securing some of the advantages of an oily preparation.

Weak alkaline lotions have often been recommended to relieve the burning pain and irritation of acute eczema, and they were extensively used by Professor Hardy at St Louis. But in the very cases of acute weeping eczema in children to which such treatment seems applicable, the parts are so excessively tender that even a 1 per cent. solution of bicarbonate of soda is ill borne, so that in such cases the lead lotion (liq. plumbi subacetatis dilutus of the British Pharmacopœia) is more useful. At all events, if soda is used at all it should be in quantities only just sufficient to react to test paper.

Quite apart from the ordinary use of a lotion, the whole object of which is to keep the part continually wet, is that of a solution which when painted on is allowed to dry. For this purpose nitrate of silver or other strong astringent solutions may be used.

Another useful method is to suspend insoluble powders like oxide of zinc, starch, or sulphate of lime or bismuth in water by help of a little mucilage or tragacanth, without, however, attempting to form a perfect emulsion. The milky liquid is applied freely with a large camel-hair brush or sponge, and is allowed to dry over the weeping surface, and in some cases of irritable and profusely secreting eczema, as also in pemphigus, this is found to be

the most effective application. Care must be taken not to have too much of the colloid ingredient, or hard cakes are apt to form which crack and become painful. In fact, chalk or gypsum shaken up with water and applied like whitewash is sometimes the simplest and pleasantest method.

Lastly, we may apply our remedies directly as dry powders. In this way oxide of zinc, chalk, and other fine insoluble powders may be used, and such applications are usually better than starch. They dry up discharges, protect from the air, and are often the best applications in cases of intertrigo. On the other hand, they are unsuitable for pustular eczema, where they would form massive and troublesome crusts.

It must, however, be admitted that nothing but experience, insight, and previous knowledge of particular cases will guide one aright in the selection either of appropriate astringents, in the strength of the application, or in the kind of vehicle. Some patients assure one (and prove right again and again) that they cannot bear any kind of ointment. With others all lotions are apt to produce pustules or even boils. Not infrequently, especially in the acute stage of eczema, an inert powder or unmedicated vaseline, accord ing as the surface is moist or dry, will do more good than anything else. In all cases we should remember that the ointments are not to be rubbed in, but gently smeared on the skin, and afterwards kept in continual contact by well-adjusted soft linen bandages; that the lotions should never be allowed to get hot, and must be frequently renewed; and that the strength of our applications should be small in the acuter stages, and greater as the case becomes inveterate.

The writer, in accordance with Hebra's teaching, and contrary to that of most English writers, believes that the majority of cases of eczema can be cured by well-directed local measures of the kind above indicated; but it must be admitted that in the great reforms he established Hebra undervalued the treatment by internal measures, which undoubtedly holds an important though a secondary place.

Diet.—In the acuter stages of eczema the patient should be put upon almost fever-diet, but should be encouraged to drink freely of any cooling beverage. He should take no stimulants or meat, and eat sparingly, chiefly of bread, milky dishes, green vegetables, and ripe or stewed fruit.

In ordinary chronic eczema no such strict diet is necessary. It is, however, usual to forbid certain articles of food, and I think that the experience of patients shows that, at least in some persons, one or all of these really aggravate the disease, chiefly perhaps by producing thirst, increased irritation of the skin, and scratching. The kinds of food referred to are salt meats of all kinds, including ham and cured fish, cheese, pepper, spices, and other hot condiments. The stronger wines and malt liquors are also usually forbidden, but although in the necessarily generalised treatment of hospital out-patients this is doubtless good advice, there does not appear to be any evidence that the moderate use of malt liquors or wine (with food) does harm in eczema or any other affection of the skin—except in cases where, independently of dermatitis, even moderate stimulants provoke dyspepsia with flushing of the face or symptoms of gout. In many patients, especially those in middle and later life, wine or beer with the principal meal of the day helps digestion and certainly does no harm to the eczema, while a little spirit and water at bedtime will help sleep, and in that respect prove a useful adjunct to other treatment. Sometimes, however, even weak whisky and water produces heat and discomfort after retiring to bed, and must then of course be inter-

dicted. In almost all cases a somewhat free supply of unstimulating diluents should be taken between meals, and a glass of water while dressing of a morning, and again the last thing at night, is almost always useful.

Watering places.—In chronic and obstinate diseases like eczema, patients frequently ask whether change of air would do them good. They are usually recommended to go into the country if they live in town, or go to the seaside if they live in the country, or if they can afford it, to go to Scotland or Switzerland, or some other attractive place of resort. The only point on which one may speak with confidence as to the effects of air and climate upon eczema is that, just as it is aggravated by the east winds of an English spring, so it is more difficult to cure in the eastern counties of England and Scotland, and is often favourably influenced by removal to the moist and soft air of the western Highlands, of Devonshire, or of Ireland. As one sees, however, in so many other diseases, it is the change which does the good, and this is most apparent when the change is from an unfavourable climate. Secondly, there is no doubt that in many cases of the more irritable forms of eczema sea air proves a decided irritant. It is only now and then, in chronic and non-pruriginous eczema or in the impetigo of childhood, that sea air and even sea bathing do good instead of harm.

With respect to baths generally we have already sufficiently insisted upon the evil effects of frequent contact with water, but there is no doubt that in the very chronic and intractable forms of eczema, saline and sulphurous baths act beneficially, probably like the stimulant and alterative applications above described. When the period has arrived for their use it is difficult to say, and each case must be judged by the tact and experience of the physician. A single bath may bring back in all its virulence an eczema which had nearly disappeared. Long-standing dryness, thickening of the skin, and absence of excessive irritability are the features which should generally weigh with us in advising or permitting this mode of treatment. The baths best adapted for the purpose are perhaps those of Harrogate.

Internal treatment.—Lastly, we come to the treatment of eczema by drugs, which I regard as less important than that by external applications and by what may be generally called the hygienic treatment of the skin. Still there is no doubt that while we could better dispense with this group of remedies than with the others we should often fail for want of them, or the success of our treatment would at least be less rapid and complete.

In the acute stage of eczema with profuse exudation and much irritation, it is the practice of the French school to purge freely, and most English physicians adopt the same plan, though perhaps less systematically. Saline laxatives are, I believe, the most useful in these cases. The old-fashioned white mixture of sulphate and carbonate of magnesia taken three times a day, or the pleasanter combination of Epsom salts with carbonate of soda in peppermint or cinnamon water, are useful and popular medicines. A seidlitz powder or a dose of Rochelle salts or Carlsbad salts every morning is suitable for less acute or less extensive cases. Often it is sufficient for the patient to take a draught of Püllna, Friedrichshall, or Hungarian bitter water. Of these three Friedrichshall is perhaps most often suitable, particularly when the eczema occurs in a gouty subject. Sometimes, however, it is less efficient than a seidlitz powder, and occasionally it produces much griping without satisfactory result. In such cases it may be changed for the Hunyadi Janos with advantage. Some find that this Hungarian bitter water agrees better with women than with men. Whichever form of laxative is selected it should

be taken with a large draught of warm water early in the morning and on an empty stomach. Such a dose should give one or two loose motions after breakfast without griping or subsequent irritability, whereas even larger doses, if undiluted with water or taken with the stomach already full, are more slowly absorbed and produce more frequent and less effectual irritation. In cases of eczema in which the patient has other independent evidence of gout, it is well to combine with moderate laxatives the exhibition of a pill containing colchicum and aloes or rhubarb every or every other night. In persons who have lived freely and who are subject to hepatic dyspepsia, beside restricted diet both in food and drink and moderate laxatives, it is important to prescribe small doses of mercury, either a single grain of blue pill with a little nux vomica and rhubarb before dinner, or two, three, or four grains with an equal quantity of the compound rhubarb pill every other night or twice a week.

In many cases, especially in women affected with eczema, there is considerable anæmia, and then steel must be added to the laxative medicine. There is no better combination for this purpose than that of sulphate of iron, given in doses gradually increased from two to five or even ten grains, with half a drachm or more of sulphate of magnesia, five or ten drops of dilute sulphuric acid in peppermint, cinnamon, or chloroform water. Along with laxatives it is usual in cases of chronic eczema to prescribe acetate of potash and other diuretics. Their action is somewhat uncertain, more so than digitalis, squill, or the resin of copaiba; but salines, and especially those of potash, have other actions beside that upon the kidneys, and in ordinary cases of eczema with much secretion and extensive inflammation, citrate of potash or acetate of potash is often found more beneficial as well as more agreeable than the alkaline carbonates.

We have seen above how important a point it is to relieve the itching of eczema, not only for the comfort of the patient, but to secure the physiological rest which the night should bring to all inflammatory processes, and also to save him from the serious aggravation of his disease which scratching and rubbing the eczematous parts infallibly causes. Moreover, it is at night (even during sleep) that the irritation, increased by the warmth of the bed, reaches its maximum, and that the self-control of the patient is weakened or abolished. Beside the various measures above mentioned for securing coolness, protection from the air, and such help as local sedatives can give, it is often necessary to call in the aid of internal narcotics. Of these opium and its preparations should generally be avoided. Unless given in large doses they are apt to increase rather than to quell the irritation of the surface; they also check secretion and bind up the bowels. It is therefore better to prescribe chloral hydrate or bromide of potassium, or the two together.

Chloral should be avoided with old people, and with patients who may have disease of the heart or atheromatous arteries. On the other hand, it is extremely well adapted to young children, and I have found a dose of syrup of chloral the most harmless and useful sedative in the case of infantile eczema. The safest plan is to give a moderate dose when the child is put to bed, and repeat it towards midnight, and, if necessary, again towards morning. Fifteen or twenty drops (two to three grains) may be given with perfect safety to a child of six months old; half a drachm twice or even thrice repeated in the night may if necessary be given to a child of twelve or eighteen months; and after infancy, say from two to five or six years old, half a drachm, or for older children a drachm of the syrup may be given at

bedtime with safety. Again and again this treatment has been followed by the best results. In the first place the child gets rest, and in the morning is ready for food, and all its organs have profited by the natural refreshment of the night. Next, the skin has been free from fresh irritation, and instead of being marked with the little sufferer's nails, is paler and less angry than the night before. All the processes of repair have had opportunity to go on; the habit of pruritus is broken for the time, and the nervous apparatus concerned has escaped from a vicious circle of inflammation, itching, scratching, and increased irritation.

The bromides are unsuitable to infants from their bulk and disagreeable saline taste, but with older children five or ten grains of the bromide of potassium may be sometimes added with advantage to the chloral draught if suitably covered with syrup of lemon or orange. With adults nocturnal irritation is not usually so severe as with children, and a draught of bromide of potassium or ammonium, with or without the addition of chloral hydrate, is usually sufficient when a sedative is required. Fifteen or twenty grains of ammonium bromide, with ten of the potassium salt, and twenty drops of aromatic spirits of ammonia in an ounce of camphor water, forms an effectual and not unpleasant sleeping draught.

In some cases, especially in old persons, neither bromide, nor chloral, nor a combination of them acts well. Henbane may then be prescribed with advantage, but in doses of not less than a drachm of the tincture, either alone or with a little compound tincture of chloroform in camphor water, or the tincture of hyoscyamus may be combined with that of hop. Indeed, in old persons where the irritation is not severe, and the want of sleep is rather dependent on general conditions of their age than upon pain or pruritus, two to four drachms of tincturi lupuli is a pleasant form of sedative, and may take the place of the whisky and water which the ascetic habits of the patient may render distasteful, or which habits of the opposite kind may render too agreeable. In the happily rare cases of severe and intractable pruriginous eczema in aged persons we are sometimes compelled to resort to continual doses of opium as the only means of obtaining rest.

The exhibition of antimony has been recommended in the acuter forms of eczema, and in the writer's experience has sometimes proved useful by subduing vascular excitement and hastening the passage of the acute into the chronic stage of the disease.

In many cases of eczema, especially in children, the patient is thin and pale, with a poor appetite, and a frequent and feeble pulse. It is in these cases that a little wine or malt liquor is not only admissible but often extremely useful; and here it is that the exhibition of iron finds its proper place. In the case of anæmic women with constipation the best combination is that of sulphates of magnesia and iron. For children the syrup of the phosphate, the saccharine carbonate, or the citrate of iron and quinine, are all valuable remedies. For infants steel wine is also a popular and valuable remedy, but after eight or ten years old effectual doses are too large to be convenient. In cases of great anæmia in children, especially where there is diarrhœa, no preparation of iron is so useful as the tinct. ferri perchlor., guarded, if necessary, by twice the number of drops of glycerine, or its taste concealed by a little syrup. It would almost seem as if the astringent quality of the drug had an effect upon the profuse secretion of the eczema. The result, at all events, is often striking as well as beneficial.

With the exception of iron the group of so-called tonics are not generally

indicated in the treatment of eczema. Quinine, however, has a very distinct effect, particularly in the case of infants, children, and persons below adult age, in preventing itching. Half a grain of sulphate of quinine may be given for this purpose to a child a year old an hour before bedtime, a grain if a year older, and as much as five grains to a boy or girl of fifteen. This effect of quinine was well known to the lamented author of this work, and it has been independently and strongly recommended by Dr Eustace Smith in his work on 'Disease in Children.' This writer also recommends guaiacum in the treatment of eczema, especially where there is reason to suspect a disposition to gout.

There remains a drug which, in England especially, has been very largely and often far too indiscriminately used in the treatment of eczema, as of all other diseases of the skin, namely, arsenic. It is undoubtedly a therapeutical agent of the utmost value in psoriasis, in pemphigus, and in certain other cutaneous diseases to be afterwards described, and no one of experience can doubt its efficacy in certain cases of chronic deforming arthritis, neuralgia, idiopathic anæmia, leuchæmia, and anæmia lymphatica; but, like all powerful medicines, it is powerful for evil as well as for good. In the acute stages of eczema, in most cases where there is extensive and active inflammation, and in most cases accompanied by severe pruritus, arsenic is decidedly injurious. In other cases, however, its success is so marked that in spite of its frequent failures it has never lost a certain reputation in the treatment of eczema.

The first indication for the exhibition of arsenic is that the eczema must be in a chronic condition—the greatest benefit is obtained in cases which have persisted for years. Secondly, the more dry and scaly the surface, the more infiltrated and indurated the skin, the less there is of active inflammation and the less disturbance of the stomach and intestines, the more likely is arsenic to be beneficial. As a rule, children with eczema do not need it, but some of the most striking instances of its value are in very obstinate and long-continued cases in young patients. One was a boy of fourteen, who from five years old had been the subject of what by his own and his mother's testimony was really uninterrupted eczema, spreading from time to time with excessive violence from its favourite seats over almost the whole body, but never absent from the scalp, the ears, and the limbs. When he was taken into the hospital there was dry scaly eczema of the head, face, and neck, and the hair was very thin. There was eczema rimosum of the ears and axillæ, papular dermatitis of the arms and back, and eczema rubrum madidans of the abdomen, genitals, perinæum, nates, and thighs. The only parts of the whole surface free from the disease were the palms, the soles, and the shoulder. He was thin, worn, and miserable, and the whole skin was so deeply pigmented that he looked like a mulatto, but the urine was perfectly healthy, and he had no other disease than this severe dermatitis. He was carefully treated with zinc and lead ointment and unguentum metallorum as he had been before while an out-patient, but he was also given arsenic in steadily increasing doses from three drops up to fifteen three times a day. Under this treatment the inflammation gradually subsided, and at the end of five weeks it was reduced to a little ordinary eczema of the arms. This also gradually disappeared. Meantime he had also grown into a stout, healthy-looking lad. He has from time to time come to me again with slight return of eczema, chiefly in the scalp and arms, but it has never in the least approached its former severity, and the

skin generally, instead of being thick, rough, hard, and infiltrated, with almost entire absence of subcutaneous fat, is now smooth, soft, plump, and elastic, while his head is covered with a thick growth of hair.

In prescribing arsenic the following rules will be found useful:—To begin with a small dose, and gradually but steadily increase it until either obvious benefit results, or the physiological action of the drug is shown by itching of the eyes or slight nausea. When these occur the dose should be at once stopped, and then resumed in somewhat smaller proportion, and if necessary again cautiously increased. Secondly, it should always be given either with or immediately after food and sufficiently diluted with water. There appears to be no advantage in any other form of the drug over Fowler's solution. The arseniate of soda may be given in somewhat larger doses, but is probably converted into the same form during digestion. The liq. arsenici hydrochloricus is useful if we wish to combine it with perchloride of iron. The "Asiatic pills" of Vienna are in every respect less eligible.

Troublesome and difficult to treat as many cases of eczema are, sometimes rebellious to the very treatment which in apparently similar cases has proved effectual, and always liable to relapses which are most trying both to patient and physician, it is nevertheless very rare for us to fail in at least relieving the miseries of an attack, and in a great majority of cases we may be fairly said to cure a disease which, without skilled treatment, would linger on almost indefinitely. Hebra concludes one of the most masterly and original chapters in his great work by saying that he who having once decided upon his plan of treatment, follows it out with patience and determination, will attain his object sooner than he who often changes the measures that he uses. We may venture to add that while keeping steadily in view the broad principles of treatment based upon rational pathology and tested by experience, the most successful practitioner will be he who knows how to vary their application in accordance with the perpetually varying needs of each individual patient.

There are, however, certain practical points in the treatment of local varieties of ordinary eczema which must be briefly mentioned.

Eczema of the ears is one of the commonest local forms of eczema and is sometimes extremely troublesome. Ointments will be found almost always to suit better than lotions—lead, zinc, or equal parts of the two, or in some cases weak carbolic oil, 1 in 40. When extremely moist, powders suspended in thin gum are better than dry powders, which are almost sure to form thick crusts and produce bleeding.

Chronic *eczema of the meatus* may cause deafness by swelling or the accumulation of its products. This must be treated by syringing with soap and water and, if necessary, application of an alkaline wash followed by unguentum plumbi or unguentum metallorum made soft by an equal part of carbolic oil.

Eczema of the scalp is complicated by the presence of hair and of sebaceous secretion and is apt to become more or less pustular. The hair should always be kept short, but shaving is unnecessary. Unguentum metallorum is commonly a good application. In the drier form of eczema of the scalp with scarcely any exudation, which is often combined with seborrhœa sicca under the name of pityriasis capitis, tarry applications are most efficient, and none is better than liquor carbonis detergens, either diluted to form a lotion or, as I have found better, with vaseline in the pro-

portion of a drachm or half a drachm to the ounce. Impetigo capitis will be presently considered separately.

Eczema of the eyelids and adjacent parts is apt to cause considerable inflammatory œdema which resembles erysipelas, but the colour, undefined edge, and the absence of marked febrile symptoms, together with the almost certain presence of ordinary eczema in other parts, distinguish the two.

Eczema of the lips is sometimes confined to that part and has then a peculiar aspect, there being very little serous or purulent secretion, great swelling, deep cracks, thin scabs, and considerable hæmorrhage. When chronic, large thin scales, partly epithelial and partly dry secretion, are formed which have led to its being called psoriasis labialis. The difficulty is to keep the parts from movement. Very mild ointments, vaseline with zinc, yellow oxide of mercury, or honey and borax will be found useful. Deep and painful fissures should be touched with nitrate of silver, either in strong solution or (what is less painful) with a pointed pencil.

Eczema of the palms is usually bilateral and confined to these parts, or it may persist here after it has disappeared from the rest of the body. As above mentioned, it is often directly dependent upon irritants. Having made sure that the case is not one of syphilis, the first and essential point of treatment is to protect the hand from contact with all other irritants, and especially with soap and water. For this purpose scabs, scales, and crusts should be carefully removed with sweet oil, or if necessary by poulticing. The cleansed surface should then be anointed with unguentum metallorum, and thin rags covered with the same ointment should be closely applied to each affected part. A well-fitting thin kid glove should then be worn over the whole, and the dressings should be changed night and morning only. At the end of a week the improvements will generally be striking, or if not it will be due to some neglect of the patient in uncovering his hands or in washing them. If the parts are very irritable it is better to use diluted white precipitate or yellow oxide ointment, or occasionally unmedicated vaseline will be most effectual of all. In chronic indolent cases, on the other hand, a little of the red oxide ointment will often stimulate most usefully. If there is great accumulation of epidermis it must be removed with soft soap or Hebra's diachylon ointment. Deep and painful fissures should be touched at once with lunar caustic.

Chronic *eczema of the sole* is not nearly so common. It must be carefully distinguished from a syphiloderm of those parts by the greater pain, deeper fissures and more exclusive range. Sometimes it is accompanied by enormous hypertrophy of the epidermis, with deep bleeding cracks and horrible odour (p. 878). Such cases may be cured by the application of salicylic acid in an ointment (ʒss ad ʒj) until the horny masses are removed, and then assiduous treatment with the *empl. plumbi.*

Eczema of the matrix of the nail is still more local and residual than eczema of the palms. It is comparatively rare, as a complication of ordinary eczema, and a precisely similar inflammation of the matrix of several nails is sometimes seen where there is no other evidence of its eczematous character. The ill-formed nail may be scraped, but its removal is unnecessary and useless. The grooves around it should be carefully anointed with some form of mercurial ointment.

Eczema of the nipple has been observed to lead to carcinoma, and if for this reason only should be cured as quickly as possible. It often spreads to the whole breast quite independently of intertrigo of the lower part, and is

sometimes most difficult to heal. In obstinate cases the application of liquor potassæ has been recommended.

Eczema of the anus, perinæum, and genitals is sometimes confined to the immediate neighbourhood of the rectum. This eczema ani, prurigo podicis of Willan, lichen podicis of Hardy is, as these names imply, most frequently dry and papular and is apt to be intolerably itching; the irritation is sometimes most severe, especially while in bed, while the disturbance of the rest, and the remarkable effect of mental depression which is common to most of the disorders of this region, make it sometimes a truly miserable complaint. French writers describe it as sometimes associated with a profuse and almost paroxysmal discharge of mucus from the rectum. This form of eczema is most common in elderly persons and is often associated with portal congestion, hæmorrhoids and the hepatic dyspepsia described in a previous chapter. In children it most commonly depends upon the presence of thread-worms. Occasionally it is started by fissure of the anus, and disappears when this has been cured by division of the sphincter.

The scrotum and penis are frequently the seat of eczema, most often of the weeping form: eczema vulvæ closely resembles eczema ani in its symptoms and, like it, most frequently affects persons beyond middle life. It is sometimes associated with, and probably dependent upon, diabetes, and sometimes appears clearly due to inflammation, new growths, or degenerative changes in the uterus or bladder. Eczema of the anus, perinæum, or genitals often proves very rebellious and leads to great thickening and induration of the parts affected. Borax lotion or lead ointment, according to the degree of moisture, relieve perhaps more frequently than other applications, but this is one of the forms in which one must be content with tentative measures in each patient. In some obstinate cases a drying lotion of nitrate of silver proves effectual when other means fail.

For the *intertrigo* of infants, finely-powdered starch and oxide of zinc or chalk is the best application. When it affects the fold of the nates in adults it is better treated by extreme cleanliness and the application of vaseline or diluted white precipitate ointment. Glycerine to most skins proves an irritant rather than a healer.

Eczema of *the legs* due to *varicose veins* must be treated like varicose ulcers, by elevation and bandaging. An old-fashioned flannel bandage often proves a cheap and efficient method. Martin's elastic bandage often produces the most valuable results, but in wearing it or an elastic stocking care should be taken that the pressure is not too great.

So-called *eczema marginatum* of the thighs is essentially a form of ringworm, and will be described under parasitic diseases.

Pustular Dermatitis.—*Impetigo capitis, Pustular eczema of the scalp*—is one of the most frequent diseases in children. It was known to our forefathers as "scald-head," but has happily become far less common than it was when children's heads were more neglected than at present, and especially when the bad habit prevailed of covering the scalp with caps and linen hoods, indoors as well as out, by night as well as by day. Perhaps the majority of cases are due to the irritation of pediculi capitis, but there remain a large number where no such cause can be found, and where a similar eruption upon the face or other parts establishes its independent character. There is no doubt that we are right pathologically in counting these forms of dermatitis as belonging to eczema. They are superficial and

never leave scars, they are often associated with ordinary characteristic eczema of the ears, the limbs, or the trunk, and the same child may be affected at one time with what will be called impetigo of the face or scalp, and at another with eczema of the same parts; or in an infant with ordinary eczema of the scalp the dermatitis will be seen to become more pustular as the hair grows thicker over the head until it has assumed all the characters of the porrigo favosa of older writers. Nor does the fact that this dermatitis of the scalp is often dependent upon dirt, lice, and other irritants, prevent our regarding it as true eczema, if the principles laid down on p. 873 are correct. That something beside a traumatic cause, an *irritabile* as well as an *irritans*, is necessary for the production of impetigo, is proved by the fact that some children and most adults may have pediculi capitis for many years, and may even suffer from the irritation and yet be free from impetigo.

We have already mentioned the best treatment for impetigo of the scalp when associated with ordinary eczema. The children who are the subjects of it are often rosy, plump, and in every way healthy, though here as in other cases it is necessary to judge by the trunk and limbs as well as by the face. If, notwithstanding fat cheeks and ruddy complexion, the child is found to have flat shoulders and nates, thin arms and thighs, apparently disproportioned knees, and ill-developed pectoral muscles, his impetigo should be treated not only with equal parts of ung. zinci and ung. plumb. acet. or ung. metallorum, but also by careful attention to diet, by Gregory's powder, with or without a little grey powder, and when the digestive disorder is corrected, by cod-liver oil.

Impetigo affecting the scalp or face alone without ordinary eczema, and in a healthy child, is happily not difficult of cure. Indeed, apart from the purely pustular secretion, from the eruption being discrete and with a defined margin, and from the absence of severe itching, these typical cases of impetigo are separated from eczema by the fact that they are not prone to recur. Zinc ointment is the popular remedy for the eruption, but its efficacy is much increased by the addition of equal parts of white precipitate ointment or by the substitution of unguentum metallorum. The hair should be cut short, but there is no need to shave it, and the parents may be assured that it will grow all the better afterwards. It is only in extremely rare cases after the inflammation has penetrated to the hair-sacs, owing to a deeper suppuration of the scalp from too strong local irritants, that the hair-sacs are destroyed, and a bald cicatricial patch results. Such an event is more often seen from impetigo of the scalp in an adult than in the far commoner cases in children. When, as is usually the case, the scabs are thick and massive they should be removed first by poulticing. In circumscribed cases the bread and water poultice may be used, but where the whole scalp is covered it should be anointed with linseed oil and a large linseed poultice be then applied. For circumscribed and strongly adherent crusts, soft soap or even liquor potassæ may be necessary. Great patience and gentleness should be used in removing the scabs, or the child will suffer considerable pain, and the cure will be retarded.

Impetigo a pediculis.—In all cases of pustular inflammation of the scalp the hair should be carefully searched for pediculi. Equally decisive of the cause is the discovery of the nits, which consist of small triangular cases containing the eggs made of hard material, in colour and consistence like dried size, adhering to the hairs by one side of the triangle, and visible to

the naked eye. The impetigo which results from their presence is produced more by the scratching of the patient than by the irritation of the lice. It affects the back of the scalp chiefly or exclusively, and is attended with great consecutive swelling of the posterior cervical lymph-glands. Indeed, occipital impetigo is almost synonymous with impetigo a pediculis. The treatment is decisive and efficient. In bad cases, the whole of the long, tangled, and filthy elf-locks should be cut off, and the head washed with soap and water; but in slighter cases it is not necessary to cut the hair at all. The noxious insects are readily destroyed by mercurial washes, but an equally efficient and harmless remedy is the stavesacre ointment (℥ij ad ℥j). Common petroleum oil is also a cheap and efficient parasiticide, or if the hair is cut short, as is much the best plan, in hospital practice the white precipitate ointment which cures the disease will also kill the vermin. The egg cases are less easily attacked, and might be sources of future trouble. They must be either combed off, or removed with spirits of wine, or cut off, hair and all. The impetigo which results from pediculi will sometimes heal spontaneously as soon as they are removed, but usually unguentum metallorum, or white precipitate ointment, diluted to 1 in 3, hastens recovery.

Contagious porrigo.—This form of impetigo of the scalp has been separately described, and some authors have laid much stress on its distinction both from pustular eczema and from impetigo from pediculi, but no sharp line can be drawn. All impetigo is more or less traumatic and more or less eczematous, and all impetigo is more or less contagious. Nor is it in the scalp alone that contagious pus is secreted. The most virulent of all is the pus of a gonorrhœa; but no one can doubt that even it is of varying degrees of activity, when we consider the frequency of the urethral inflammation compared with the comparative rarity of gonorrhœal ophthalmia. Leucorrhœa is supposed as a rule to be non-contagious, and no doubt with justice, but the most experienced surgeons admit the possibility of infection from an idiopathic and apparently innocent discharge. Again, the pus of boils is extremely contagious, and is often the source of what is called ecthyma. The pus of scabies, too, is contagious. So also a whitlow on a child's finger may cause by contagion impetigo of the hand, of the nates, and sometimes of the scalp. Impetigo *a pediculis* often secretes pus of a most actively contagious kind, the proof being not only in the outbreak of similar pustules on other parts of the child's body, especially the fingers and the buttocks, but also in the spread of the disease to other children of the same household. Most cases of impetigo are only slightly if at all infectious, whereas in others a whole family or a whole street may be infected from a single case. It is said that the most contagious forms of impetigo are characterised by thick yellow scabs, by a sharp line of demarcation, and by readiness of cure by local means, as well as by absence of itching, by restriction to the scalp and face, and by being practically confined to children; but these are only the characters of impetigo generally, as distinct from ordinary eczema.

PAPULAR FORMS OF CHRONIC SUPERFICIAL DERMATITIS

(*PIMPLY TETTERS*)

Definition—Relation to common dermatitis and to eczema.
LICHEN.—*Its traditional species—Strophulus—Lichen scrofulosus—Lichen planus.*
PRURIGO.—*Pruritus—Summer and winter prurigo—Prurigo senilis a pediculis—Idiopathic prurigo: Hebra's and milder forms—characters and treatment.*

THE group of diseases to be now described is far from being as natural and well defined as one could wish. They agree with eczema in being inflammatory, in beginning as papules, in affecting only the epidermis and the papillary layer of the cutis, so that they never leave scars, in their esssentially chronic course, in the itching rather than pain that they produce, and in their persistence and liability to recur.

But they differ in the following important points :—(1) The inflammation never goes on to the stage of exudation either of serum or of pus; they are all "dry tetters." Even when, as the result of scratching, common inflammatory exudation follows, the pustules or raws thus produced are limited by the cause and do not assume the form of eczema or impetigo. (2) They are much less symmetrical, and rather avoid than choose the favourite places of eczema. Their locality may be said to be undefined, and widely diffused, but they affect rather the trunk and the outer side of the limbs than their flexures.

LICHEN.—Many dermatologists express by this term a papular dermatitis which by subsequent more or less free exudation of moisture, or by its symmetry, or by its association with previous or later attacks of ordinary eczema, proves itself to be more properly termed papular or *abortive eczema.* Its pathology, natural history, prognosis, and principles of treatment are precisely those of the drier forms of eczema. It most often affects the arms and legs, and the extensor rather than the flexor aspect.*

Lichen circumscriptus† was the name given by Willan and Bateman to a peculiar and characteristic form of papular dermatitis. It occurs upon the trunk, usually between the shoulders, but may spread over great part of the back, or may affect the chest or abdomen. The papules are small, red, and arranged in patches with somewhat well-defined margins. It is not very irritable, and rarely, if ever, ends in ordinary moist eczema.

Many cases which have been described under this head are probably nothing but papular dermatitis, or local eczema, depending upon the irritation of decomposing sweat. The locality between the scapulæ and on the front of

* Bazin divided lichen, according to its origin, into parasitic, dartrous, herpetic, scrofulous, and syphilitic,—an excellent example of his classification and pathology.

† *Synonyms.*—Lichen annulatus (Wilson)—L. marginatus (Liveing)—L. circinatus—Lichen acnéique—Eczema flavum—Seborrhœa of the trunk (Duhring).

the chest is just where sweat accumulates; the eruption is most common in summer, and in persons who sweat freely; moreover, with the papules true vesicular sudamina may often be detected. But apart from this, it must be admitted that there is a distinct, though somewhat rare, circumscribed papular dermatitis, which, from the shape of its patches and from their spreading at the edge while the centre returns to its natural condition, reminds one of spots of tinea. Parasitic fungi are apt to occur in the locality and under the conditions named, as in tinea versicolor, but the affection under consideration is certainly distinct from tinea versicolor.

Dr Payne has pointed out that it is usually associated with wearing thick woollen vests, often night as well as day, and calls it flannel-rash.*

The areæ of the circles present a yellowish tint (*Eczema flavum*), and are sometimes covered by branny desquamation. When several circles combine they form irregular lines, and the eruption thus formed was called *Lichen gyratus* by Biett and Cazenave. Two of Mr Towne's models in the Guy's Hospital museum (Nos. 257, 258) show this affection perfectly.

This affection may be frequently seen, and always in young men or young women, never in children or persons over fifty. They are usually cleanly, and, in fact, seen almost as common in private practice as at the hospital. Since the eruption generally does not itch much, and is most marked where it is not seen, one often comes upon it accidentally when examining the chest. In every case the patient has been wearing a thick woollen jersey next the skin, and almost always another or even the same has been worn at night.

* The following are Dr Payne's observations:—"In Dr Colcott Fox's interesting remarks (Jan. 8th) on a peculiar form of lichen, called L. annulatus by Erasmus Wilson, it is not stated that this is really the disease called lichen circumscriptus by Willan and Bateman, and figured by the former in 1808, so that his name clearly has the priority. It has also been called L. circinatus and L. marginatus, and, as such, is described by Dr Liveing. For so unimportant a disease, the number of synonyms it has received is remarkable; it being also known as lichen acnéique, eczema flavum, &c. Unna, Fagge, and Pye-Smith regard it as an eczema. The Americans, as Dr Fox points out, have called it seborrhœa.

"Which of the three names—lichen, eczema, seborrhœa—should be retained? I have described several cases in the 'St Thomas's Hospital Reports,' 1884 and 1885, and have suggested that, as the disease is not really either lichen or eczema, a neutral name, such as circinaria, which does not beg the question, would be preferable.

"With regard to the seborrhœa question I would beg to remark (1) that this affection is certainly often associated with seborrhœa of the scalp, as I have pointed out in the 'St Thomas's Hospital Reports;' (2) that minute examination undoubtedly shows that the starting-point of each so-called papule is a sebaceous gland; (3) but that it is not accurately described as merely seborrhœa or excessive secretion. The bright red colour of the papules or margins of the patches, which strikes every observer, depends not only on hyperæmia but on dilatation and elongation of the capillary vessels, which project above the skin level, as in psoriasis. Hence it is that slight scratching causes hæmorrhage. This is something more than over-secretion.

"Dr Colcott Fox has not mentioned a condition which I believe to be constant in this disease—namely, the wearing of thick woollen under-vests, especially flannel, and generally the same day and night. Hence the name 'flannel rash,' which has long been used at the Hospital for Diseases of the Skin. I have failed to establish this connection only in a few cases, in which the eruption existed on the limbs as well as the body, and which I now believe to belong to a different disease, apparently identical with pityriasis rosea of Gilbert.

"This curious affection is commoner than might be supposed. A good many of my cases came before me in the general out-patient department, when they applied for some other disease, and the eruption was discovered on stripping for auscultation.

"No one can help suspecting that it is a parasitic disease; and like Dr C. Fox, I have examined a large number of cases without finding any distinctive parasite. Single or scattered spores of fungi are, I may say, not uncommonly found in epidermic scales from any kind of diseased or even healthy skin, and so are micrococci."—('Brit. Med. Journ.,' Jan. 22nd, 1887.)

When this is corrected, soap and water and a little liq. carbonis detergens as a lotion (1 in 10), or an ointment (ʒij—ℨj), is speedily effectual in curing the disorder.

The form of lichen described by Wilson, and also by Hardy (*lichen circonscrit*), affecting the extensor aspect of the forearm and the back of the hands, and running an acute course, should probably be regarded as eczema papulatum.

Lichen tropicus.—The writer has seen only three or four cases of this curious affection, so well known in the East and West Indies under the name of *prickly heat.* It occurs also in Australia and on the West Coast of Africa. Its characters are the sudden appearance of the eruption, its almost universal distribution, and the intense irritation it produces. After once attacking a patient it is apt to return with each hot season, and though usually cured by a temperate climate sometimes comes before us in England. In the cases referred to the eruption has been entirely papular, with no other lesion but scratch-marks or occasional wheals. The parts most affected were the abdomen, buttocks, and thighs. The face and scalp, the hands and feet, and the genital organs seem to be usually free. One must speak doubtfully about a disease of which the personal experience of an English physician is small; but from its acute character, the absence of moisture, and its un-eczematous distribution, it is at present best classed as a form of papular dermatitis. The small red papules are frequently associated with sudamina.

It was once supposed, of prickly heat as of other eruptions of the skin, that driving it in by a cold bath was extremely dangerous; but more than fifty years ago Dr James Johnson, who gave a graphic account of it in his own person, justly ridiculed this superstition.

The late Dr Tilbury Fox regarded this disease as essentially an *adenitis* of the sweat-glands, the direct result of excessive heat and perspiration, and Dr Duhring, of Philadelphia, in his excellent text-book of dermatology, calls it *miliaria papulosa* on the same theory.

The *Lichen agrius* of Willan is, from its acute course and the presence of small vesicles filled with a straw-coloured fluid, clearly a papular eczema.

Lichen pilaris was a name applied by Willan to a familiar condition which is, however, not a dermatitis at all. The hair-sacs of the affected part of the skin become filled up with horny cuticle, which forms rough papular projections, hard, pointed, and very characteristic, both in appearance and feeling. They do not occur in places where the hair is long, but are almost exclusively confined to the outer side of the limbs, over the vastus externus most often, but not uncommonly more or less developed on the outside of both arms and legs, on the buttocks, and the shoulders. The condition is most common in the brawny skin of muscular working men, and may be readily removed by soap and water and friction. Occasionally it may be seen on the limbs of delicate children, in girls of only seven or eight years old. This affection was described by Devergie under the equally inappropriate term of *Pityriasis pilaris.* It may be better called *Keratosis pilaris.* Dr Fagge proposed for it the name *Rhinoderma*, from ῥίνη, a file ('Guy's Hospital Reports'); but as he himself says, this term has such obvious disadvantages that he prefers Devergie's title.

Occasionally keratosis pilaris is complicated by local inflammation and large flat red papules, or pustules may result, each surrounding a minute hair. These cases have been called sycosis of the trunk or limbs. Several examples were shown at the Dermatological Society in 1885–86.

Willan's *Lichen lividus* is purpura, the petechial spot being perforated by a minute hair.

Lichen urticatus (Bateman) seems to be nothing but papular erythema combined with urticaria. It corresponds to much of what is now called prurigo infantilis and prurigo æstivalis.

Lichen hypertrophicus is a name given by French writers to what is probably in most cases identical with the rare affection to be described under the name of *Mycosis fungoides.*

The term STROPHULUS,* applied by Willan and Bateman to certain papular eruptions in infants, is now deservedly abandoned. Willan defines lichen as a papular eruption occurring in adults, so that the original distinction between the two diseases was merely one of age.

Green remarks that "strophulus differs from lichen in no essential particular, a circumstance that might warrant us in discussing the two diseases under one and the same head" ('Compendium of Diseases of the Skin,' 1836, p. 174). This author points out the difference in age of the patients, the more frequent intermissions of strophulus, and its milder character. Rayer regarded strophulus as infantile lichen, but Wilson described them separately. Most authors admit that the papules so closely resemble those of lichen as to appear identical with that disease. They are indeed only modified by the age of the subject.

Strophulus albidus it not dermatitis, but *milium*, a variety of comedo which will be mentioned among affections of the sebaceous glands.

Strophulus intertinctus and *S. confertus* may be called *infantile lichen* by those who keep to this name. They are papular dermatitis of more or less acute form, and in most cases may fairly be termed eczema.

Strophulus volaticus, with its acute course and slight maculæ following the patches, is a typical form of *erythema papulatum.* Bazin and Hardy are unable to class these papular eruptions of infants among the chronic inflammations which they ascribe to the dartrous diathesis. The former writer places them under it among the scrofulides, the latter among what he ingeniously calls "Maladies cutanées accidentelles." The *Strophulus prurigineux* of these authors is identical with infantile prurigo.

Lichen scrofulosorum, or, as it is more conveniently called, *Lichen scrofulosus*, is a somewhat rare form of eruption, which was first accurately described by Hebra. He describes it as consisting of papules arranged in groups with some amount of pigment and slight desquamation, not itching, and lasting for a long time without change. It is almost always confined to the trunk, and in forty-five out of fifty of Hebra's original cases the patients had swollen lymph-glands, or chronic disease of the bones, or scrofulous ulcers, or were supposed, from a fulness of the abdomen, to be subjects of tabes mesenterica. On the other hand, in none of these cases was there evidence of phthisis. All Hebra's cases occurred in young men, the youngest patient being fifteen, and the eldest twenty-five. His description has been followed

* Strophulus.—This name, derived from *στρόφος*, a swaddling band, was apparently first used to describe any skin-eruption occurring in an infant. A popular English name is *red* or *white gum*, or *tooth-rash.* These names point to the popular explanation of all cutaneous rashes, and most other affections which occur during teething: but it is probable that originally "red gum" was only a corruption of another vernacular title—"red gown," a not inapt description of a child covered with general erythema; and this word *gown*, though in English meaningless without the prefix, is only a translation of *Strophulus.*

by subsequent German writers, who have added little to the account which he gives. The writer saw two of his cases in Vienna, and can testify that they were not, as has been naturally supposed by some writers, cases of pityriasis scrofulosorum sive tabescentium, the xerodermia or dry rough scaly condition of the skin not uncommon in phthisis and other wasting diseases.

Hans von Hebra calls it *scrofuloderma papulosum.* It does not appear under any form of Hardy's scrofulides. It might, however, be well included under Bazin's large group of scrofulides bénignes.

Kaposi has made sections of the affected integuments, and describes the sebaceous glands as blocked by epidermic plugs, and surrounded by a copious infiltration of the leucocytes, so that, according to this excellent observer, the disease would be a chronic inflammation of the corium surrounding the sebaceous glands. The late Dr Tilbury Fox transcribed Hebra's account without comment, only stating that the condition is "of infinitely rare occurrence in England." Dr Liveing has met with a few typical cases among poor out-patients, and thinks that the inconspicuous colour of the papules and the absence of itching leads to its being overlooked. Several well-marked cases have been lately shown before the Dermatological Society; one was a patient of Dr Payne's, a girl aged seven, pale, and with swollen glands. From English experience it is more common in children than in adults, and is as common in one sex as in the other. Its locality, the circumscribed patches, the pale colour of the papules, and the yellowish pigmentation, together with entire absence of itching, are sufficient characters for diagnosis, and justify its recognition as a distinct variety of chronic papular dermatitis.

The treatment consists in the internal administration of cod-liver oil, and is said to be uniformly successful.

*Lichen planus.**—There is a form of chronic superficial dermatitis which is so distinct from all others that it is well entitled to a separate name. Of all forms of papular dermatitis it recedes furthest from typical eczema, and approaches nearest to the dry tetter or psoriasis which will be described in the next chapter. It does not appear to have attracted the atttention of the older dermatologists, and is indeed a somewhat rare disease. It was first described under the title *lichen ruber* by Hebra, and shortly after from a different point of view by Erasmus Wilson under the more distinctive name which has been generally adopted in this country. Dr Unna, of Hamburg, has proposed to distinguish *lichen ruber acuminatus* and *lichen ruber planus,* recognising also a third variety, *lichen ruber obtusus.*

Hebra gives an elaborate table of the differences between lichen ruber and lichen scrofulosus, psoriasis, eczema, and pityriasis rubra. Some dermatologists question whether the disease described in Vienna is really the same as lichen planus, but in pathology and the essential points of their natural history the affections named by Hebra and by Wilson are one, although they represent two varieties which may be recognised.

Mr Hutchinson and Dr Liveing, as well as Dr Duhring in America, regard Hebra's and Wilson's disease as unquestionably the same. Wilson himself suggested that the cases described by him were varied examples of the lichen ruber of Hebra, but Hebra himself considered the two affections to be distinct, and this was also the opinion of Dr Fagge.

* *Syn.*—Lichen ruber—Lichen invétéré (Hardy), including Lichen plan corné (Vidal).

Hans von Hebra describes two forms: the first more acute, with greater formation of scales, more itching, and more generally diffused dermatitis, and also followed by more severe affection of the general health; the second more chronic, never spreading over the entire surface, with only slight irritation, and with no injurious effects on the health. This view of their relations is very much what the writer expressed in a paper in the 'Guy's Hospital Reports' (vol. xxv, p. 254).

Anatomy.—No one who has seen a well-marked example of this affection can doubt the accuracy of Wilson's description; the raised flat patches, their dull glistening surface, deep purple-red colour, slight desquamation, chronic course, and resulting pigmentation are together most characteristic. Hebra insists upon the genuine papular origin of the affection, on the deep red colour of the papules, and their not increasing in size when once formed; but fresh papules appear, so that at last they become confluent and form the raised flat patch which struck Wilson's attention (see model 260 in the Guy's museum).

Hillier in 1866, and Neumann more fully in 1869, described the histology of the disease. The hair-sacs and adjacent sebaceous glands are the chief and apparently earliest seat of infiltration. The opening of the hair-sacs is wide and funnel-shaped, a fact noted by Hebra in his original account. The cells of the rete mucosum contain granules of dark-brown pigment, the natural papillæ are enlarged, the sweat-glands are unaffected, the sebaceous glands atrophied. There is after a time considerable induration of the skin, as in other forms of chronic dermatitis.

Distribution.—Lichen planus may occur upon the extremities or trunk, but has never been observed upon the face or head. Hebra describes it as sometimes affecting the palms and soles, and this statement is confirmed by Wilson and by Hutchinson. The patches are apt to be most marked in parts subject to friction, as the waist and the circle of skin pressed on by a garter. Its favourite positions are the extensor surface of the arms and legs, especially the forearm and wrist (front as well as back), and the leg below the knee; also the thigh, and particularly the hollow over the great trochanter. In the lower limbs the colour is more deeply purple.

It is often symmetrical, but less decidedly so than psoriasis or eczema.

In cases which agree more nearly with Hebra's lichen ruber the papules are of a brighter colour, and more generally distributed over the limbs and trunk. The papules and raised patches are not arranged in groups, as in most forms of lichen.

Natural history.—Hebra's patients were almost all men. In England it has been more often seen in women. It seldom or never attacks young children, but the writer has seen a typical case in a girl of thirteen. Observers differ as to the existence of itching. In the cases on which the present account is founded it has once or twice been absent, sometimes troublesome, but never severe, that is, not comparable to the irritation of eczema, scabies, or prurigo. In extensive cases the nails may be affected, but this complication is less common than in chronic eczema.

Lichen planus is chronic in its development and course. Hebra describes lichen ruber as leading to marasmus and death. The late Dr Fox says that in both forms of the disease, the more general and severe of Hebra and the more local of Wilson, the general health is bad. Mr Hutchinson says that the large majority of patients believed themselves to be in their usual health when it began, but that if it persists long the general health

may fail. All the cases seen by the writer were in persons in average condition, some of them in robust health. No internal organ is affected, nor are there symptoms of general disturbance, at least in ordinary cases.

Mucous lesions.—Lichen ruber is undoubtedly often associated with the white patches on the tongue and cheeks, which have been described under the varied titles of ichthyosis linguæ, psoriasis linguæ, tylosis, keratosis, and leucoplakia. The association was noted in two of his cases by Mr Hutchinson ('Lectures on Clinical Surgery,' vol. i, pp. 211, 213), and we have seen it repeatedly at Guy's Hospital and elsewhere.

Diagnosis.—This affection is distinguished from eczema by its never forming either vesicular or raw surfaces, by its avoidance of the face and ears, its general distribution, and the comparatively slight amount of itching. In some cases it undoubtedly approaches very closely to psoriasis, especially to inveterate cases of the latter disease which have become generally diffused and have lost much of their characteristic appearance. Mr Hutchinson would recognise transition cases, and indeed proposes to name lichen planus "lichen-psoriasis." But difficult as the diagnosis occasionally is, the distribution, the character of the scales, and the persistence of papules sufficiently distinguish lichen planus from psoriasis.

A more important distinction is between lichen planus and syphilis, for which it is often mistaken. The colour, the frequent absence of itching, and the somewhat irregular distribution, lead to this error, which is apt to be confirmed if white patches are found on the tongue or cheeks. This leucoplakia, however, is no proof of syphilis. The colour of lichen ruber is more purple and less brown than that of a syphilide. The freedom of the face and scalp, the absolute uniformity of all the lesions, and their persistence unchanged during long periods of time, usually ensure a correct diagnosis.

Lichen planus does not tend to cure; it continues indefinitely, may spread extensively, and, as above stated, will in certain severe and protracted cases seriously affect the bodily health.

Treatment.—The treatment adopted, both in Germany and England, is the administration of arsenic. Most writers speak of this as specific and certain in its effects, but some of considerable experience find it occasionally fail, and lichen planus is certainly slower in yielding to the remedy than average cases of psoriasis. Locally, tar ointments or some of the milder preparations which will be described under psoriasis as substitutes for tar, are important aids in treatment. In obstinate cases an ointment of pyrogallic acid should be applied. Unna recommends an ointment composed of hydr. perchlor. gr. ½, ac. carbol. gr. xx, and ung. zinci ℥j.

PRURIGO.—Prurigo, "the disease attended with pruritus or itching," was a term formerly very loosely applied and is still somewhat difficult to define. Willan described it as a papular eruption in which the papules are of the same colour as the skin and accompanied by itching. His "species" were *P. mitis* and *P. formicans*, which are merely more or less severe cases of the same affection, and *P. senilis*, characterised by the age of the patients and the difficulty of cure. Bateman thinks that pediculi are not unfrequently generated when prurigo senilis is present, thus putting the cause for the effect, since it is now well ascertained that most if not all cases of prurigo senilis are directly caused by pediculi corporis.

Willan and Bateman also mention *Prurigo pubis*, which they rightly

ascribe to the presence of pediculi, and *P. præputii* and *urethralis*, which are both sympathetic pruritus. Lastly, their species, *prurigo podicis* and *P. pudendi*, correspond to the drier and more papular and indurated forms of the irritable local dermatitis which was described in the last chapter as eczema ani and eczema genitalium.

1. *Pruritus.*—Prurigo, a papular inflammation of the skin, must first be distinguished from pruritus or subjective sensation of itching without any local lesion. Pruritus accompanies not only prurigo but eczema and the desquamative stage of many exanthems. It is the constant effect of pediculi and of the acarus; it may be produced by jaundice, and it is also the result of the various atrophic changes which take place in the senile skin. These, which have been well described by Neumann, include the wasting and ultimate disappearance of the papillæ, and it is probable that the process gives rise to senile pruritus.

Prurigo, or irritable papules, may be produced by primary pruritus. This occurs in hot weather among children, most often from the irritation of sweat or of vermin. The papules are large, flat, and discrete, but there is no pigmentation, no thickening of the skin, and the distribution is irregular. Mr Hutchinson has observed this summer prurigo affecting the face and arms of adolescents and relapsing every year. According to this author another form of prurigo is common as a sequel of varicella.

Some persons, usually young adults, are liable to great irritation of the skin in cold, frosty weather, when it is dry, harsh and pale. This affects the covered parts most, and is often supposed to be due to flannel underclothing. It has been called *prurigo* (or pruritus) *hiemalis* by Duhring, but in England is more common during the east winds of spring than in mid-winter. The scratching of the patient produces a crop of papules.

Many cases formerly described as prurigo should be called papular erythema, or urticaria, or papular dermatitis from the irritation of bugs or pediculi (which in infants do not cause the characteristic appearance of prurigo pedicularis), or from the friction of flannel next the skin. Lichen circumscriptus, papular eczema, and even congenital syphilis have been mistaken for prurigo.

2. *Prurigo senilis a pediculis.*—This is a well-characterised and common disease known as prurigo senilis, phthiriasis, or prurigo pedicularis. It is a papular dermatitis of definite clinical characters dependent on the irritation of body-lice, and is only seen in elderly persons. It is a good example of the combination of two conditions—the excitant and the predisposing cause, the *irritans* and the *irritabile*—to form a constant clinical result.

Phthiriasis is not a sufficient title, for children may be swarming with vermin, and may suffer from urticaria or ecthyma as the result, but are never affected with this form of prurigo; nor is "prurigo senilis" enough unless we recognise the exciting cause of the disease.

The papules are large and separate, not spreading over wide surfaces as in eczema, nor collected in more or less rounded patches as in lichen circumscriptus, nor coalescing as in lichen planus. Moreover, they are much larger than in eczematous dermatitis, flat rather than pointed, less red, and more persistent. But what is most characteristic is that before long each of them is capped by a little black crust of dried-up blood, the result of scratching.

Beside these papules, the disease is marked by an extensive series of scratch marks following the curves which are described by the right or left

hand respectively, working from the shoulder. The irritation of scratching not only causes excoriation and hæmorrhage, but sometimes produces wheals like those of urticaria, and raw surfaces which may be properly called traumatic eczema. Both these effects may be absent, but prurigo senilis never lasts long without the whole surface between the papules becoming more or less deeply pigmented, until in some cases the affected parts are as dark as the skin of a mulatto.

The *distribution* of prurigo senilis is as characteristic as its anatomy. It occupies the shoulders, back, and loins, the papules usually stopping abruptly at the waist or the sacral region, and sometimes not spreading below the scapulæ. They may appear over the upper arms, but rarely below the elbow and never on the hand. They are numerous on the flanks, and in severe cases may cover the whole chest and abdomen. The thighs may share in the disease; but even in the most extensive cases it is generally found that the outlying parts are rather the seat of ordinary dermatitis produced by scratching than of the true papules of the disease. Prurigo senilis never affects the face.

The itching is most severe; and, like all pruritus, it is worst at night and when the patient is warm. The absence of pain and tenderness leads to more reckless scratching than in any other disease. It is the consequence, however, and not the cause of the papules, for we can distinguish the latter from the traumatic dermatitis set up by the former.

The exciting cause of the disease can be found when carefully looked for, especially in the plaits of the underlinen about the neck and waist. It is important to remember that pediculi corporis may exist in old men and women of apparently scrupulous cleanliness.

The whole facies of the disease is so well marked that it can scarcely escape recognition. It affects both sexes. Typical cases are very rare in persons as young as fifty.

The *treatment* is simple and effectual when the disease is once recognised. The most effectual parasiticide is the white precipitate ointment, and if only applied to the shoulders no harm will ensue; when used more freely and extensively it may cause salivation. Inunction, as used with grey ointment in cases of syphilis, is quite unnecessary; it is enough for the parts to be smeared over. The clothes must be scalded or fumigated.

3. *Prurigo*—in the restricted sense.—There remain certain forms of disease which are quite independent of pediculi, but agree with prurigo senilis in the anatomical character of the papules and in the excessive itching to which they give rise. One form is the prurigo of Hebra, a striking description of which is given in the Sydenham's Society's translation of his work (vol. ii, p. 258). He admits milder cases which correspond to the prurigo mitis and formicans of Willan, but would separate them broadly from the severe form which is congenital and incurable. The writer saw at Vienna cases of this "Hebra's prurigo," as it has been called, and ventures to think that their characters were somewhat over-described, if not exaggerated. At all events cases have been described, both in America and in England, which agree with Hebra's cases in all essential particulars, and cases in London which would make an uninterrupted series connecting the worst of those in Vienna with the slightest forms of infantile prurigo.* We may therefore fairly include these affections under a common name,

* See, on this point, a paper by Mr Morrant Baker, 'Internat. Med. Congr., 1881,' vol. iii, p. 177, and the discussion which followed.

using such adjectives as *mitis, gravis, agria, congenitalis, infantilis, inveterata,* to denote the varieties which we find in practice.

The papules of prurigo are at first scarcely distinguishable in colour, and, as Hebra says, are felt rather than seen. They are not closely set, and do not appear in patches, they produce great itching which causes black spots and scratch-marks as in prurigo pedicularis. The skin between is more or less pigmented, and is generally covered with a fine branny desquamation. In course of time it becomes thick and indurated and in many cases there is traumatic eczema, often of a pustular kind. In severe cases, inflammatory enlargement of the lymph-glands occurs both in the groin and the axillæ.

The distribution of prurigo is over the trunk and limbs. The face is almost always free, and also the flexures of the joints, palms, and soles. It is generally most severe on the back, chest, and abdomen, on the buttocks, the shoulders and upper arms, and it is generally worse on the lower than on the upper extremities, and worst of all below the knee.

Prurigo begins in early life and either disappears during childhood, or if present in an adult, has persisted from that period. It is generally worse in winter. In one exceptional case it began in a lad at the age of fourteen. It first appeared on his legs, and affected the whole surface except the head, palms, soles and flexures. There were a few spots on the cheeks and neck, on the hands and penis; the trunk was moderately affected, the buttocks and thighs more so, and the arms and legs most of all. There were severe buboes, and he was thin and wasted. He improved greatly under treatment, but the disease returned from time to time.

In twenty consecutive cases observed, the ages of the patients when first seen by the writer were: under twelve months two, between two and five years eight, between five and ten one, between ten and fifteen five, between twenty and forty-five three. It is more common in men and boys than in females. In long-standing cases the skin is often much thickened as well as pigmented.

The *treatment* of prurigo, even in its most typical and severe form, is far from being as hopeless as Hebra supposed. Frequent warm baths and assiduous inunction, together with arsenic internally in steadily increasing doses, with cod-liver oil and good feeding, will restore inveterate cases to health and comfort. It is, however, almost certain to return, probably more than once, and must be kept at bay for years before it finally disappears. The slighter forms of true prurigo in infants and children are very much aggravated by scratching, and the first point is to prevent this by hydrocyanic lotion or other local anodyne, and by sedatives at night as described under eczema (p. 889). In some cases quinine appears to be almost a specific, both for the irritation and the disease.

PITYRIASIS RUBRA

(*GENERAL EXFOLIATIVE DERMATITIS*)

Name—History—Accounts by Devergie, Hebra, Wilson, Hutchinson, Baxter—Course and symptoms—Histology—Prognosis—Diagnosis—Treatment.

THE word "Pityriasis," meaning, as its etymology implies, a branny or furfuraceous desquamation of the skin, is conveniently used to describe that pathological condition, but no one *disease* is entitled to the name.

The species defined by Bateman as *Pityriasis capitis* is in most cases *Seborrhœa sicca*, an affection of the sebaceous glands of the scalp; or it may be slight local dermatitis (*Eczema capitis*) due, as he remarks, to want of cleanliness and removable by soap and water, but apt, if neglected, to degenerate into "Porrigo," that is to become pustular eczema or impetigo of the scalp.

Pityriasis versicolor, now known as tinea versicolor, is a parasitic disease.

Pityriasis nigra, described by Willan as occurring in children born in India, was not identified by Bateman, nor probably by anyone else. A case of Alibert's which Devergie calls "pityriasis nigra with prurigo," was apparently *Prurigo pedicularis* with pigmentation and leucodermia.

Bateman's fourth species, *Pityriasis rubra*, "resembling psoriasis diffusa," is a stage in the involution of eczema. The case described as pityriasis rubra by Cazenave seems to have been *Tinea versicolor*, with more irritation than usual.

But this same term, "pityriasis rubra," was unluckily applied by Devergie in 1854 to a very remarkable form of superficial dermatitis which certainly deserves a special name. It is probably identical with Alibert's "Herpes squamosus." Hebra in 1860 thought himself bound to follow Devergie's nomenclature, and his authority has made it generally accepted. Wilson's proposed names of "Pityriasis foliacea rubra" or "Eczema foliaceum" (1867), or the better title, "Exfoliative dermatitis" (1870), have not displaced the original term. "Universal exfoliative or desquamative" dermatitis is perhaps the title that would most clearly express its characters.

Devergie ('Traité Pratique des Maladies de la Peau,' p. 263) placed it next to eczema. He describes the disease as beginning with an erythematous redness, usually on the chest or flexor surface of the limbs, and spreading rapidly, with a well-defined margin, deep colour, abundant scales, and more or less thin serous discharge. It covers the whole body, is very obstinate, lasting for months, and occasionally proves fatal by exhaustion and diarrhœa. As a rule, however, patients slowly recover. Relapses are frequent. Devergie admits the difficulty of distinguishing this new disease from eczema, and bases the diagnosis on the following points:—It is of a deep red colour, it has sharply marked borders, it may affect the whole skin; the skin, and even the subcutaneous fascia, are thickened; it is less itching, more burning than eczema its secretion is thin, and does not stiffen linen; the scales

are abundant, readily detached; and from the first no red moist points (*état ponctué*) are seen when the scales are removed.

In the 'Glasgow Medical Journal' for January, 1858, p. 421, Dr McGhie recorded a case of "pityriasis rubra acuta" which he rightly regarded as one of Devergie's disease. This seems to be the first published in this country, and preceded Hebra's cases. The same patient's condition was described by Professor Gairdner ('British Medical Journal,' March 13th, 1875, p. 359) seventeen years later. Among the early cases may be mentioned one by Dr Wilks in the 'Guy's Hospital Reports' for 1861, which he called "general dermatitis;" the eruption was universal, red, dry, with abundant desquamation. Another was recorded by the late Dr Hillier ('Handbook of Skin Diseases,' p. 101) in 1864, and another by Dr Fagge in the 'Guy's Hospital Reports' for 1876, vol. xiii.

Some authors regard pityriasis rubra as essentially Eczema squamosum, and one of Mr Wilson's titles is Eczema exfoliatum. Dr Liveing agrees with Wilson and Fagge in looking on it as only a peculiar form of eczema. Mr Hutchinson ('Lectures on Clinical Surgery,' part i) would separate pityriasis rubra from eczema, and regard it as the type of a group of affections which differ in anatomy, but agree in being universal, in resisting treatment, and in often proving fatal. This would include *Pemphigus foliaceus* with certain cases of psoriasis and lichen.

The late Dr Baxter published a valuable paper on this disease under the title of "General exfoliative dermatitis" ('British Medical Journal,' July 19th, 1879). He considers the affection as clinically the result of a universal inflammation, and as arising by the general diffusion of either eczema, psoriasis, lichen, or pemphigus. The objections to this view are that eczema may be nearly if not completely universal, and for long periods together, without losing its characteristic features and without endangering the health. The same appears to be true of *Lichen planus*, if we accept Hebra's descriptions of universal chronic *Lichen ruber;* for this he carefully distinguishes from pityriasis rubra. *Pemphigus foliaceus* is seldom if ever universal, and differs markedly, as will be seen hereafter, from pityriasis rubra. That the whole skin may be occupied by a scaly eruption without interference with health is proved by many cases of ichthyosis.

Auspitz, who is followed by Hans von Hebra, separates pityriasis rubra from the inflammatory diseases and places it among affections of the epidermis (keratonoses) as *keratolysis*.

Origin, course, and characters.—Pityriasis rubra may undoubtedly arise from eczema or psoriasis, and probably from any other form of superficial dermatitis, including erythema, impetigo, and traumatic dermatitis; but it most often arises without previous cutaneous lesion.

It rapidly spreads over the trunk and limbs, but in an irregular fashion, unlike the gradual and methodical extension of eczema or psoriasis. Finally, it affects the whole of the cutaneous surface, including the scalp, the palms, and the soles. The skin is of a full deep red colour, not thickened and indurated as in chronic eczema, covered with profuse and abundant scales, which are large, thin, and usually detached, unlike those of psoriasis or of syphilis or the branny desquamation which follows eczema and the disappearance of the exanthems. They are apt to form successive undulating ridges which Wilson compared to those of the "ribbed sea sand;" they are exceedingly abundant, so that the patient's bed is filled with them by the peck. In most cases the surface is absolutely dry; occasionally there may

be a slight inflammatory exudation, especially in the flexures where the inflamed skin is apt to crack. This exudation has not the stiffening property which marks that of eczema. There is more or less pyrexia and general disturbance of health, especially at the onset. If, as is most frequently the case, the disease becomes chronic and inveterate, albuminuria is occasionally observed, and the appetite and health begin to fail. The irritation varies in different cases; it is usually considerable and sometimes almost as intense as in eczema, so as seriously to interfere with sleep.

Histology.—In a case of a year's standing examined after death by Hans von Hebra, the whole of the cutis was filled with leucocytes; in another case which had lasted several years all signs of active inflammation had disappeared, the Malpighian layer was thin and its cells shrunken, the papillæ atrophied, and the deep layer of cutis transformed into thick bundles of elastic fibres with abundant pigment; the glands had also suffered atrophy. In chronic cases the hair may be lost.

General exfoliative dermatitis is common to both sexes and to all ages. Though more frequent in the latter periods of life, it is not unknown in children.

Diagnosis.—Pityriasis rubra is distinguished from *eczema* by its abundant scales, by the absence of visible moisture, and by its not showing predilection for the ears, face, and flexures of joints; from *psoriasis* by the thin, loose scales, by its not specially affecting the elbows and knees; from *pemphigus foliaceus* by the scales not being preceded by bullæ; from all these forms of superficial dermatitis by its being universal and uniform in distribution, and by the severe symptoms which usually accompany it.

Prognosis.—This is much graver than that of ordinary dermatitis, eczema, psoriasis, lichen, prurigo, or pemphigus. For not only is it difficult to cure but it sometimes ends in death, especially in elderly people. The presence of albumen is a bad sign, though not a fatal one. Emaciation is still more serious, depending as it usually does on loss of appetite or sleeplessness or diarrhœa. Yet the disease is not, as Hebra supposed, incurable, nor is it by any means constantly fatal. Since his book was written cases of recovery have occurred at Vienna. In forty cases collected from various sources ('Guy's Hospital Reports,' series 3, vol. xxv), recovery ensued in fifteen, improvement in several more, and death only in eight. In these cases the fatal event was caused by bedsores and exhaustion, by lobular pneumonia, by acute pneumonia, or by bronchitis. In other cases marasmus ensues and diarrhœa ends the disease. It often persists for an indefinite period almost uninfluenced by treatment.

Treatment.—Locally the best applications are those which have been recommended in the drier forms of eczema—weak carbolic oil, lead and zinc ointment, liquor carbonis detergens with vaseline (℥j ad ℥j) freely and frequently applied. Warm baths are not counterindicated and usually give relief, but if too warm they lead to irritation afterwards, and the effect on the pulse must be carefully watched. Arsenic has not the power it possesses with psoriasis and with chronic eczema. It is best given in small doses combined with steel. Bark and mineral acids are often useful. Milk and farinaceous diet appears to suit best, and cod-liver oil should be taken if it does not interfere with other food. Good red wine, or sometimes porter, is in some cases decidedly beneficial. In one obstinate case in an otherwise healthy old gentleman, a patient of Dr Ford Anderson, complete recovery followed six weeks' sojourn at Strathpeffer in Ross-shire.

PSORIASIS

(*DRY OR SCALY TETTER*)

Frequency—Name—Anatomy and histology—Course—Symptoms—Distribution—Ætiology—Varieties: guttate form, inveterate form—Relation to pityriasis rubra, to eczema, and to syphilis—Prognosis—Treatment.

EXCLUDING scabies and syphilis, by far the most common cutaneous disease is eczema; next comes acne, and then psoriasis. Like the affections hitherto described, it is a chronic superficial dermatitis, and like them has been described as a dartrous or herpetic affection. It stands, however, at the opposite extreme from typical idiopathic vesicular eczema, with which it offers points of contrast rather than of resemblance.

An old and good name for psoriasis was "dry tetter." The Greek term signifies the condition of *psora* or itching and has no bearing on the present signification of the term. Certain forms of psoriasis were formerly known as *lepra*, which from its etymology, "the scaly disease," would be more appropriate, but the confusion with leprosy is decisive against the word. The Greek term *alphos*, referring to its white scales, was revived by Erasmus Wilson, but without general acceptance.

Anatomy.—Psoriasis is an extremely well-marked and characteristic form of disease. It begins as papules, which rapidly increase in size and form flat patches. From the beginning the scales can be seen upon the papules, and by the time they are as large as a pea the scales form conspicuous white spots. They are large, perfectly dry, strongly coherent, and not easily separable from the skin; they have also the characteristic white silvery lustre due to the abundance of air which is included between the layers of horny epidermis. When the scales are removed the surface on which they rest is seen to be red, shining, and dry, but the injection is not that of acute hyperæmia, and either stops at the edge of the scaly patch or only extends very slightly beyond it.

Histology.—The earlier dermatologists of the present century, Gustav Simon, and even Hebra, were unable to prove, what they recognised as probable, that psoriasis is essentially a form of dermatitis. As with eczema and most other cutaneous affections, the characteristic appearance disappears after death. By the better methods of modern histology Neumann established the existence of abundant cellular infiltration of the papillæ of the corium, extending along the tracks of blood-vessels in its deeper layers. They also ascertained that the papillæ are enlarged to ten or twelve times their natural size, and this papillary hypertrophy is present from the first, not only, as with eczema, in the later stages. The scales of psoriasis, like those of pityriasis rubra, consist almost absolutely of keratin—unmixed with fibrin and leucocytes as in chronic eczema and syphilis, or with sebum as in pityriasis capitis.

Local evolution.—Psoriasis is no less characteristic in the regions it affects

than in its anatomical lesion. Its favourite spots are over the olecranon process of the ulna and over the patella, ligamentum patellæ and tubercle of the tibia. In fact it is remarkable how very rarely these spots are found free even in the most chronic and varied forms of the disease. Here it begins and here it almost always remains. From these points it spreads downwards over the extensor surface of the forearm and on the shin and calf, not, however, by a continuous advance as is the case with eczema, but by the development of separate patches with well-defined margins, which as they increase in size become confluent with the originally diseased surface. The whole upper and lower extremities may be covered with such patches, which by their coalescence form large spaces; but there will always be found more or less extensive islands of healthy skin between the diseased parts, and these will have a concave, while the scaly patches have a constantly convex, outline. On the back or chest the same process is seen on a larger scale. As the raised, red, and scaly edge of the eruption advances, the inner parts which were first affected lose their scales and return more or less incompletely to a healthy condition, so that by this progressive spread and involution of the disease the scattered scaly patches in which it began gradually give place to extensive surfaces of almost normal appearance, bounded by sinuous lines of red and scaly skin made up by the intersecting segments of many circles. *Psoriasis gyrata* was the technical term applied to this stage.

After psoriasis has lasted for some time its colour begins to acquire a deeper and brownish tint. It no longer disappears completely upon pressure, that is to say, pigmentation has been added to hyperæmia. In inveterate cases this becomes very characteristic, the colour being of a deep brown, sometimes almost mulatto tint. When the disease has been cured, when the scales are removed, the hyperæmia has subsided, and the finger cannot feel anything but healthy skin, dark pigment-blotches remain to attest the nature of the recent malady. They always disappear in time, but, especially in old persons, their disappearance is slow. It may be said that next to syphilis, psoriasis produces pigmentation more quickly than any other form of dermatitis, and the depth of pigment may be as great as in the most chronic cases of eczema or of prurigo senilis. In this as in other respects psoriasis resembles lichen planus, and differs from pityriasis rubra.

Course.—Psoriasis is never acute. Even when it develops rapidly it is unaccompanied by the ordinary symptoms of inflammation and never causes constitutional disturbance. Often a patch on each elbow, or on the elbows and knees, may appear and remain for years before it shows signs of spreading. When it has become extensively diffused and passed through the centrifugal process above described, it will, if untreated, enter upon a very chronic and almost interminable course, the skin being habitually thick, harsh, and dry, and the general aspect resembling that of some of the forms of dry scaly chronic eczema in old persons.

Of all skin affections, psoriasis is most prone to *recur*, more so even than eczema. It is very rare for a single outbreak to occur. Sometimes when the eruption has only just disappeared under treatment, a fresh attack comes on, and the very means which will almost infallibly cure it when developed are often powerless to prevent its return.

Notwithstanding its etymology, itching is comparatively unimportant as a symptom of psoriasis; it is much less severe than in eczema, scabies, or prurigo. In many instances there is no irritation at all, in most it is

slight, but in a few it is sufficiently troublesome to demand special treatment. It is still more rare for the affected parts to smart or to feel hot and tender. Though pathologically it is an inflammation, it is the most chronic cold, and uninflammatory of all the inflammations of the skin.

It produces no constitutional effects, and persons subject to it are entirely free from special liability to any other disease. The digestive, urinary, and other functions are carried on as usual, unaffected by the condition of the skin.

Distribution.—Psoriasis is of all diseases the most completely and constantly symmetrical: not more so, it is true, than typical forms of eczema, but its range is so much more restricted and its varieties so unimportant, that while typical eczema does not include three fourths of the whole number of eczematous cases, we seldom meet with one of psoriasis which deviates from the characteristic type. As above stated, its favourite or practically its constant seat is upon the two elbows and the two knees; next it is common over the whole extensor surfaces of the extremities, specially the forearm, the front and outside of the thigh, and the peroneal side of the leg. Even when most extensive it shuns the bend of the elbow and the popliteal space. It not unfrequently extends from the forearm to the back of the hand, and from the leg to the dorsum of the foot, and occasionally may cover the fingers and even affect the nails. *Psoriasis unguium* is known by the excessive and unsightly thickening of the nail, and by the absence of soreness and suppuration of the matrix. It sometimes occurs when the rest of the fingers or toes is free from the disease, but almost always spots of psoriasis will be found on the elbows or knees, or the patient has previously suffered from the disease. The only other affections of the nails which at all resemble it are eczema unguium above described, and onychomycosis, to be mentioned under ringworm.

Psoriasis very rarely affects the palms or the soles, and never unless other parts of the body are previously the seat of the disease. What used to be described as primary psoriasis palmaris was probably either eczema squamosum or scaly syphilis. When present the scales of palmar psoriasis are comparatively small, but the patches keep their well-marked edge. There is little or no disposition to form cracks, and the soreness and irritation of eczema of the palms or soles is absent.

Next to the extensor surfaces of the limbs psoriasis is most common on the trunk. The shoulders, back, and loins, the sacral and gluteal regions, are very commonly its seat, the chest and abdomen somewhat less so. Indeed, we never see psoriasis of the abdomen which does not also affect other parts of the trunk, and it is very rare to find psoriasis of the trunk when the limbs are completely free. The genital organs are occasionally the seat of psoriasis, which has usually spread from the abdomen or the thighs; but this is far less frequent than eczema of the same parts, and what used to be called psoriasis scroti is really syphilis squamosa. The face and head are less frequently attacked by psoriasis than the trunk, far less frequently than the limbs; but with so common a disease, cases often occur in which the red scaly patches appear upon the neck, the cheeks, the forehead, and the scalp. The scales are usually smaller upon the face, the whole aspect less characteristic and apt to be further confused by slight ordinary dermatitis, but the presence of unmistakeable psoriasis on the limbs or trunk prevents any mistake in diagnosis. On the scalp the closeness of the hairs prevents the formation of large scales, and the sebaceous secretion gives

them a greasy consistence and a yellowish tint. *Psoriasis capillitii* is a not infrequent affection, and broadly distinguished from eczema and impetigo capitis, from syphilodermia and seborrhœa sicca or pityriasis capitis. It is always dry, the scales are coherent, the hair does not fall out, and it is coincident with existing or previous psoriasis of other parts.

The affection called *psoriasis labiorum* has been described already as a form of eczema. It is doubtful whether psoriasis affects the mucous membranes. Psoriasis linguæ is *leukoplakia*, white patches on the tongue, distinct from syphilis and often antecedent to epithelial cancer: when it is coincident with an affection of the skin, that affection is usually lichen planus. The occasional coexistence of these patches with ordinary psoriasis must be admitted; but the question is whether the coincidence is accidental or not.

Ætiology.—The cause or causes of psoriasis are absolutely unknown. By French writers it is generally ascribed to a dartrous diathesis, and by those who go still further into speculation a *dartrous* is distinguished from an *arthritic* psoriasis. In England it is very commonly regarded as *gouty*, while by some authors it is considered, especially in children, as a *scrofulous* disease; aud there is much the same evidence for the one hypothesis as the other. Dr Yandell regards a great deal of psoriasis as well as of eczema as *malarial* in origin; and in the last century psoriasis was by most physicians considered undoubtedly *scorbutic*. Some writers have speculated on the possible connection of psoriasis with *leprosy*, and would have us regard it as the expiring and gradually mitigated manifestation in modern times of the scaly leprosy, white as snow, which is described in the Old Testament. The remarkable centrifugal progress above described naturally suggests the idea of a parasitic vegetable growth; but we may confidently assert that no fungus is present, and although it has been asserted that *bacteria* may be found in the affected skin when the scales have been removed, their presence, which is certainly not universal, must be regarded as purely accidental, and would neither explain the course and spread of the disease nor help in its treatment. So unwelcome is it to find a disease without a cause, that some dermatologists refuse the title to psoriasis, and regard it as a condition of health.

With respect to gout it must be remembered first that the word, in Germany and even in England, is often applied upon very insufficient evidence. In fact those who use it would sometimes not even imply the presence of urate of soda in the joints. Moreover, the diagnosis of gout is always acceptable to an Englishman of the middle class. On the other hand, it is true that few families of rank in this country are free from unmistakeable gout in some of their members. But psoriasis is comparatively rare in private practice as compared with that of hospitals. Very few of those who have unmistakeable podagra are liable to psoriasis, and psoriasis is as common in Scotland, Germany, and America, where gout is rare, as in England, where it is frequent. If we determine that every disease must have for cause some condition already known, it will be easy to find one in the list given above for every case of psoriasis; but such a practice hinders the progress of knowledge of the real causes of disease, interferes with rational and successful treatment, and leads to an acquiescence in superficial statements and arguments which is fatal to medicine either as a science or an art.

Psoriasis is, after eczema, scabies, and syphilis, the commonest disease of the skin. It occurs equally in both sexes and at all ages above infancy; it becomes more common from the age of six or seven up to puberty, and the

first attack usually falls in childhood or early adult life. It may, however, begin after fifty and even in old age.

Varieties.—As above stated, psoriasis compared with eczema is singularly uniform in its anatomy and natural history. The description above given applies to ninety-nine out of a hundred cases, of course with individual variations, but less than those of even such typical diseases as typhus and variola. There is, in fact, only one variety which demands notice; in children the ordinary form is frequently seen, but more commonly the early spots of *Psoriasis guttata* as above described never grow into the nummular stage, and the large patches of gyrate psoriasis are decidedly rare under puberty. This would be scarcely worth mentioning in itself, but the spots are also remarkable for having little or no red border. They produce no irritation, and—as is common with other diseases affecting children—the local distribution is less rigidly marked than in adults. The guttæ are frequently seen on the face, and they are perhaps more abundant on the trunk than on the limbs. Dr Liveing thinks, moreover, that guttate psoriasis occurs particularly in children who are scrofulous, *i. e.* pale and thin. It is, however, often seen in those who are robust, and certainly in most cases there is no chronic enlargement of the lymph-glands, no caries, no chronic synovitis, and no evidence of tubercle.

When psoriasis has lasted for many years and has spread over the greater part of the surface, it loses much of its characteristic appearance, the scales are less abundantly formed, the margins are less definite, and the whole skin becomes thickened and indurated, so that it often requires careful investigation and a knowledge of the earlier stages of the affection to distinguish this *psoriasis inveterata* from the dry and chronic eczema described in a former chapter.

Again, there is no question that psoriasis may pass into, or be supplanted by, the dry scaly and universal dermatitis described in the last chapter as pityriasis rubra. As was there stated, the late Dr Baxter thought that any dermatitis—eczema, psoriasis, lichen, or pemphigus—might, if sufficiently extensive, assume the characters of that remarkable disease. In a paper in the 'Guy's Hospital Reports' (vol. xxv, p. 266), reasons are given against accepting this hypothesis. The writer had a remarkable case in which a woman, who had been in St Thomas's Hospital under Dr Payne with ordinary psoriasis of the elbows and knees, and whose daughter was a patient of his own also with psoriasis, came under his care with marked and typical pityriasis rubra. It is very probable that some at least of the cases of general psoriasis, described by Hardy as very rare, would have been recognised by Devergie as pityriasis rubra.

One may admit that eczema and psoriasis, which in so many ways are allied by points of contrast, have connecting links on the one hand with pityriasis rubra or universal exfoliative dermatitis, and on the other with lichen planus, which, as we saw, sometimes so closely resembles psoriasis, while by its relation to ordinary forms of lichen it has affinities with papular eczema. We draw lines as nearly as we can in accordance with the broad demarcations of pathology and natural history, but here as in other departments of medicine it would be pedantry to deny that there are transitional forms which it is difficult or may be impossible to classify.

The important question, however, of the diagnosis between psoriasis and the scaly forms of syphilis is one which rests on the absolute distinction of cause and is of the utmost practical importance. The locality

and symmetry, the character of the scales, the colour, the presence of itching, the uniformity of the lesion, and the absence of other signs of syphilitic disease are the points to be attended to. The last, however, may deceive, for a man with psoriasis may acquire syphilis, as he may scabies; and the writer has notes of three or four cases in which true psoriasis and a secondary syphilitic eruption existed in the same patient, ran independent courses, and were cured by different treatment.

Prognosis.—Psoriasis if left to itself lasts for an indefinite time, though almost always getting better or worse at intervals. It never interferes with the health or affects other organs than the skin. After being cured, it is of all diseases most apt to return.

Treatment.—The external treatment of psoriasis consists in inunctions of some preparation of tar. Nothing is so effectual as the unguentum picis liquidæ of the Pharmacopœia well rubbed in at night and allowed to stay on while the patient sleeps in a special suit of underclothing; it may then be washed off in the morning, to be reapplied at night. When the scales are very thick and indurated, it may be well to precede this application by hot baths and soft soap. When the smell and colour of tar are objected to, useful though less efficient substitutes may be found in the liquor carbonis detergens, made into an ointment with lanoline, two drachms to an ounce, or in the huile de Cade, oleum rusci, &c. Another plan of obtaining the same result is to apply a spirituous solution of tar or the liquor carbonis detergens diluted with water. Goa powder and the chrysophanic acid which it contains are powerful cutaneous stimulants, and have been often used with success in the treatment of psoriasis. They occasion, however, with many patients, considerable pain, and stain the skin and linen unpleasantly, as well as the hands of the person applying it.* In cases where tar is inadmissible a better substitute is pyrogallic acid, gr. xv—xxx to an ounce of benzoated lard or lanoline, the strength being increased with caution. For rapidity of cure with freedom from unpleasant smell, this is perhaps the most eligible of all applications.

Beside the local treatment, it is almost always desirable, after the scales have been thus removed, to put the patient upon a course of arsenic. It is usual to prescribe it in a bitter infusion, but it will be found to agree quite as well, and to be more constantly taken, if merely diluted with water or flavoured with syrup or peppermint. It should always be taken at or immediately after a meal. Three, four, or five drops in an ounce of water three times a day is the dose to begin with, and it may be increased to ten or beyond. If properly diluted, and taken with food, even full doses very rarely cause pain, sickness, or diarrhœa. The first sign of the physiological limit being reached is usually irritation and slight injection of the conjunctiva. As soon as the patient feels his eyes begin to itch he should be instructed to leave off his medicine for a couple of days, and then resume it in slightly smaller doses. He has then reached what is for him the full therapeutical benefit.

* Dr Crocker writes ('Brit. Med. Journ.,' November 19th, 1887): "The stains in linen are quite indelible without injuring the fabric, but they may be avoided by using the Auspitz-method. A gutta-percha varnish, called traumaticin, is made by dissolving ʒj of pure gutta percha in ʒx of chloroform, ʒj of chrysarobin is added, making an emulsion, which is painted on with a stiff brush after removing the scales every day, until a thick coat is formed: it is then allowed to peel off and renewed. It acts effectually and does not stain. Besnier modified this by brushing in ʒj of chrysarobin in ʒx of chloroform, and then varnishing with traumaticin. Both plans are equally good."

Psoriasis may often be cured by arsenic without any external application whatever, or by local treatment, without internal medication; but in most cases the cure will be hastened by the application of tar, and will be rendered more permanent by the administration of arsenic.

In the case of anæmic persons it is desirable to give steel. We may then combine the liq. arsen. hydrochlor. with the liq. ferri perchlor. In other cases Fowler's solution is the best. It is the fashion to administer Pearson's solution (the arseniate of soda) to children, but there is little doubt that it is absorbed in exactly the same form. When arsenic disagrees it should not be hastily given up, but the dose should be diminished until unpleasant effects no longer follow, or we may sometimes prevent them by adding a few drops of laudanum or a little compound tincture of camphor to the dose. When the patient suffers from gastritis and sore eyes, four drops of Fowler's solution, three, or even two, will probably be sufficient to cure the psoriasis; when a patient can take ten without discomfort, it may be that fifteen will be needful to cure his psoriasis.

Children take arsenic very well. When they are pale and thin and ill-nourished, cod-liver oil is often a useful coadjutant. In the guttate form of the disease, most common under puberty, local treatment is often scarcely necessary.

Purgatives and diuretics are quite unnecessary; and colchicum is not needed, as it undoubtedly is in the treatment of certain irritable and probably gouty forms of eczema.

In some cases psoriasis is very obstinate, not only returning again and again after being cured, which is common, but yielding very little to the most careful local measures and to the most persevering use of arsenic. In these cases liquor potassæ in half-drachm doses sometimes succeeds, sometimes iodide of potassium* and other diuretic remedies, and sometimes prolonged maceration with soft soap and water. But we occasionally meet with cases which seem to be quite incurable.

* This has been advocated by Dr Cæsar Boeck and other physicians in Norway and Denmark. Dr Haslund gives enormous doses without harm (see 'Brit. Med. Journ.,' 1888, vol. i, p. 27).

PEMPHIGUS

(*BLADDER-TETTER*)

Names and definition—Anatomy—Histology—Local distribution—Age and sex—Question of an acute form of pemphigus—Diagnosis—Prognosis—Pemphigus malignus—Pemphigus foliaceus—Serpiginous form—Hutchinson's cases—Chiropompholyx—Hydroa of Bazin—Frequency of pemphigus—Treatment.

We now come to a form of superficial dermatitis which is decidedly rare compared with eczema or psoriasis. Although not less remarkable than these in its anatomical characters, its course and natural history are far less characteristic, its pathology more obscure, and its origin entirely unknown. It has been called by two names, *pemphigus* and *pompholyx*,* but of these terms, which, like lepra and psoriasis, were made separate genera by Willan and his disciples, there is no need to retain more than one.

The name *pomphus* seems to have been originally applied to what we now call a wheal; *pemphix* meant a bulla, and pemphigus was applied to a supposed "febris bullosa" of doubtful nature. Bateman practically admits only one bullous disease, a chronic superficial dermatitis, characterised by blebs.

It was by Willan associated with erysipelas, a striking example of the results of following an anatomical, or any exclusive, basis of classification for so complex conditions as diseases. He had previously united it with vesicular diseases, but distinguished the two orders in consequence of the criticism of Tilesius, of Leipsic ("über die flechtenartigen Ausschläge," in Martin's 'Paradoxien,' 1801).

Anatomy.—The bullæ of pemphigus begin with a scarcely demonstrable papular stage. The first lesion seen is usually a small transparent vesicle which rapidly increases to the size of a pea or larger. These bullæ are sometimes seated on perfectly natural skin; sometimes, however, they are surrounded by a rose-coloured injected ring, but this is narrow. They are never found upon an actively inflamed or swollen surface. They may burst when not bigger than a pea or a marble, but, on the other hand, will sometimes increase to the size of a billiard-ball, or more. They are usually tense and hemispherical, occasionally oval. There may be either a single bleb or several of various sizes, irregularly scattered over the same region, and when in such groups the intervening skin is often injected. Each bulla, however, forms separately, and it is very rare for two to run together. The contained liquid is transparent, and gives the bulla a pearly appearance. When removed by pricking it is thin, watery, colourless, not coagulating, becoming opalescent or turbid on heating, and showing a few leucocytes under the microscope. After a time, however, it often becomes turbid from increase of the inflammatory corpuscles, and

* Πομφόληξ means a bubble, almost synonymous with φύσαλις, and is applied by Hippocrates to the froth which forms on urine (Aphor. vii, § 34).

before the bulla bursts the contents may be opaque and yellow, in fact almost purulent. They do not, however, acquire the thick creamy character of pure pus, and always begin as serous and not purulent cavities. Threads of fibrin also appear not unfrequently before the rupture of the vesicle. Still more common is an admixture of blood, which gives a pinkish aspect to the bulla. After it has burst, fresh secretion soon ceases, the ruptured cuticle is either torn off or adheres to the exudation, and the lymph, whether serous, puriform, or coagulated, dries up into a thin yellow crust which may be more or less stained by hæmoglobin. This soon falls off and leaves a smooth, healthy surface, with scarcely any desquamation; but some passive injection remains, and with this may be mingled more or less pigmentation, so that the circular patches, of sizes varying from a sixpence to a florin, remain for some time as characteristic evidence of pemphigus.

Histology.—The inflammatory exudation of a bulla produced by such an irritant as cantharides takes place in the deepest part of the Malpighian layer of the epidermis. The cells of this layer are first drawn out into bands by the accumulating serum, so that in the early stage each vesicle consists of a series of loculi, as in the case of a burn, first described by Biesiadecki; this stage is long and well marked in the case of traumatic bullæ and in the vesicles of smallpox.

The bullæ of pemphigus, however, as shown by Leloir and Auspitz, consist of an exudation of serum between the horny layer of epidermis and the deeper Malpighian layer, or between the granular layer and those beneath it, without the cells themselves being much affected. There is no vacuolation, the cavity is at no stage multilocular, and the formation of the bleb is far more rapid than that of an inflammatory vesicle or pustule of half its size.

No scars are left after pemphigus, and only moderate pigmentation.

Distribution.—Pemphigus differs from eczema and from psoriasis in being *unsymmetrical*, and having no definite local predilection. The bullæ appear sometimes singly (*P. solitarius*), or succeed one another indefinitely upon distant parts of the body (*pompholyx diutina*); more often two or three up to half a dozen form an irregular patch; and isolated bullæ, or one or two other patches follow on other parts of the surface. Occasionally the trunk and limbs may be so covered that scarcely any region can be said to be entirely free, yet even then the lesions show no preference for one part over another. There is scarcely any part of the surface on which pemphigus may not be seen. On the trunk and limbs it is most frequent; the abdomen and thighs, the genital organs, the ears, the hands and the feet; even the palms and soles and the matrix of the nails may occasionally be the seat of pemphigus; the hairy scalp is least frequently affected. It is said that bullæ have been observed in the mouth and on the conjunctiva.

Age and sex.—Pemphigus, though belonging to the rarer diseases of the skin, may be seen in patients of almost any age. It is commonest in children, decidedly infrequent in adults, but may sometimes be observed in elderly patients, when it is apt to assume its more severe characters. Among 38 consecutive cases of the writer's, 26 occurred in males, and 12 in females; 7 between one and five years of age, 15 between six and ten years, 11 between nineteen and fifty, and one in an old woman of sixty-eight.

Acute pemphigus. — Hebra discusses the existence of acute or febrile pemphigus (*febris pemphigodes*), and like Bateman before him, concludes that when urticaria, erysipelas, rupia and other forms of syphilis, and herpes iris

are excluded, there is no such disease. The late Dr Sparks, however, in 'Quain's Dictionary,' says that the existence of such cases is now certain. Dr Southey has recorded a case ('Clin. Soc. Trans.,' viii, 1875), Dr Payne another with a table of temperatures ('St Thos. Hosp. Rep.,' vol. xii) and Dr Duckworth a third ('St Barth. Hosp. Reports,' vol. xx). The last occurred in a man of fifty-four, suffering from Bright's disease, and he died on the ninth day; but the event was probably not due to the eruption on the skin, and if not cut short by death this might have proved chronic. Moreover, it is possible that this as well as other cases might be interpreted as bullous erythema, though it would doubtless be difficult to maintain the distinction in every instance.

Diagnosis of pemphigus.—The bullæ of this disease are so characteristic that it cannot be overlooked, and cannot be mistaken for eczema, lichen, psoriasis, or any other of the forms of superficial dermatitis already described. But all bullous eruptions are not pemphigus.

1. Blisters may be produced designedly or accidentally by local irritants, especially by scalding water, or by cantharides. The traumatic bullæ which follow extreme heat are of two kinds—true inflammatory products containing serum or pus and bladders filled with gas, which have been formed by the lymph of the living skin being turned into vapour and expanded by heat. The latter condition was long ago described by Hilton as the result of burns and scalds; it is of rare occurrence. Its purely physical nature he proved by the fact which the writer has himself verified, that it is possible to produce it in the skin after death. If a hot iron be held close to the surface, the cuticle rises in a blister like that produced by the sun on a painted board, and on pricking it, no liquid is found within. This is strikingly seen in a negro's skin, when the white cuticle is raised from the dark rete mucosum beneath.

Factitious inflammatory bullæ are usually seen on the arms, and in doubtful cases the glistening scales of the elytra of the Spanish-fly may often be distinguished by a lens.

2. Scabies is sometimes accompanied by bullæ, especially in children. An example of this was figured in the face of a child whose appearance closely simulated that of pemphigus ('Guy's Hospital Reports,' 1877).

3. Syphilitic eruptions in the later stage of the disease are often bullous. Usually the exudation becomes purulent and the resulting crusts are massive, dark from blood-pigment, and more or less conical, forming the condition described as "rupia," and leaving a superficial ulcer when they fall off, with considerable pigmentation and final cicatrisation. In cases of congenital syphilis, however, bullæ exactly like those of pemphigus may be observed; so-called *pemphigus neonatorum* is probably always syphilitic. Besides other signs of the congenital disease, the appearance of the bullæ upon the palms and soles is a character which is diagnostic.

4. More difficult of distinction from true pemphigus are the bullæ of certain forms of erythema, to be presently described as herpes iris and erythema bullosum. Their locality, their symmetry, and their acute or subacute course, are the chief marks which distinguish these erythematous eruptions from true pemphigus.

5. Iodide of potassium occasionally produces along with other lesions bullæ which have been mistaken for those of pemphigus.

Prognosis.—In children pemphigus is rarely fatal (excluding so-called *syphilitic pemphigus*), and under suitable internal treatment it is in

most cases quickly curable. But in old persons it is apt to spread very widely; sleeplessness and loss of appetite follow, and death may result. This is most to be feared when there is chronic renal disease present, but it may occur independently of this complication.

Such cases have been made a distinct variety, *pemphigus malignus vel cachecticus*. The bullæ are very numerous, are never tightly filled with serum, but look flaccid and rupture early. There is little effort at healing, and extensive raw patches cause much pain and distress, combined sometimes with more or less itching. The exudation is frequently hæmorrhagic and sometimes fibrinous, or, as the German writers call it, "croupous." As in other severe and extensive forms of dermatitis there is sometimes albumen in the urine, independently of previous Bright's disease.

These serious cases, though rare except in old persons, may occur at any age; in children, bullæ after bursting are sometimes succeeded by gangrene, and this *pemphigus gangrœnosus* has also been separately described. It has no doubt been frequently confused with what used to be called "rupia escharotica" and "pemphigus neonatorum," that is to say with a bullous syphilitic eruption. But there is no question that true gangrenous pemphigus does occur. In a little boy aged four, who died of it in Guy's Hospital, 1882, we found *post mortem* all the viscera perfectly normal, and there was no reason to suppose the presence of congenital lues. In one case it occurred after varicella.

Ætiology.—The cause of pemphigus is absolutely unknown, although, as in other cases, teething, gastric irritation, excess in diet, irritability of the system, mental affections, anxiety, fatigue, amenorrhœa, exposure to cold, and residence in damp situations have been confidently stated as each a cause of the disease. According to Alibert, the "lymphatic temperament" predisposes to pemphigus, which probably means what is true, that it is more common in children than adults.

It is now well established that pemphigus is never contagious. Hebra relates one remarkable case of heredity.

Pemphigus foliaceus.—Cazenave described a remarkable form of cutaneous disease under this title, which has since been recognised by Hebra and other dermatologists. It is rare, and the writer has only seen two well-marked cases, one at Vienna, and the other in Guy's Hospital.

The patients are usually adult women. The blebs appear at first like those of ordinary pemphigus, but they never become tense and pearly in appearance. They rupture early, and form thin, dirty-white laminæ, which continue to exude a scanty secretion. The aspect of the affected skin has been likened to that of flaky pie-crust, to birch-bark, and to dead leaves—whence the specific name.

Beside the anatomy, the distribution of this form of pemphigus is remarkable, in being more or less universal. On this ground and its malignancy the late Dr Baxter associated it with pityriasis rubra.

Its course is very slow, and there is no disposition to recovery. Indeed, it is doubtful whether any genuine case of pemphigus foliaceus has ended favourably. Drugs have little or no influence upon it, and after a protracted illness the patients die emaciated, or are carried off by some intercurrent disease.

Serpiginous pemphigus is a rare form, which arises only in chronic cases. The bullæ, which are small, are seen on the red advancing border of a considerable space of skin, formerly the seat of others that have disappeared.

When first seen in this latter stage it might well puzzle an observer. A well-marked case of pemphigus serpiginosus, which began as an ordinary case, recurred, and each time was cured by arsenic ('Catalogue of the Models of Cutaneous Disease in the Museum of Guy's Hospital,' p. 92).

Hutchinson's bullous disease of hands and feet.—A curious form of bullous eruption, which may be at least provisionally called pemphigus, was shown by Mr Hutchinson at the Pathological Society, as "hand-and-mouth disease." Beside the bullæ on the trunk and limbs there was severe inflammation of the hands with loss of nails, and also inflamed mucous membrane of the mouth and tongue. A case was exhibited at the Dermatological Society in 1885, which was recognised by Mr Hutchinson as of the same character. There was unmistakeable pemphigus here, and both loss of nails and sore mouth in pemphigus have been described by Hebra, so that no doubt he would have included under that title the curious cases described.

Moreover, the same combination was described by Rayer as complicating pemphigus.

Chiro-pompholyx.—This affection also was described by Mr Hutchinson. It is chiefly confined to the hands and feet, is symmetrical, affects the nails, is recurrent, and the bullæ are small without dermatitis around them. Dr Robinson, of New York, and Dr Liveing have described similar cases. It affects the palm and sides of the fingers, as well as the dorsum of the hand. Before rupture the small bullæ, or large vesicles under the thick skin of the fingers, are described as like sago grains. There seems no doubt that this disease is quite distinct from the affection of the sweat-glands, described by the late Dr Fox as dysidrosis. It rather resembles erythema bullosum.

Hydroa.—The group of eruptions named hydroa by Bazin is not a natural one, either clinically or pathologically. Of the three species described by him, the first, or vesicular hydroa, would seem by its localisation on the back of the hands and wrists, and on the front of the knees, as well as by its acute but sometimes recurrent course, to be clearly a form of erythema. Other cases are identical with the curious affection long known as herpes iris, which will be referred to in the chapter on erythema. Bullous hydroa probably includes *pemphigus pruriginosus* or herpes gestationis (*infra*, p. 934), and the bullous form of erythema multiforme. Some cases, again, which have been described as hydroa have turned out to be iodide rashes (see an elaborate paper by the late Dr T. Fox in the Philadelphia 'Archives of Dermatology' for 1880, p. 16, and a more recent one by Dr Crocker in the 'Lancet' for May 22nd, 1886).

Frequency of pemphigus generally.—Hebra, writing of twenty years' experience in the enormous General Hospital of Vienna, as well as in his large private practice, could reckon only about 200 cases. He estimates that, excluding infants, one case of pemphigus occurred in 10,000 cases of illness generally. But we must remember that the hospital statistics apply to all medical (as well as surgical) diseases, whereas his own practice was exclusively dermatological. He found in thirty years' statistics at the General Hospital that there were ten cases of pemphigus in men for rather more than three in women, excluding pemphigus foliaceus. In a report to the American Dermatological Association in 1881 only twenty cases occur in 11,000; and Erasmus Wilson reported only nineteen cases among 10,000 private patients.

Treatment.—In Joseph Frank's work on diseases of the skin, he stated that the best treatment of pemphigus is to leave it alone. Hebra proved the uselessness of diuretics and purgatives, tonics and quinine, mineral

acids, Rayer's vinegar and Carlsbad waters. Former English physicians recommended venesection or leeches with antiphlogistic regimen, but with a caution to pursue the plan guardedly, which probably meant not to pursue it at all. They go on to recommend acids and bark. In the first edition of Wilson's work, he writes, "When there is reason to believe that the eruption is an effort on the part of nature to determine to the surface a morbid disposition, I should strongly recommend the employment of mustard baths to the entire surface of the skin, or a stimulating liniment of some kind, such as that of croton oil in the proportion of a drachm to an ounce of olive oil, to be well rubbed into the parts of the skin." Hardy says: "Le traitement général du pemphigus est encore à trouver." German authorities speak doubtfully of the prognosis in this disease, and depend chiefly upon local applications for its treatment. The late Dr Tilbury Fox recommended chlorate of potash, good food, and above all quinine, which he preferred to arsenic. At the present time, however, English physicians are agreed that arsenic is as much a specific remedy for pemphigus as for psoriasis. No doubt it occasionally fails even in ordinary cases, which can also be said of mercury in syphilis, and no one pretends that it will cure gangrenous pemphigus, or pemphigus foliaceus, or bad cases of extensive pemphigus in aged patients; but in nine tenths of the cases of pemphigus occurring in children and young adults, the statement holds good which was made by Mr Hutchinson* that arsenic may be esteemed almost a specific remedy, even in severe cases. The late Dr Fagge, as well as Dr Habershon, Dr Hillier, Dr Gee, and many others support the same opinion. The drug should be administered on the same principles and with the same determination as recommended in psoriasis.

Occasionally, however, we meet with cases in which after perseverance with varied doses and varied forms of administration we are obliged to abandon arsenic, not because of its disagreeing (that can always be met by diminishing the dose), but because the disease is unchecked. The best remedy then is tincture of steel. Sometimes quinine, guaiacum, or cod-liver oil succeed when other drugs have failed.

Locally no applications are very useful; whatever is most soothing is best, either zinc ointment, or oxide of zinc in powder, or what is often more pleasant and effectual, oxide of zinc with finely powdered chalk or gypsum suspended in water, and applied with a large soft brush. In very extensive and severe cases, continuous baths have proved useful. In true gangrenous pemphigus of children, excluding syphilis, brandy and strong broths, or raw meat, with chlorate of potash internally, is the best treatment, and commonly proves successful. But even in infants one-minim drops of Fowler's solution should be administered.

In bad cases, especially in aged patients, attended with restlessness and distress, opium is a most valuable remedy, but unfortunately its use is forbidden, or much circumscribed by the not infrequent presence of albuminuria. When this is absent it has a most valuable effect.

So far as is known, neither arsenic nor any other drug is of service in cases of pemphigus foliaceus.

* 'Med. Times,' Feb., 1854. See also the 4th of Mr Hutchinson's 'Lectures on Clinical Surgery:' "Can Arsenic cure Pemphigus?"

SCABIES

Importance of the disease—Its nature—The acarus—The superficial dermatitis it produces—The distribution of the parasite and of the inflammation—Diagnosis—Treatment.

SCABIES,* though once scarcely accounted worthy of a place in nosology, and though without the interest of danger, is really one of the most important diseases from a scientific point of view; for, if this were the place to enter fully into its history and pathology, we should find that it illustrates the whole progress of scientific medicine,—the ancient method which still survives of inventing explanations instead of investigating circumstances, the fallacy of ascribing results to dyscrasiæ of which the existence has never been proved, the survival of doctrines in pathology which have long been exploded in physiology, the value of apparently useless knowledge, the bearing of pure sciences like zoology upon practical therapeutics, the nature of inflammation and the relation between an irritant and an irritable tissue, the eradiation of sensations, the pathology of pruritus, and the importance of a patient's nails in the production of cutaneous lesions. Finally, scabies is the typical example of a disease which is now as fully known as it is perhaps possible for us to know any disease—of which we know the pathology and the cause, of which we can explain the symptoms, which we can diagnose with certainty, which the boasted *vis medicatrix naturæ* is utterly powerless to affect, but which we can cure by definite, simple, and rational means, quickly, safely, and completely.

Scabies, like the affections which have hitherto occupied us, is a superficial dermatitis; in the character of its lesions it may even be called a common superficial dermatitis, for they do not essentially differ from those which will be produced by any common mechanical or chemical irritant of sufficient energy, and are exactly comparable in their anatomy to the vesicles of eczema, the papules of lichen or prurigo, the bullæ of pemphigus, and the pustules of impetigo. Hebra, therefore, since as we have seen he called all common superficial dermatitis of traumatic origin "eczema," logically describes scabies as a form of eczema. But, as explained on a previous page (p. 873), eczema is not a mere traumatic dermatitis, and scabies must be separated from all other diseases because its cause, its prognosis, and, above all, its treatment, are totally different. We may define it as a superficial dermatitis of various degrees of severity, but always accompanied with intense pruritus, which results from the invasion of the skin by a parasitic acarus and from the scratching which ensues.

The itch-mite.—The living cause of this disease is the female itch-mite now known as the *Sarcoptes hominis*, formerly as the *Acarus scabiei*, belonging to the acarine division of the class Arachnida. It has four pairs of legs (which at once distinguish it from parasitic insects) and is clothed in a chitinous integument furnished with abundant bristles. The male acari

* *Synonyms.*—Itch—*Fr.* La gale—*Germ.* Die Krätze.

which are much the smaller in size and fewer in numbers, live upon the surface of the body, but do not burrow. The female after impregnation digs her way into the integument, forming a straight, curved, or sinuous *cuniculus* (mite-burrow, *Milbengang, sillon* or "run") which is visible to the naked eye as a slightly raised ridge, with a dark depression at one end (the entrance clogged with dirt) and a papule or small vesicle at the other, where the parasite lies.* A lens of low power shows these characters more clearly, but it is comparatively rare to see the runs perfectly well developed, for they are injured by the inflammation set up, by the patient's scratching, by friction and by dirt. When fresh they are best seen in the soft skin between the fingers and on the ulnar and palmar side of the wrist, still better when present in the skin of the prepuce and penis, or in that of the mammary gland in women. In children their locality is less certain, and they are much less easily found. With quick eyesight and a little dexterity the burrow may be laid open with a needle from the entrance to its blind extremity, and the acarus, a minute white grain just visible to the naked eye, extracted. It generally clings to the point of the needle, but a microscopic slide with a drop of water, glycerine, or liquor potassæ should be ready to receive it. The needle should be sharp, stout, and not too long or elastic; some prefer the broader needle of the oculist. Another plan is to excise the parasite, burrow and all, by means of a sharp pair of scissors curved on the flat. The winding passage can then be demonstrated, with the black granular fæces of its inhabitant, and often with a row of oval eggs in chitinous shells, which are laid one by one as the acarus bores deeper into the skin. Sometimes the scissors fails to secure the parasite, but proves its presence by that of one or more of its ova.

The dermatitis.—The presence of the acarus produces irritation which in most cases is intense, equal to that of the most irritable eczema or the worst kinds of prurigo; but often it is comparatively slight, only annoying the patient after he is warm in bed, when the skin is more vascular, the papillæ more sensitive, and possibly the acarus more lively, while the patient has nothing to divert his attention from his own sensations. The degree of inflammation also varies extremely, and cannot always be explained by the more or less severe scratching of the patient. As above stated, there is usually a small vesicle formed at the end of each run; but beside these, large vesicles, bullæ, and pustules frequently follow, first on the hands and then (probably through transfer of pus and serum by the patient's fingers) on various other parts of the body. Small acuminate papules are also very characteristic, and not less so are the scratch marks, often accompanied, especially in children, by wheals like those of urticaria. In severe cases of scabies the dermatitis may be intense, both hands and arms swelling as if with phlegmonous erysipelas; or arms, hands, legs, and feet may be the seat of weeping, raw surfaces like those of eczema madidans; the lymph-glands of the axillæ and the groin become swollen and painful, and the excessive itching is at last replaced by the smarting and tingling of acute dermatitis. More often, especially in children, the pustules resemble impetigo or ecthyma, and form as they dry up thick scabs and crusts. In chronic cases—for unhappily we often see scabies which has lasted for weeks and months without detection, and has been therefore ineffectually

* Compare the figures given by Dr Bristowe in his 'Practice of Medicine' with those of M Hardy in his recent 'Traité des Maladies de la Peau." The writer's experience would be that short straight runs are more frequent than would appear from either of these figures.

treated—the skin becomes thickened, indurated, hard, scaly, and fissured, resembling the condition of the more chronic forms of dry eczema. Bullæ as large as those of pemphigus are less frequent lesions of scabies, but are not uncommon in children. A case was figured in the 'Guy's Hospital Reports' for 1877. In fact, we may say that any of the inflammatory lesions of eczema, erythema, urticaria, pemphigus, impetigo, ecthyma, and lichen, may be more or less perfectly represented. The large, flat, and discrete papules of prurigo, the imbricated scales of psoriasis, and the thin, dry, abundant squames of pityriasis rubra are never simulated by scabies.

Localisation.—The acarus itself infests the thin skin of the hands between the fingers, the flexure of the wrist, particularly its ulnar side, the flexor surface of the front of the forearm less frequently, the foot and ankle occasionally, the axilla and the groin, the genital organs, the inner part of the thigh, and the fold of the nates. But the lesions indirectly caused by its presence have a far more extensive though perfectly definite range; in fact the local distribution of scabies is so well marked that in a majority of cases a glance is sufficient to identify it. The inflammatory lesions are always present on the hands, except occasionally, when the patients are engaged in some handicraft which leads to the constant immersion of their hands in oily or strong-smelling substances or in metallic solutions, and forbids the development of the parasite. The same result is often seen in private patients, where the hands escape owing to the frequent use of soap and nail-brush. With these exceptions, which are important for diagnosis, the fingers and ulnar side of the wrist may be said to be the favourite seats of scabies as they are of the acarus. Some lesions will almost always be found upon the prepuce, and the inflammation usually affects not only the thin skin of the genitals, but that of the lower part of the abdomen, at least as high as the umbilicus. The whole of the forearms is very liable to be affected, and the eruption is more general than is the case with eczema. The axillæ seldom escape altogether. The buttocks are almost always more or less affected, and in children nearly constantly, especially at the gluteal fold, but the perinæum and the sacral region usually escape. The toes, feet, and ankles, especially the inner ankle where the skin is thinnest, are very frequent seats of the dermatitis of scabies; less often the knees; the whole of the inner side of the leg and thigh may share in the inflammation. The back, shoulders, and chest are but little affected, the thick or hairy skin being apparently less favourable to the parasite. The neck, face, ears, and scalp almost invariably escape, in striking contrast to the frequency of eczema and impetigo in these parts. The only exceptions are in children, of which every experienced dermatologist must have noted occasional cases. Why the skin of the face is shunned is hard to say. It is not more exposed to the air than the hands, it is as thin and delicate and vascular as that of the abdomen, but for some cause it is shunned by the acarus. May it be that the large sebaceous sacs and thick cutis with thin cuticle which are characteristic of the face, as of the shoulders, the chest, and the scalp, furnish a fatty secretion which repels the invader?

It has often been remarked that a line drawn across the waist and elbows of a man standing in "the first position" will have below it the regions of scabies acarorum, and above it those of prurigo pedicularis.

In children, localisation of scabies is much less strict than in adults, as we found to be the case with eczema and psoriasis. In them only do we ever find the face affected; in them the hands frequently escape, and runs

are found as well or better on the ankle or in the skin of the sole; in them the trunk, and particularly the nates, are often more affected than the limbs.

Diagnosis.—This depends on a recognition, first, of the characters of the dermatitis, next of its very constant localisation, and thirdly of the cuniculi, the ova, or the acarus. The general facies of the disease is so characteristic that nine out of ten cases will be recognised in a moment when the patient is stripped, but in private practice anyone may be thrown off his guard who is accustomed to diagnose by probabilities rather than by facts. As Sir William Gull said, there are three diseases which we all sometimes overlook,—phthisis and syphilis and itch.

Where the inflammation has completely obscured all trace of acari, the existence of the itch-mite may be proved by removing the crusts, boiling them in solution of potash or soda, and allowing the dissolved mixture to stand in a conical glass. On decanting and removing the deepest layer with a pipette, fragments of the chitinous skeleton may be recognised.

It need not be said that scabies is always *contagious*, and its occurrence in an entire household often leads to its recognition. It is remarkable, however, how cases may remain isolated, and we must remember that impetigo and prurigo, not to mention variola and varicella, may also be contagious. The mode of transference is not always easy to follow; direct contact of hands is probably one method; often the ova are conveyed by clothes or other articles of constant use. Bedfellows seem particularly liable to infection. There is no doubt that scabies is frequently a venereal disease, the acarus having first invaded the genital organs.

Treatment.—Experience long ago discovered that sulphur is good for the itch; it is an effectual poison to the acarus, and all we need is the best method of applying it.

The general practice is inunction of unguentum sulphuris into the affected parts, especially those which are the chief seat of the acarus. The colour of the application may be disguised, but its smell is always unpleasant. Sulphur lotions or sulphur fumigations may be substituted, but neither are so effectual. The best method is for the patient to rub the ointment well in every night, to lie in merino clothing all night, and next morning to wash with hot soap and water, and apply a little dilute ointment to the most irritable parts.

A rapid cure may be effected by first rubbing the skin with soft soap so as to remove crusts and epidermis, and then using thorough sulphurous inunction. In this way patients are cured in a few hours at St Louis on a large scale, and their clothes meanwhile are baked and washed. This last precaution is important, since otherwise the patient may readily reinfect himself from his own clothing. With private patients the disease rarely gains such extension by neglect as to be severe, and its cure is usually quick and easy.

It may, however, happen that the sulphur ointment is itself too irritating; so that, although it kills the acarus, it perpetuates or sets up a fresh and even more severe dermatitis. One often sees these cured but over-treated cases of scabies, and all that is necessary is to recognise their nature.

With children, diluted ointment, two to one, or equal parts,—in infants with much dermatitis, one to two,—are the best proportions, the dilution being made with benzoated lard or with zinc ointment.

In slight cases, especially in children, balsam of Peru is a pleasant and generally an efficient parasiticide.

ERYTHEMA

AND ITS ALLIES

Definition of the group—Its characters—The anatomical lesions—Course—Locality—Symptoms—Ætiology—Symptomatic and traumatic erythema—Pernio and pernio-like inflammations—Varieties (1) *Erythema multiforme—Herpes—Erythema iris—Herpes gestationis—*(2) *Erythema nodosum—* (3) *Urticaria—Urticaria pigmentosa—Treatment of erythematous affections.*
RASHES PRODUCED BY DRUGS.—*The erythematous rash of Copaiba—Bromides and Iodides — Belladonna—Opium — Quinine — Salicylates —Arsenic—Mercury.*

WE have hitherto considered diseases which, though differing from one another in many particulars, are all examples of chronic superficial dermatitis; never leaving scars, chronic in course, apt to return, and accompanied with more or less decided irritation.

We now pass to diseases which also form a natural group, though the line is perhaps more difficult to draw. They also are *superficial inflammations* of the skin and therefore leave no scars, but they are *acute or subacute* in their course. Moreover, their lesions are usually slight and evanescent, and although occasionally they produce one as conspicuous as a bleb, yet this is quite exceptional, and none of this group is attended with pustules or crusts. Perhaps the most conspicuous anatomical character is the presence of inflammatory *œdema*, and this is often accompanied with slight *hæmorrhage*. The sensations accompanying them are usually *smarting* rather than itching.

The previous group of diseases, eczema and lichen, scabies and psoriasis, are usually chronic, and even when their onset is acute they run an indefinite course afterwards. They are more or less closely related to local irritation.

The present group has an acute or subacute, self-limited course, often recurrent but seldom or never chronic. It has no relation to cutaneous irritants but may often be traced to gastric or other internal causes.

Pemphigus forms a natural link between the true tetters—eczema, lichen, and psoriasis, and the erythematous affections. Indeed, while Hardy classes it decidedly with the dartres, Hans v. Hebra places it in close relation to erythema multiforme.

Erythema is the name which has been given to some affections belonging to this group, and it may be conveniently extended to the whole.*

Willan classed erythema with roseola, urticaria, scarlatina, rubeola and purpura under the title "exanthemata," the general character of the order being hyperæmia of the skin without further lesion. Subsequent writers have called a mere hyperæmia "rose-rash" or roseola, while the word erythema, or, as Hebra calls it, *erythema exudativum*, has been confined to a rose-rash with palpable inflammatory exudation, diffuse or forming pimples.

* This word, denoting "redness" of the skin, was used in classical Greek, either as ἐρύθημα προσώπου or alone, for a blush. It was also used by ancient writers as almost, if not quite, synonymous with ἐρυσίπελας.

Hebra included also the so-called tubercular and nodose species of Willan's genus erythema, and invented a convenient term, *erythema multiforme.*

But in truth we have no need of a special title for mere hyperæmia, that is, dilatation of the blood-vessels without inflammatory exudation, such as follows division of a vaso-motor trunk in an animal. A mere blush is always a physiological phenomenon. Clinically, hyperæmia is always inflammatory. Even the erythematous eruption of scarlatina, of measles, or of enteric fever can be proved by its course and sequelæ to be in each case true dermatitis. Bateman himself remarked that the efflorescence to which Willan appropriated the title of roseola is of little importance practically, and quotes the dictum of Fuller in his 'Exanthematologia' that it is "rather a ludicrous spectacle than an ill symptom."

We must recognise two meanings of the word "erythema" just as we are obliged to recognise two of the word "eczema." We saw that eczema is, as Hebra proved, a common superficial dermatitis, which has reached the stage of visible and usually serous exudation; but we saw also that the most important peculiarity of the disease eczema is that it is not traumatic, not called forth by ordinary irritants, and not limited by their action. An artificial or traumatic eczema is therefore for practical purposes better refused the name. Accordingly we added to the definition of eczema as a disease the character of being *idiopathic*, with its own peculiar distribution and course.

In the same way "erythema" may be defined, and has been used by Hebra and other authorities to denote the slightest form of dermatitis in which the classical signs of redness, heat, and pain are accompanied by little or no perceptible swelling. The irritation of a mustard plaster, for instance, will in most persons produce such a typical erythema, the scorching of the sun does the same, and if the skin be more than usually delicate and the mustard or sun more than usually strong, what was an "erythema" becomes an "eczema." It would be better if the term "superficial traumatic dermatitis" were used for both stages of the inflammation; or we might speak of the earlier as an erythematous and of the latter as an eczematous or weeping dermatitis.

Just as we define the disease eczema by its clinical and pathological features apart from its mere anatomy, so we can define the group of affections which we have called erythematous. These are distinct from the erythematous stage of common dermatitis, and though often strictly "erythematous," sometimes exhibit other lesions, to which Hebra's adjective "multiform" very well applies.

1. The characteristic *anatomical lesion* is a rose-rash, resembling the first degree of traumatic dermatitis, that is to say *injection* of the surface; sometimes with obvious general *œdema*, sometimes with circumscribed œdema, forming *wheals*, and sometimes with *papules*—which are distinguished from those of eczema by not developing into vesicles, from those of lichen by their bright colour and transitory duration, from those of impetigo and prurigo by never becoming pustular, and from those of psoriasis by never becoming scaly. In certain rare forms of erythema separate *bullæ* are formed which may simulate those of pemphigus. The rash is usually followed by a slight branny *desquamation*. It will be seen that, after all, the multiformity of the lesions of erythema is less than that of the lesions of eczema.

2. Whatever the nature of the lesion, the *exudation* is of a watery

rather than a corpuscular nature, so that œdema, diffused or circumscribed, is its characteristic, in contradistinction to the sero-purulent or purulent vesicles and pustules of eczema and scabies. Moreover, along with the hyperæmia and œdema there is very apt to be a certain amount of escape of blood-corpuscles, an event which never occurs in eczema except as the result of direct injury, as by scratching. The result of this hæmorrhage is sometimes so marked as to give the title "purpura" to the eruption. Willan and Bateman rightly included *purpura urticans* with erythema among the exanthemata, although other kinds of purpura are altogether distinct from any form of dermatitis, and are only parts of a general hæmorrhagic condition. The result of the hæmorrhage is to leave bruise-like pigmentation behind, so that this when present is very characteristic of true erythema.

In these characters, as in some others, and especially in the fact of the occasional occurrence of bullæ, the erythematous group bears a closer relation to pemphigus than to any other of the chronic forms of dermatitis belonging to the so-called dartrous group, but if one attempts to include pemphigus as an erythema its course and treatment forbid the conjunction. Dr Hansvon Hebra, however, whose classification differs widely from that of his father, unites under the title "Angioneurotic affections of the skin" the erythematous rashes of infectious diseases, the rashes produced by drugs and poisons, and, thirdly, the essential erythemata, together with pemphigus and acne rosacea.

3. The *course* of erythema is subacute, that is to say it begins quickly, sometimes with slight febrile symptoms, and does not last indefinitely. Even when its course is comparatively chronic it will be found that the protracted disease is really made up of a series of outbreaks, which may sometimes run into one another, but always preserve a recurrent or intermittent character. No erythematous disease ever acquires the chronic, stable, and inveterate stamp of eczema, lichen planus, psoriasis, or pityriasis rubra.

4. The *locality* of erythema is much less definitely marked than that of psoriasis or of eczema. On the whole it is symmetrical, sometimes accurately and exclusively so, but there are frequent exceptions to the rule.

The favourite localities are, first, the extensor surface of the forearms and legs, especially the back of the hand, wrist, and ulnar side of the forearm, the dorsum of the foot, and tibial side of the shin;* secondly, the face, cheeks, and neck; thirdly, the chest and abdomen. The back of the trunk, the buttocks, thighs, and upper arms are much less frequently affected; while the scalp, the flexures of the joints, the palms, and the soles are scarcely ever attacked by erythema.

5. As a rule smarting and tingling are the *symptoms* which accompany erythematous eruptions, while severe pain and itching are rare. Local tenderness is more marked than in eczema. Sometimes, and especially when wheals are present, the irritation is considerable, though never comparable to that of chronic eczema, scabies, or prurigo.

6. The erythematous rashes do not spread. They appear simultaneously at different spots, and fresh patches appear which may occasionally unite, but we never see the affected part of skin gradually enlarge its borders—the characteristic course of eczema and psoriasis.

7. Of the *ætiology* of erythema we in most cases know nothing. The

* Although the true homology of the tibia is undoubtedly with the radius and not with the ulna, yet the tibial aspect of the skin, from its having no underlying muscles, agrees pathologically with the skin covering the subcutaneous surface of the ulna, just as pathologically the patella answers to the olecranon, and the second metacarpo-phalangeal to the first metatarso-phalangeal joint.

lesion can, as above explained, be produced by moderate irritation, the diffused forms by heat or friction, those with wheals by a lash, or by the poison of the stinging-nettle, the hairs of certain caterpillars, and the thread-cells of certain anthozoa. But in the non-traumatic, idiopathic, or "true" cases of erythema, the eruption can, in striking contrast to those of eczema and psoriasis, be in most cases traced to some *internal* disorder. In other words, erythema is usually *symptomatic*. The most striking instance of this is the erythematous rash produced by copaiba and by certain articles of food. Many other cases are dependent upon *dyspepsia*, others, again, upon *rheumatic fever*. This is particularly true of E. nodosum, urticaria, and the hæmorrhagic form of erythema known as *Peliosis rheumatica*.

Moreover, one may fairly adduce in this connection the fact that the symptomatic early rash of syphilis, the exanthema of scarlatina and of measles, and the occasional prodromic roseola of smallpox, enteric fever, and cholera, all belong to the erythematous type.

8. Erythema occurs most commonly in children and young adults; it is comparatively rare after forty. Among persons past their prime it is less uncommon in women than in men. In these, as in so many other points, we observe a marked contrast to eczema and psoriasis, and a resemblance to pemphigus and also to rheumatism.

Before entering on the varieties and treatment of erythema proper, one must say a word of the traumatic dermatitis of slight degree which is still often called erythema. Thus *intertrigo* mentioned above under eczema, is classed under erythema, as it was by Willan, Hebra and Neumann.

Common *chilblains* are a good example of chronic inflammation from the effect of cold and feeble capillary circulation of an erythematous anatomical type. The occasional bullæ which end in broken chilblains are not unlike those of erythema iris. Such chronic erythematous dermatitis may be seen not only in the fingers and toes of children and young persons during winter but also on the ears and nose of a subject in whom, whether from central or peripheral causes, venous stagnation is apt to occur in the parts of the body most distant from the heart.

This kind of chronic venous congestion, which on the one hand may be quickened into chronic subacute dermatitis, and on the other may lead to hypertrophy, is seen in the blue and swollen ears, cheeks, and fingers of children with chronic bronchitis, and especially with bronchiectasis; in persons with chronic affections of the heart, especially of a congenital kind; in those patients, especially women, who habitually suffer from cold feet, and what they rightly call a languid circulation. It also is seen with somewhat different characters in the red, swollen, and irritable nose and cheeks which accompany dyspepsia, particularly, though not exclusively, in alcoholic dyspepsia, and is known as "acne rosacea" or gutta rosea.

Lastly, a not dissimilar condition of cyanosis and chronic venous congestion with œdema is seen in those remarkable cases of symmetrical gangrene of the extremities which have from time to time been observed, and which are now spoken of as Raynaud's disease* since that physician

* A striking case of this remarkable condition occurred in Guy's Hospital several years ago. A boy about twelve years old had ulcerative endocarditis with embolism. This led to gangrene of the fingers and toes, and intermittent hæmaturia, with great pain and fever. He went out with the mutilated members healed, and well except for a cardiac bruit. See also a case of Prof. Billroth's in the 'Wiener med. Wochenschft.,' 1878, reported in the 'Lond. Med. Record' of that year, p. 343.

called attention to the more severe cases connected with disease of the arteries, with cyanosis and sometimes with hæmatinuria (*cf. supra*, p. 590).

The erythemata which are symptomatic of measles, scarlatina, enterica, rubeola, as well as choleraic roseola, and that which sometimes precedes the characteristic rash of smallpox,—all these have been described in the first volume of this work.

The erythematous rashes which follow the administration of drugs will for convenience be considered together at the end of this chapter.

There remains a group of skin affections which agree in the general characters of anatomy, course, and natural history described above, which are not traumatic, nor secondary to diseases of the vascular system, and which are not symptomatic, either of febrile diseases or of what the continental writers call "intoxication" with drugs or poisons. These we may style idiopathic essential erythema. Their common characters have been already sufficiently explained. It remains to point out the principal varieties which they present.

1. *Erythema multiforme.*—Simple or ordinary erythema, erythema papulatum, erythema exudativum.

The commonest kind of erythema is that which consists in general hyperæmia with œdema of the skin, a diffuse dermatitis which may either spread over a large surface with indefinite edges, or, as is more frequently and characteristically the case, occurs in patches with defined edge. On careful examination small papules may be often distinguished scarcely rising above the level of the skin as in the eruption of measles; sometimes these are well marked enough to deserve the title *erythema papulatum*, but this is comparatively rare, and most lesions of the skin which receive this name are probably either traumatic dermatitis or an early stage of papular eczema. Large, firm, and persistent papules such as occur in prurigo are never seen in true erythema. The inflamed patches have usually a very short duration; they may disappear in a few hours (*Erythema fugax* of Willan) and be succeeded by others, but if they persist for a day or two they may form rings which have been specially described as *Erythema annulatum* (*E. circinatum* of Willan), or *Roseola annulata*. When closely set several of these rings unite, and a sinuous reddish band is produced which has been named *Erythema marginatum* or *E. gyratum*. Finally, the redness fades, the œdema subsides, and may leave no trace behind. If there is desquamation it is very slight and furfuraceous; more frequently a slight amount of pigment marks the seat of the eruption.

The favourite localities are the back of the wrists and forearms, the legs, and the face and neck; sometimes the trunk, but very rarely the thick parts of the skin or those covered by hair.

Associated with rheumatic fever is often observed *Erythema annulatum*.

Erythema leve is a common dermatitis which is apt to appear upon the tense skin of dropsical parts, and may go on to deep dermatitis and sloughing. It is not uncommon as the result of acupuncture or of tapping, and is allied to traumatic erysipelas.

2. *Vesicular and bullous erythema.*—The exudation of erythema, instead of being a somewhat deep diffused œdema, sometimes appears in superficial collections of serum. These when small are called vesicular erythema or "herpes;" when large, "erythema bullosum."

Herpes,* the common Latin term for an eruption of the trunk, in contradistinction to porrigo or an eruption of the head, was limited by Willan to vesicular eruptions which he distinguished from the vesicles of smallpox and chicken-pox, from sudamina and from the inflammatory vesicles of eczema. His species of herpes were as follows :

Herpes zoster or zona, an eruption erythematous, it is true, in its anatomy and course, but which is so demonstrably connected with nervous disorder that it is rightly separated from all other forms of dermatitis, and will be described in the present volume among diseases of nervous origin.

Herpes circinatus, which we shall afterwards describe as the form assumed by ringworm when it affects the body, is a parasitic disorder, and is now classed with *Tineæ*.

Willan and Bateman's remaining species are *Herpes phlyctænodes* of uncertain seat, called *H. labialis* or *H. preputialis* when affecting the lips or the foreskin respectively, and *H. iris* when found on the back of the hands or the instep.

These from their course and natural history may be well included in the general group of erythemata. This was, indeed, to some extent admitted by Hebra and even by Rayer before him.

Herpes labialis or *facialis* consists of a little group of vesicles upon a red patch of skin which appears almost suddenly, most often upon the upper lip. In a day or two the clear, pearly contents become somewhat turbid and puriform, and dry up into a thin brownish crust which speedily falls off and leaves no trace behind. The vesicles entirely differ from those of eczema or ordinary traumatic dermatitis by their large size, by their not running one into another so as to form a weeping surface, by their acute course, and by the sharply limited edge of the patch. They also are unattended with itching or pain and never consist of pure pus like the eruption of impetigo or scabies. Moreover, they are always symptomatic of some internal disorder, most characteristically perhaps of acute lobar pneumonia. Many persons are liable to such patches of herpes, either on the lips, or less frequently on other parts of the face, when they are attacked by acute catarrh. Bronchitis, catarrhal pneumonia, whooping-cough, asthma, are rarely accompanied by herpes. Sometimes the eruption appears to follow a rigor even when this symptom does not prove the precursor of pneumonia or catarrh.†

This curious eruption has clearly little or no connection with eczema and its allies, nor can we link it with zona, for it frequently recurs, it is not unilateral, it does not follow the course of a nerve, and is unattended with pain. Its superficial character, sudden onset, and rapid course agree with the erythematous group as here defined, and the fact that it is symptomatic of internal disturbance and usually of irritation of a mucous tract completes the analogy.

Herpes preputialis when no longer left, as Willan placed it, and as Hebra was content to leave it, among vesicular inflammations, is difficult to classify. Hardy is even driven to the untenable assertion that it is nothing but local

* The word ἕρπης is derived from ἕρπειν. "Herpes dicitur eo quod videtur ἕρπειν, quod est serpere per summam cutem, modo hanc ejus partem modo proximam occupans." From the same creeping progress the disease was, according to Bateman, called *formica* by the Arabs.

† See an interesting autobiographical account of a case of the kind by Mr Symonds in the 'Clinical Transactions' for 1884, p. 60.

vesicular eczema. The rapidity of its onset and course, the superficial lesion, the patches, the absence of notable irritation or pain,* all point to its close connection with erythema, while its occurrence at the orifice of a mucous tract and the anatomical lesion bring it into still closer relationship with herpes labialis. The chief difference is that, occurring as it usually does on the inner side of the prepuce or glands, the vesicles are broken almost as soon as they form, and very superficial ulcers take the place of scabs. The condition is exactly like that of a vesicular eruption on the tongue. Like herpes labialis, it often recurs in the same patient; like it also it is often symptomatic of inflammation or stricture of the urethra, although it does not seem to be produced by cystitis, and certainly does not follow inflammation of the kidney as labial herpes does inflammation of the lung.†

Herpes iris is a rare and remarkable form of eruption well described by Willan, which is unmistakeably erythematous in its nature and is better named *erythema bullosum*, or *erythema iris* or *iris*. It occurs sometimes as a single, sometimes as two or more rose-coloured patches with all the characters of erythema, almost always upon the back of the hand, the wrist, or extensor aspect of the forearm, more rarely on the corresponding part of the foot and ankle. It rapidly becomes annular, but before the ring is faded the patch of erythema reappears in the middle, and may thus be surrounded with one or by a repetition of the process by two or even three concentric rings. This is the condition described by authors as erythema iris, but almost always the irritant patch becomes the seat of a large single vesicle or a group of smaller ones exactly like those of one of the patches of herpes labialis or herpes preputialis. The surrounding ring may exhibit similar vesicles, or they may be more or less abortive, so that one might often question whether if we adopt the anatomical nomenclature we should describe the lesion as erythematous or bullous or vesicular. In its most striking form with a single large tense bleb like one of pemphigus, surrounded by vesicular circles, the whole patch as large as a crown-piece, it is one of the most remarkable of eruptions. The inflammation is very superficial, produces little pain or irritation, and after forming thin scabs passes off after a few days, leaving more or less pigmentation yet not a trace of scar behind. Partly the resulting pigment and partly the rosy red of the rings, the pearly grey of the vesicles and the more or less yellowish contents of the older bullæ seem to have combined with the bow-like form to give the title iris. The course of this curious disease, its superficial character, and its locality, all make it unmistakeably erythema, as also the fact that it occurs almost exclusively in young persons; but it appears to be symptomatic of nothing.

Iris is not, however, the only bullous form of erythema. Beside the form associated with imperfect circulation above described (p. 930) which is known as *pernio bullosa*, other cases of bullous erythema have been recorded by Dr Duffin ('Pathological Transactions,' 1875), by Dr Crocker and Dr Frederick Taylor ('Clinical Society's Transactions,' Feb. 25th, 1881), and the writer reported two typical cases in the 'Guy's Hospital Reports' for 1880 (3rd series, vol. xxv, p. 211). The best account of them is given

* Pruritus may, however, be considerable at the beginning of the eruption.

† The maintainer of a thesis might, however, maintain that herpes labialis does not follow ordinary inflammation of the lung, and that pneumonia is not an inflammation of the lung at all, and produces herpes only as an acute pyrexia ushered in by a rigor. The chief practical importance of preputial herpes is its diagnosis from a soft chancre.

by Mr Hutchinson in the 29th of his 'Clinical Lectures' on certain rare diseases of the skin: "Of some Peculiar Eruptions allied to Chilblains."

*Erythema gestationis bullosum.**—There is a remarkable and rare affection which has been named as a species of pemphigus, of herpes, or of erythema, or has been included under the title "Hydroa." Its pathological alliance appears to be with the form of erythema which depends on ovarian irritation, but the existence of bullæ makes it liable to be confounded with pemphigus.

It occurs only in women during pregnancy. The bullæ, vesicles, and vesiculo-pustules appear in abundant crops over the trunk, and often on the face and limbs also. Pruritus is marked. There is more or less constitutional disturbance, and sometimes the temperature rises high. The clinical aspect is therefore serious and occasionally alarming. But the result appears to be always favourable. The disease is cured by delivery.

We have had one instance—and others are on record—in which this remarkable form of pemphigus or bullous erythema appeared again and again in successive pregnancies.

Cases of "herpes gestationis" have been carefully described since Chausit and Hardy, by Dr Liveing, Dr Bulkley, and other observers in this country, on the Continent, and in America. The general features are very uniform, and there is no doubt of the reality and distinctness of the disease, but its true nature, pathological relations, and prevention or treatment are still obscure.

3. *Erythema nodosum.*—This curious affection was well described by Willan and Bateman, and subsequent authors have added little to their account. It occurs "in large oval patches, the long diameter of which is parallel with the tibia, slowly rise into hard and painful protuberances, and as regularly soften and subside in the course of nine or ten days, the red colour turning bluish on the eighth or ninth day as if the leg had been bruised." In this form of erythema the anatomical lesion is especially characterised by œdema; the spots do not itch, but are somewhat painful and very tender, more so than any other of the erythematous group. There is almost always not only deep venous congestion of the typical erythematous rose tint exaggerated by its position on the legs, but there is almost always a slight indication of actual hæmorrhage. Probably from this cause the pigmentation, which like other forms of erythema it is apt to leave behind, is much deepened by the chemical transformations of the effused hæmoglobin which are familiar in a bruise.

The locality of erythema nodosum is, as Willan says, most frequently over the tibia, but it is not confined to this part, for it may be seen on the ankle or the calf, and it is not uncommon over the corresponding surface of the ulna. It is usually symmetrical and often affects the whole extensor surface of both forearms and both legs. It is very rare in any other part.

It has a slower course than most kinds of erythema, but like them is prone to recur. Willan and Bateman, and also Green in his 'Practical Compendium,' state that erythema nodosum only affects women, but Plumbe in 1824 ('Practical Treatise on Diseases of the Skin') notes its occurrence in children, and it is not unfrequently seen in men, but almost always in those subject

* *Synonyms.*—Hydroa (in part)—Pemphigus uterinus, hystericus v. pruriginosus—Herpes gestationis—Impetigo herpetiformis—Dermatitis herpetiformis.

to hysteria, chorea, or other female disorders, that is in boys under or about the age of puberty.

It occurs very frequently in those who have suffered from *rheumatic fever* (see Dr Thos. Barlow, 'Brit. Med. Journ.,' Sept. 15th, 1883, p. 511). Dr Cæsar Boeck has also published a monograph on this point.

Dr Stephen Mackenzie brought before the Clinical Society in April, 1886 (vol. xix, p. 215), more than 100 cases of erythema nodosum collected from the four largest hospitals in London. Ninety patients were females, and 18 males. Only 25 were over thirty years of age, 30 were between twenty and thirty, 39 between ten and twenty, and 14 were children under ten. In 17 cases there was also past or present rheumatism (acute in 13, subacute in 4), besides about as many more in which the existence of true rheumatism was asserted or probable. There was a cardiac murmur in 13 cases, in only 2 of which there was history of rheumatic affection of the joints.

The course, the lesion, the œdema, the hæmorrhage, the locality, the subjects of this affection are all typically erythematous.

4. *Urticaria.**—Willan rightly placed urticaria in close relation to erythema. Almost all subsequent writers have followed this indication; and if convenience did not forbid innovations, it might be called "erythema pomphosum," for the characteristic lesions are *pomphi*—wheals, *i. e.* raised flat white patches sometimes surrounded by an erythematous blush. Their histology is that of acute inflammatory œdema of the cutis which fills the lymph spaces and expels blood from the venules. The exudation takes place very rapidly and may be called forth either by a mechanical or by a chemical irritant as in the wheals produced by the nettle (*Urtica urens*) from which the disease receives its name. In persons liable to the affection it can be produced by the finger only drawn across the skin, so that it is possible to write characters in raised wheals. This last has been defined as "factitious" urticaria. The anatomical lesion has therefore its counterpart in those traumatic wheals produced in any skin by the sting of the nettle or the stroke of a whip, and produced with very slight irritation in susceptible subjects. The relation of such traumatic urticaria to the idiopathic disease precisely corresponds with that which was expounded at some length between common superficial dermatitis from the sun or other irritant and idiopathic eczema (p. 873), between prurigo senilis a pediculis and idiopathic prurigo of Hebra (p. 904), between erythema congestivum et bullosum and chilblains (p. 930).

Beside the well-marked oval or linear wheals of ordinary urticaria, we often see the lesion in the form of small round patches or in large white plateaux formed by the coalescence of several smaller ones. Both these forms are frequently produced by nettles. We may also include as essentially of the same nature the large, flat, white papules which are obviously distinct from those of ordinary eczema, and which have been described as *strophulus albidus*, and also not unfrequently under the name of *infantile prurigo* and *lichen urticatus*. These papules are distinguished by rising rapidly, and by following, not causing, pruritus; for they are the result and not the occasion of the patient's scratching. They are most often seen in infants, but may be observed along with more obvious wheals in

* *Synonyms.*—Nettlerash—Cnidosis: including lichen urticatus, purpura urticans, and much of strophulus.—*Fr.* Urticaire.—*Germ.* Nesselsucht.

ordinary cases of adult urticaria. Very rarely the wheals last, so as to produce a chronic condition (*U. perstans*).

The *distribution* of nettlerash is less definite than that of other forms of erythema, and indeed of most other cutaneous affections. We do not observe any predilection for the erythematous regions, the extensor surface of the forearms and legs. It is quite as common on the back and trunk generally as on the limbs; the only parts it avoids are the scalp, face, arms, and soles. It is not symmetrical. The mucous membrane of the mouth is occasionally affected.

Urticaria is abrupt in origin and sometimes acute in *course*, but often persistent and obstinate in successive attacks.

Of all forms of erythema, urticaria is the most irritable, the severity of the itching being comparable to that of eczema, scabies, or prurigo. There is no pain or smarting, and no subjective symptoms except from the restlessness and sleeplessness which it occasions, especially in children. It is most frequent in them or in young adults, but is not confined to any age. According to Dr Liveing, it sometimes alternates with neuralgia of the same parts.

The *ætiology* of urticaria is uncertain. As above explained, it is often purely secondary to some local irritant as pediculi, or complicates a previously existing malady as prurigo, and is probably always aggravated by the patient's scratching. Its close alliance with erythema is shown not only by sometimes alternating with it, but also by its following precisely the same kind of gastric disturbance, both in the most marked forms which are the direct result of drugs or of poisons, and in the less evident cases associated with ordinary dyspepsia. Like erythema again it is, according to general experience, a not infrequent complication of rheumatic fever.

*Urticaria pigmentosa.** — A singular and rare form of skin disease belonging to the erythematous type, but as chronic as erythema nodosum, was first described by Mr Edward Nettleship, and again by Mr Morrant Baker and the late Dr Tilbury Fox, in the 'Clinical Society's Transactions,' vols. viii and x. A case in a child of two years old was shortly described in the twenty-fifth volume of the 'Guy's Hospital Reports,' 3rd series, pp. 212, 213.

It has received several names, among others the uncouth and misleading term Xanthelasmoidea, but Dr Sangster's proposed title, Urticaria pigmentosa, is now generally accepted.

It is an erythematous eruption with occasional wheals and considerable yellowish pigmentation lasting for an indefinite period, though its chronic course is probably always made up of more or less distinct subacute attacks. It affects the back, and trunk generally, rather than the limbs, and the face very little. The characteristic buff colour always follows the rose-rash.

An excellent account of this affection is given by Dr Colcott Fox in the 'Med.-Chir. Trans.' for 1883, where nineteen recorded cases are tabulated, to which Dr Crocker has since added one ('Clin. Trans.,' vol. xviii, p. 12). See also Dr Cavafy's article in 'Heath's Dictionary of Surgery.'

Dr Paul Raymond has written a valuable monograph on this curious malady, in which he has collected sixteen cases observed in England, two

* *Synonyms.*—Urticaria perstans pigmentosa—Xanthelasmoidea—Erythema tuberculatum—Permanent erythema.

in America, eight in Germany, and four in France. All these cases occurred in children under two years old, often within a fortnight after birth. The majority of the infants were boys.

Microscopical examination of the affected skin by Dr C. Fox has shown that the lesion is truly a wheal, the tissue of the corium being opened out by œdema. Pick had found minute hæmorrhages in another case.

The affection recurs again and again, but as the child grows older gradually ceases, and the pigmentation still more gradually fades.

In an exceptional case recorded by Lewinski it was still present in a lad of eighteen. In one case of erythema under the writer's care in a woman of thirty-two, a condition which might be called E. perstans had been present for four years on the trunk, and was followed by decided pigmentation.

Treatment of erythematous affections.—The erythemata are seen in their true relationship from a practical point of view. They are none of them contagious, they are none of them attended with serious consequences, they are mostly indicative of some other primary disorder, and they are rather to be palliated by local applications or indirectly cured by treating their internal cause when discovered, than met by a specific plan of treatment. In particular it may be said that they are either unaffected or aggravated by arsenic, and this is one of the points which separate them from pemphigus.

In many kinds of erythema, especially symptomatic herpes and iris, no treatment is needful.

The local treatment of the erythemata consists in the astringent and sedative applications described at p. 884; although the surface is dry, it is found by experience that lotions in most cases answer better than ointments. Goulard's wash, evaporating lotions, spirit and water, eau de Cologne, hydrocyanic acid well diluted, or solution of borax are the best local applications. When urticaria is severe and these means fail, chloral hydrate may be used locally in solution or chloroform and ung. cetacei (℥x—xx ad ℥j). Warm baths should be avoided, as also excessive heat and perspiration; tepid water is better than either cold or hot. The patient should be urged to stoical abstinence from scratching; tepid bathing or continued, steady pressure will be found to relieve the intolerable irritation of urticaria without aggravating it afterwards as scratching always does.

For the painful swellings of erythema nodosum, strong lead lotion gives most relief, or lead and opium. Collodion painted over and allowed to dry is often useful, or alum, tannic acid, or other astringent remedies may be used with advantage, or the affected part of the leg may be painted with a strong solution of nitrate of silver. Ointment containing zinc or zinc and lead may be applied to the herpetic and bullous forms of erythema.

Internally, our first care should be to relieve the gastric disorder which often accompanies common congestive or papular erythema, most often by discovering certain articles of food to which it is due. Salt fish, pickles, preserved fruit in the form of jams and crystallised sweetmeats, pork, sour or otherwise inferior wine, malt liquor, stone fruit, and even strawberries—any one of these may in certain persons excite erythema or urticaria, and it is said that eggs and other articles of diet may occasionally act in the same way in certain persons. Those first on the list should be strictly forbidden. Of all kinds of food, lobsters and crabs, and by a curious coin-

cidence mussels and other molluscs united with them under the title of "shell-fish" are the most frequent causes of erythema or urticaria, and they are the most severe in their effects. Oysters are harmless. Many drugs have a similar result, copaiba being probably the most effectual.

If the eruption continues after its supposed cause is removed, or if we are unable to discover any cause of disorder, such remedies as bicarbonate of soda with gentian or calumba or chiretta or a few drops of liquor potassæ in peppermint or cinnamon water should be prescribed. Where there is evidence of gastritis, bismuth is a most valuable remedy, given either in powder or thus: ℞ Bism. subnitr., sodæ bicarb., pulv. tragac. co. āā gr. x., aq. chloroform. sive menthæ pip. ʒj. M.; to which ten or twelve drops of solution of morphia may be added if the pain is severe. With flatulent disorder, thymol, creosote, or carbolic acid in the form of pills are often the most effectual mode of treatment. In the more atonic forms pepsine given before meals is found practically useful, notwithstanding our physiological doubts; and occasionally dilute mineral acids with nux vomica or bitter infusion will be more valuable than anything else. Gentle saline laxatives taken before breakfast in a large draught of warm water are almost always indicated; and, for women especially, a pill containing aloes or rhubarb, taken before a late dinner or on going to bed, is a useful adjunct. In many patients occasional doses of blue pill are of unmistakeable value.

In cases of erythema nodosum and in other forms of erythema which follow rheumatic fever, and occur in pale young women or lads, the preparations of steel are strongly indicated. When there is constipation a good formula is three or four grains of sulphate of iron, half a drachm of sulphate of magnesia, and five drops of dilute sulphuric acid, in peppermint water or calumba. When this is not the case the tincture of steel is a most valuable remedy. In some patients sulphate of iron with carbonate of potash and extract of Barbadoes aloes forms the most valuable Martial remedy. In whichever form iron is found to agree best it is important to increase the doses until a decided effect is obtained.

Eruptions produced by Drugs.—Since the most frequent and characteristic effects of drugs upon the skin are erythematous eruptions, it will be convenient to consider this group of dermatoses here. Drugs and poisons act much in the same way upon the skin as do irritant or poisonous articles of food.

The most striking and frequent of these eruptions is perhaps that produced by *copaiba*. This has sometimes been confounded with an early syphilide. It usually takes the form of a papular erythema, often combined with urticaria and not unfrequently more or less hæmorrhagic. The occurrence of bullæ or vesicles is mentioned by trustworthy observers. In some cases there is no itching, which makes the diagnosis from syphilis the more difficult. The rash is generally distributed over the whole surface of the body, and does not spare even the face, as most other erythemata do. Occasionally it simulates purpura. Some writers have suggested that it is not the copaiba but the urethral inflammation for which copaiba is commonly given which produces the rash. There can, however, be no doubt of the existence of a true copaiba rash. It is not uncommon from the exhibition of the oleo-resin, but is rarely observed in persons who are taking the valuable diuretic, mistura copaibæ resinæ.

Cubebs is generally said to produce a similar eruption, but some of the

reported cases appear to have been due to accidental mixture with copaiba. So at least Dr Bulkley believes.

Somewhat similar rashes have been observed in patients taking *chloral hydrate, turpentine, cannabis indica,** and some other drugs, and have been described as purpura, urticaria, pemphigus, herpes, or erythema *a medicamentis*. It is very doubtful whether iron is one of them, and some at least of the eruptions ascribed to salicylic acid were probably peliosis rheumatica.

Bromide of potassium comes perhaps next in frequency to copaiba as a rash-producing drug. The lesion here simulates very closely that which will be described in the next chapter as acne, but the diagnosis is generally clear from its not being confined to the very characteristic localities of true acne, or to the equally characteristic age which is specially liable to that disease. Occasionally the bromide eruption is more severe, and produces pustules and crusts.

Less frequent, but much more varied, more severe, and more misleading is the eruption produced by *iodide of potassium*. This is perhaps most frequently a papular erythema, widely or irregularly distributed on the trunk, limbs, and face, free from itching, and usually unfelt by the patient. Sometimes, however, there is considerable erythematous dermatitis between the papules. A follicular inflammation undistinguishable from that described above as bromide-acne is a less frequent effect of the iodide salts. More often the rash which was at first papular becomes vesicular, bullous, or pustular. In these cases the inflammation is often very severe, and the constitutional disturbance considerable. They have been, there is no question, often confounded with herpes and so-called "hydroa," and indeed until one has seen several cases it is difficult to believe that so severe a dermatitis can be due to a drug which in most cases has no effect whatever upon the skin. The eruption may simulate scabies or eczema, but the absence of definite localisation, of chronicity, of the secretion of eczema or of the cuniculi of scabies should make the diagnosis not difficult. Along with the pustules there may arise what the older dermatologists would have called a tubercular disease of the skin, raised fleshy nodules simulating papillary growths, condylomata, mucous patches, and the later forms of syphilodermia. They may resemble rupia or lupus, or even malignant disease. Inasmuch as these severe effects are apt to follow the large doses of iodide of potassium given in the later stages of syphilis, the difficulty of discriminating them is naturally increased.

In a patient under the writer's care suffering from an ordinary pustular syphilide, some of the lesions on the face and the back of the hand became so swollen, hypertrophied, and covered with profuse granulations, that both cheeks were deformed, the eyes almost occluded, and one hand was covered with exuberant granulations, which, when seen alone, suggested to different observers lupus hypertrophicus or epithelioma. There was, however, no doubt of the nature of the case. The diagnosis was confirmed by the patient's recovery perfectly when the drug was discontinued. The chief point which guided one aright in this case was that, notwithstanding his frightful appearance, the patient was eating and sleeping well, so that it was with great difficulty he was persuaded to come into hospital.

Another form of iodide rash is punctiform, and resembles scarlatina rather than measles, the patches and rose tint of which are more nearly

* In a case reported by Dr J. N. Hyde in the 'New York Medical Record' for May 11th, 1878.

simulated by the copaiba rash. This iodide eruption is often purpuric (Dr Duffey, 'Dublin Journal of Medical Science,' vol. lxix, April, 1880).

More often pustules may appear, and when deep and occupying a hair-sac cause crops of boils. The presence of iodine and bromine has been actually demonstrated in the pustules by Adamkiewicz and Guttmann.* The iodide produces its effects on the skin much more rapidly than the bromide. In both cases there appears to be a true excretion of the drug through the sebaceous glands. For an account of the histology see a paper by Dr Thin ('Med.-Chir. Trans.,' vol. lxii, p. 189).

Weeping dermatitis, curious wart-like nodules, and other peculiar eruptions have been described as the result of bromide of potassium by Voisin and Veiel, quoted by Behrend ('Berlin. klin. Wochenschrift,' vols. xvi and xxii, pp. 626 and 714, 1879). Two cases of severe iodide eruption were figured by the late Dr Tilbury Fox ('Clinical Society's Transactions,' vol. xi, November 23rd, 1877).

Various measures have been adopted to prevent these unpleasant effects. The addition of carbonate of potash or aromatic spirits of ammonia is sometimes sufficient. Moderate doses of arsenic have been recommended, but they often fail in preventing the eruption. Changing the potash to the soda salt of iodine is sometimes followed by the disappearance of the rash; this is perhaps a coincidence. It is at least certain that persevering with the drug in even larger doses is often followed by the disappearance of the unpleasant effect it had produced.

Belladonna in full doses often causes a bright red and almost universal erythematous rash. It may be recognised by its association with dilated pupils and a dry throat, together with the characteristic delirium if the dose has been large. Children, who bear as large doses of this drug as adults, are not more liable to these symptoms of intoxication. In one case under Mr Hilton it was caused by the mere application of a large belladonna plaster in a woman who must, one supposes, have been more than commonly susceptible.

Similar rashes have been observed as the result of *hyoscyamus* or *stramonium*.

Opium and *morphia* sometimes produce considerable pruritus, and this leads to erythema or urticaria by the scratching which results.

Quinine.—There can be no doubt that quinine may produce a general acute erythema, which was first described by Skinner, Fleming, and other English authors, and has been since observed abroad. Its symptoms closely resemble scarlatina. It begins in the face, spreads rapidly over the whole trunk, and is accompanied by severe fever, the temperature sometimes reaching 103·5° Fahr. It is certainly a very rare effect of so popular a medicine, and its occasion may be regarded as due to an idiosyncrasy. In one case of Köbner's the eruption followed the exhibition of quinine three times in the same patient. A still more severe local erythematous rash of the face, sometimes vesicular, has been observed as the result of quinine by Hebra, von Heusinger, and some other physicians.

Morrow, who has collected sixty cases of quinine eruptions, found that in thirty-eight the rash was erythematous, in twelve it resembled urticaria, in two it was vesicular, and in five hæmorrhagic ('New York Medical Journal,' March, 1880). One case was reported by Dr Fagge ('Medical Times,' February 29th, 1868). See also Dr Farquharson's paper in the 'Brit. Med. Journ.,' February 15th and 22nd, 1879.

* 'Virchow's Archiv,' 1878, vol. 74; 'Charité Annalen,' vol. iii, p. 381, 1878.

Eruptions from *salicylic acid* * have been reported. Since erythema and urticaria are common in the disease for which salicylic acid is usually given, and since adulterations with carbolic acid and consequent gastric disturbances are not unknown, the interpretation of these cases may admit of doubt. The internal use of carbolic acid itself, of tar, turpentine, and petroleum have all produced rashes usually erythematous, but the cases are comparatively rare. An excellent bibliography of the whole subject is given at the end, of a paper by Dr Van Harlingen in the 'Archives of Dermatology,' Philadelphia, October, 1880.

Arsenic is said in some persons to produce an acute vesicular eruption which has been styled herpes. This occurrence is, however, very rare even when large doses of the drug are given. Zona† has sometimes appeared during a course of arsenic too frequently perhaps to be considered a mere coincidence. But if when a patient is taking arsenic and zona breaks out the fact that he has not been exposed to cold, and that the eruption is not epidemic, may be accepted as evidence that arsenic is its cause, all inquiries into ætiology become at once easy and useless.

Mercury was one of the first drugs to be regarded as the cause of a cutaneous rash. Early in the present century Alley‡ described what he called Hydrargyria, before the first description of a copaiba nettlerash by Montègre in 1814. Alley's cases were mostly vesicular and corresponded with what we should now call eczema, chiefly of the abdomen, thighs, and scrotum, but sometimes they assumed a more severe pustular form, and still more rarely that of bullæ with severe pain and lymphatic inflammation, combined with angina. We may doubt whether local inunction of the drug or the effects of syphilis itself, or a mere coincident attack of eczema, may not explain these cases. Hebra, with a scepticism justified by his enormous experience, denies that any eruption on the skin is ever brought about by the internal use of mercury. The cases reported by Behrend and Engelmann were erythematous, and sometimes complicated with scarlatina.

* Cavafy, 'Clinical Society's Transactions,' vol. x, 1877, p. 88.

† Hans von Hebra, 'Die Krankhaften Veränderungen der Haut,' p. 204. He, however, is convinced that the relation is merely accidental. See also Hutchinson, 'London Hosp. Reports,' vol. v.

‡ 'Observations on Hydrargyria,' Dublin, 1804; London, 1810.

DISORDERS OF THE SEBACEOUS GLANDS, THE HAIR-SACS, AND THE SWEAT-GLANDS

ACNE.—*Nomenclature—Anatomy and course of local lesions—Distribution—Age and sex—Symptoms—Ætiology—Treatment—Acne tarsi—Acneiform eruptions produced by tar—by bromide—Acne varioliformis.*

Comedones without inflammation (acne cornée)—Milium—Seborrhœa oleosa—Seborrhœa sicca—Xerodermia—Steatoma, meliceris and sebaceous cysts.

MOLLUSCUM CONTAGIOSUM.—*Name and history — Anatomy — Pathology — Treatment.*

SYCOSIS.—*Name—Anatomy and course—Locality—Diagnosis—Distribution—Treatment—Sycosis capillitii frambœsiformis.*

FURUNCULI.—*Pathology—Anatomy—Course and distribution—Age—Contagion—Treatment—Carbuncle.*

AFFECTIONS OF THE SWEAT-GLANDS.—*Anidrosis—Hyperidrosis—Bromidrosis or fœtid sweat—Chromidrosis—Hæmatidrosis—Sudamina.*

IN the long series of inflammatory diseases of the skin we find certain affections which may be arranged, on clinical as well as anatomical grounds, in a third large group. The first we considered was that of the chronic forms of dermatitis which in various degrees resemble the common superficial inflammation produced by irritants. Traumatic eczema, idiopathic, symmetrical, weeping eczema, papular eczema, lichen, lichen planus, pityriasis rubra and psoriasis—these form a natural group, of which pemphigus is an outlying member. Although there is no evidence that their pathological relationship depends upon the presence of an antecedent dartrous or arthritic or gouty diathesis, yet they undoubtedly are really related to each other. The erythematous group of affections treated in the preceding chapter, to which pemphigus may be considered as the link, form as natural though not so extensive a family.

The present chapter deals with inflammatory processes which do not affect the skin generally but only the hair-sacs and cutaneous glands. They have also, as we shall see, peculiarities of distribution and of natural history which are no less characteristic than their anatomy.

ACNE.*—This disease is referred to by Cicero,† Martial, and other classical writers, as a blemish rather than a disease.‡

* *Synonyms.*—Acne vulgaris—Acne disseminata.—*Germ.* Finnenausschlag.

The derivation of the word is unknown, but is commonly supposed to be a corruption of ἀκμή, and to refer to its occurrence in the prime of life. Its proper Latin name was *varus*, and it was called ἴονθος by the Greeks.

† "Miro quid sit quod Servilius pater tuus, homo constantissimus, te nobis varium reliquit." Cic. fragm. ap. Quint. vi, 3, 48. *Vārus* is, of course, unconnected with *vārus*, knock-kneed, and may likely enough be related to the adjective varius.

‡ "Pæne ineptiæ sunt curare varos et lenticulas et ephelidas; sed eripi tamen feminis cura cultus sui non potest." (Celsus, 'De Med.,' lib. vi, cap. v.)

The sebaceous glands become occluded, either by their secretion being too thick or by want of cleanliness in removing the accidental obstructions from dirt. The first effect is to produce a number of small, firm, and somewhat pointed papules (*comedones*), each of which is produced by accumulated sebum and is marked by a black head, which is nothing but the dirt obstructing the orifice of the gland. This condition, which has been named *acne punctata*, may continue for an indefinite time, but sooner or later some of the papules show signs of irritation, and in most cases this very speedily supervenes in each obstructed gland. The papule becomes red, swollen, and before long yellow from suppuration having taken place. This pustular form or pustular stage of acne is no less characteristic. When the surrounding inflammatory œdema is considerable the deformity is of course increased. At last the minute abscess bursts, and the inflammation slowly subsides. When slight, no trace remains, but a second inflammatory process with the same course and termination often follows. When severe, a minute white scar is left behind, the gland is destroyed and incapable of renewed action. When the hair-sac into which the sebaceous gland opens is deep, the inflammation is the more severe, and sometimes causes a minute slough which leads to the pain and swelling characteristic of a furunculus. Such little boils are naturally slower in their course and lead to deeper scars. The face or shoulders may be seen covered with acne spots in all the above stages, so that the pain and irritation become great and the deformity distressing. Another severe form used to be called *acne indurata*.

Histology.—A section of an acne pustule shows not only the papillæ but the deeper layer of the cutis œdematous and filled with leucocytes, and the small blood-vessels dilated. In the pustular stage the leucocytes increase in number and assume the character of pus-corpuscles ; the acini and duct of the gland are filled with pus, often mingled with blood-discs. The process in the larger acne pustules is found to affect the hair-sac into which the sebaceous gland opens, so that the hair itself is uprooted and the entire follicle destroyed. When the destruction of the papillæ has taken place—in other words, when the inflammation has become "deep" instead of superficial—a scar always results after the acne is cured.

Dr Liveing has found that in a sebaceous sac which is the seat of a comedo many minute abortive hairs may often be found, the growth of which may perhaps be the immediate cause of obstruction.

It may here be observed that the presence of the minute parasitic mite known as *Demodex folliculorum* is frequent in healthy sebaceous glands and never causes acne.

Distribution.—Acne is confined, almost without exception, to the *face*, shoulders, and chest. It usually begins about the forehead, the cheeks, the alæ of the nose, and the chin ; but pimples may cover the whole of the face and the intervening skin be occupied by an erythematous dermatitis. Comedones, as the black-tipped early lesions of acne are called, are also to be generally seen on the auricle, but here they rarely suppurate. On the *back* the pustular and indurated form is more common, perhaps because it is more apt to be neglected ; and there it is that we see the most extensive cicatrices. The lesion may extend from the back of the neck and the scapular and interscapular regions down to the loins, but very seldom lower, nor does it pass round the flanks towards the chest and abdomen. A few scattered papules may be sometimes found over the deltoid or on the upper arm. The skin over the *sternum* is the least frequently affected of the three

acneic regions. The lesions are precisely the same and never extend to the abdomen, the axillæ, or the front of the neck.

Occasionally isolated comedones or acne-pustules may be found elsewhere, most often on the outerside of the thigh and peroneal surface of the leg in men with coarse hairy skins and large follicles. These, however, are not more common in the subjects of acne than in other persons, and are either accidental or connected with inflamed lichen pilaris.

Age and sex.—This singular follicular inflammation is in its origin and greatest extent confined to the age of puberty and early adult life, although acne when thus begun may continue up to thirty or even later.

Comedones may be seen in a few rare cases in children; but though numerous and apparently characteristic they do not suppurate, and they are found upon the forehead and even on the scalp without the characteristic distribution of true acne.

Any chronic inflammation of the skin, eczema, recurrent erythema, and especially that form which will be described as gutta rosea, may lead to pustular inflammation of the hair-sacs, so that the latter affection has been commonly described as *acne rosacea;* but the distribution, the origin, and the whole natural history of the two diseases are different. In fact, the more closely the subject is studied, the more decisively does true acne separate itself from all other affections.

The disease most commonly begins in lads of about sixteen, that is to say, when the changes which accompany puberty have already begun. It is not common for it to make its first appearance after the beard has begun to grow, but it may begin at from sixteen or seventeen up to one or two and twenty. It is very slow in its progress, and the worst cases are usually those of a year's standing or more. When once thoroughly established the morbid process continues until the beard has fully grown, but in most cases it then begins to subside and seldom continues after the age of thirty. When acne occurs in a patient above this age, it is usually confined to the back and has been preceded by ordinary acne of the face. This, as well as the occasional occurrence of severe acne of the shoulders with only slight affection of the face, is probably sufficiently explained by the greater attention given to a visible eruption and the less efficient treatment of all affections which cannot easily be reached.

Although the evolution of acne is, as we have seen, so closely connected with that of the beard at puberty, yet the disease is very far from being confined to the male sex. Indeed, Erasmus Wilson stated in the first edition of his treatise that acne occurs more frequently perhaps in the female than in the male. This is certainly not the case, but acne is common enough in young women about the same time as in lads, or perhaps a little later. The affection is, as a rule, more diffused in the case of women, the papules more numerous, not so large and with more erythema between. It is also more often confined to the face; and it is certainly much more rare to see the worst forms of acne indurata, and the disfigurement which follows, in women than in men. On the other hand, while it is somewhat later in its appearance it is decidedly slower in its disappearance, so that acne may be more often seen about the age of thirty in women than in men, and it is chiefly in women that a lingering acne is overtaken by an early gutta rosea, a combination which has no doubt helped in confusing the two disorders.

Course and symptoms.—Acne is always a chronic affection lasting, if left to itself, for years, but liable to occasional exacerbations. These often coincide with ovarian disturbance in women; in men they are less marked, but sometimes appear to be connected with gastric disturbance, especially with the more acute forms of indigestion, such as in some people result from eating pork or salmon or preserved viands, whether salt, like herrings, or sweet, like crystallised fruits and jams. There is but little local irritation, and the other organs are completely unaffected; indeed but for the disfigurement few patients with acne would apply to the physician.

Ætiology.—The immediate cause of acne is the obstruction and inflammation of the sebaceous glands, and in the severer cases of the hair-sacs also; but when we ask why this obstruction and inflammation occurs, the answer is extremely difficult. To say that the presence of acne indicates a disordered state of the cutaneous nerves, which interferes with the vascular action of the skin; to say that it depends on torpidity of the capillary circulation or general want of cutaneous activity; to say with Biett that it is the result of keeping the head bowed down, as in many sedentary occupations, or of drinking cold water when heated, or of smoking tobacco; or with Alibert, that it is caused by spending nights in gambling and living in anxiety—all this is trifling with pathology. It may be asserted that acne has no such connection with feeble circulation, as is shown by chilblains, nor with local irritation as eczema solare, nor with gout or tubercle, nor with the ingestion of cold water or hot water, or alcohol, or any kind of food, nor with any diathesis or disposition to anything but acne.

It is obvious, if we consider its natural history, that acne has to do with the great change which passes over the organism at the time of puberty: first and principally with the growth of the beard, yet not as a mere mechanical result, for in the great majority of men the beard appears without acne,—men have acne who never develop a beard, and women frequently have it also. Acne, moreover, affects the skin of the shoulders, which is unchanged at this period, as well as that of the face and chest, where hair grows, and it does not affect the hair of the pubes.

It is stated by Rigler ('Die Turkei und deren Bewohner,' Wien, 1862), quoted by Hebra, that acne, though common in the Levant, is extremely rare in eunuchs. Moreover, in young women affected with acne the eruption is often aggravated during the menstrual period. There is no reason to adopt the suggestion of Rayer, followed by many French writers, that acne is connected with vicious habits.

The old adage of Plenck, "Matrimonium varos curat," is well exchanged for Hebra's dictum, "Tempus varos curat." It is not continence nor vice, nor celibacy nor marriage, nor even the growth of a beard, which are the causes of acne; it depends upon the general changes which occur in the passage from childhood to adult life. The glandular apparatus of the skin is then apt to be disordered, most apt on the region where the beard is developing and loses this aptitude when complete development is once attained. With regard to acne in women we can only say (as conversely of hysteria in men) that though they have no beards their fathers had, that is that secondary sexual characters are more or less transmissible to both sexes.

Acne appears in those in good health and those in ill-health, in the blonde and so-called lymphatic, as well as in the dark and atrabilious; it is said to

be rare in persons with red hair, and to be less common in Ireland than in England.*

There is no proof that acne is an hereditary disease, although it is not infrequently seen in brothers and sisters, and although the disposition to its development at puberty would, we might expect, be transmitted more or less completely, in the same way as the early growth of a beard, its weakness or abundance, and the early or late supervention of baldness.

Prognosis.—Few cases of acne cannot be decidedly relieved by careful treatment, and in many the face can be restored to its natural appearance; but success depends not only upon the physician's adapting his treatment to the wants of each case, but also upon the perseverance with which the patient will follow it out. In many cases irremediable mischief has already been done when the patient comes before us. "Tempus varos curat," though generally true, proves often tedious in performance, and when such a cure is complete the disfigurement it leaves is often considerable.

Treatment.—In the early stage of acne, when comedones are present with little or no inflammation, the principle of treatment is to set free the obstructed ducts, to keep them clear by extreme care, and to stimulate the local circulation. The plan found most successful is the following: On going to bed the face should be first steamed over a basin of boiling water. It should then be thoroughly washed with a piece of flannel and yellow soap, and dried with a rough towel. On careful scrutiny in the glass the patient will then find that the acne punctata has lost a good many of the black points; but he should go over the whole of the face, and wherever a pimple shows by the slightest point of yellow that suppuration has begun, it should be emptied—not by squeezing with the fingers, but by pressing over it the end of a key of suitable diameter. When this has been effectually done, the face should be again washed and a lotion applied which should be allowed to dry. This drying lotion may be of sulphur suspended in liquor calcis, alum water or lead lotion, or a dilute solution of corrosive sublimate (gr. ½ with tr. benz. co. ʒss in ʒj of mist. amygd.) The old cosmetic known as *lac virginum* was of somewhat the same composition, as was the famous Gowland's lotion, which is said by Bateman to have contained oxymuriate of mercury in an emulsion of bitter almonds.† The sulphur is the more stimulant; the mercurial wash when too strong is apt to cause a feeling of constriction and tension of the skin. Next morning any fresh pimples which have ripened should be emptied and the face again washed with soap and water and a little dilute mercurial ointment (ung. hydr. ox. rubri with two parts of benzoated lard) applied to each. With many patients thorough washing and the application of white precipitate ointment serves the same purpose very well.

The same plan of treatment answers, even if a good many pustules are present, supposing that there is not much inflammation around them; but the more pustules there are, the less vigorous should be the friction used

* According to Bazin acne is of scrofulous origin, and even Hardy, while denying this, thinks that acne has a preference for lymphatic subjects, although "on peut avoir un tempérament lymphatique sans être atteint de scrofule."

Dr Erasmus Darwin, who properly distinguished acne from gutta rosea (in his 'Zoonomia'), nevertheless named the former affection *gutta rosea hereditaria*, "because it seems to be hereditary, or at least has no apparent cause."

† "Merely Gowland," said Sir Walter Elliot, "I should recommend Gowland, the constant use of Gowland, during the spring months." ('Persuasion,' vol. ii, chap. 4.)

and the more important it is to apply some ointment containing mercury to the pustules. Ung. hydrarg. ammon. or dilute citrine ointment are often well borne; in other cases the unguentum metallorum (p. 885) suits better.

When the inflammation, judged of by the erythema between the papules, by the amount of swelling, or by the presence of true furunculi, is severe, we must begin with other measures. Steaming is still useful and generally proves soothing, but friction must be much more sparingly used, and sometimes omitted altogether. Instead of a stimulating lotion or ointment, the patient must at bedtime use Goulard wash or a drying lotion of oxide of zinc suspended in water, or the almond wash may be used alone; during the day lead ointment or zinc or a combination of the two must be applied, but for women and others who are not obliged to be out of doors, the frequent application of lead lotion is better than ointment.

It is in these cases only that diet needs regulation by abstinence from stimulants, spices, and the other viands which we found tend to excite erythema of the face (p. 937). It may also be desirable for the patient to take a little carbonate of soda or citrate of potash with a saline laxative. By these means the erythematous inflammation will soon be subdued, and it is then desirable to return as quickly as may be to the more stimulant treatment.

In inveterate cases the stronger mercurial ointments are indicated. When, as occasionally happens, large furunculi are present, they must be treated with carbolic oil, and if necessary with poultices.

Acne of the shoulders, though often severe, is naturally less troublesome than that of the face; the skin is also less susceptible to irritation and almost always bears rougher treatment with advantage. The individual attention to the several papules which is so important in the case of the face is of course difficult to carry out here, and we must depend more upon the use of mercurial ointments and on friction with rough towels or flesh gloves.

Acne tarsi.—Anatomically allied to acne is the inflammation which not unfrequently, especially in children, affects the large and specially modified sebaceous glands that serve to lubricate the eyelashes. These Meibomian glands are apt to become the seat of chronic inflammation, when a gummy secretion is exuded which sticks the eyelids together. It may either occur independently or as a complication of catarrhal or other forms of ophthalmia. It is generally cured by the application of unguentum hydrarg. ammon. or yellow oxide of mercury ointment.

Tar and bromide acne.—Inflammation of the sebaceous glands, papular or pustular, is occasionally called forth by external irritants, and especially by tar. This so-called tar-acne differs, however, in its distribution and natural history from the true disease, and needs no treatment but the removal of the exciting cause.

Bromide-acne is the name given to a somewhat similar follicular inflammation caused by the internal use of the bromides in certain patients (p. 939).

*Acne varioliformis.**—This name was given by Bazin, who has been followed by other French authors, to what will presently be described as *molluscum contagiosum.* It is unfortunate that Hardy has accepted such a

* Neumann, following Hebra, calls it *acne frontalis*, and describes it as a variety of true acne. Dr Bulkley, of New York, names it *acne atrophica* or lupoid acne. See a case figured by Dr S. Mackenzie in the 'Clinical Transactions' for 1884, p. 227, and remarks by Dr Mackey in the 'Lancet' (Jan. 22nd, 1876).

confusion of nomenclature and of pathology. The name has, however, been since applied to a rare and curious affection which consists in large pustules resembling those of the more severe kinds of acne, and situated chiefly upon the forehead, the temples, and the sides of the cheeks. After they have burst and healed a deep scar is left, sometimes pitted but not pigmented, resembling that which follows the most severe forms of acne. It is in these scars that the resemblance of the affection to variola chiefly consists, for the distribution, the course, the absence of a vesicular stage, and the unimpaired health of the patient could never allow of its confusion with variola. In a patient of the writer's, a man about forty, the affection encroached upon the scalp, and also spread to a considerable part of the chest, shoulders, and back. In this, as in the other cases, this curious affection was quite distinct from true acne. It is not preceded by comedones, and it seems doubtful whether the pustules are really seated in the sebaceous glands. The distribution and the severity of the eruption also distinguish it from acne, and it is unconnected with the period of puberty. It is more difficult to distinguish it from a pustular syphilide, and some cases which have been described as acne varioliformis were probably syphilitic. In one patient, in whom there was neither history nor proof of venereal disease, the pustules, which had lasted for a long time, disappeared under iodide of potassium. Dr Liveing, however, has seen instances of acne frontalis which resisted antisyphilitic treatment and yielded to large doses of arsenic. We must wait for further observations before the true nature of this affection can be decided.

It will be convenient to refer briefly in this place to other affections of the sebaceous glands, less important than acne.

Comedones are generally the first stage of that disease, and then occur in the persons and under the circumstances above described; but beside the accidental comedo or even pustule which may be produced here and there by obstruction of a duct in any part of the body, and which no more make acne than one swallow makes a summer—there are occasionally to be seen large numbers of comedones in children affecting the forehead, scalp, and other parts not undergoing inflammation, and without the locality or other characters distinctive of acne.

Acne cornée is the name given by French writers to this remarkable and rare condition, which does not deserve the name of acne, first, because it is not inflammatory, and, secondly, because it has not the natural history of the disease of that name. It consists in the presence of a multitude of *comedones*, which remain as passive papules, hard pointed and black tipped. They occur in children before the age of acne, and upon the scalp and other parts unaffected by acne. We have had several cases of this singular condition, which requires to be distinguished from "lichen pilaris." Once it occurred on the forehead and scalp of two brothers, once on the temples of a boy of eight or nine suffering from pleurisy, and once on the lumbar region of a girl aged thirteen, under treatment for erythema multiforme, but without a trace of acne, or even comedones on the face, chest, or back (see 'Guy's Hospital Reports,' 3rd series, vol. xiii, p. 213).

Milium.—A commoner condition is passive obstruction of a sebaceous gland with complete occlusion of the orifice. A minute white or yellowish papule is thus formed without the pointed top or the black mark of a comedo. It

has been called milium from its size. It never inflames, and is of no practical importance; it occurs most often on the thin skin of the eyelids and the genitals; occasionally it grows larger than a pin's head; when this occurs it usually affects only a single gland. Its contents then not unfrequently become liquid, and it forms a small cyst, such as may be occasionally seen on the eyelids, and have been noticed by Mr Hutchinson to occur in association with xanthelasma and with sick headaches.

On dissection the acini of the gland are found filled with a dark refracting substance, which yields on analysis cholesterin, olein, palmitin, and stearin. A good drawing is given by Neumann (fig. 10).

Seborrhœa.—The functional disorders of the sebaceous glands may lead to too abundant liquid secretion, or to too solid scaly products, or by suppression to abnormal dryness and harshness of the surface. The first of these conditions has been named *seborrhœa oleasa* or steatorrhœa. It is physiological at a certain period of life, when it forms the *vernix caseosa* of newborn infants. It is not uncommon about the face, and on especially the alæ of the nose. It also occurs on the genitals, where a local vernix caseosa may lead to pruritus and inflammation: cleanliness and a little lead lotion or ointment is sufficient treatment.

The affection described by some authors as seborrhœa corporis has been mentioned above as *Lichen circumscriptus* (p. 897).

Seborrhœa sicca appears to depend upon the more solid fats, stearin and palmitin, being secreted in greater abundance than the liquid olein. The secretion forms little yellowish scales, added to by the natural desquamation, and frequently by the local irritation of a slight dermatitis, which increases the desquamation. The condition is most common on the scalp, where it constitutes what is known as pityriasis capillitii, dandriff, or scurf. In most cases this is rightly termed seborrhœa sicca; but although it begins as a sebaceous affection, in cases which have lasted long one finds that the scales consist in large part of epidermic cells, and there is often beside local irritation, injection, and other signs of dermatitis. That this is secondary is shown by its not spreading beyond the scalp, and by its being unconnected with eczema or psoriasis of the scalp. Beside the irritation of this common disorder it undoubtedly leads to the hair becoming thin and weak, and in some cases produces early alopecia.

The treatment is not very satisfactory. Mild mercurial ointments or carbolic oil sometimes prove useful.

Xerodermia.—Diminution or absence of sebaceous secretion leads to the skin being dry, harsh, and apt to crack. The sweat-glands may be active and abundant; but sweat is apt to exert an irritant effect upon the skin undefended by its natural oily secretion. This condition is usually congenital, and was rightly described under the name "xeroderma" by Wilson as the slightest degree of ichthyosis. It will be again referred to under that head.

A similar state of skin is, however, not unfrequently observed in children who are thin and ill-nourished, and in patients of any age suffering from prolonged wasting diseases, especially phthisis. The diminution of subcutaneous fat is accompanied with diminished supply of oily material to the sebaceous glands, and the skin becomes dry, pale, rough, scaly, and dirty. This condition, which is of only symptomatic interest, has been described as *asteatosis* and as *pityriasis tabescentium.*

The only treatment indicated is to supply the deficient oily material by inunction with olive- or cod-liver oil.

STEATOMA.—When the orifice of a sebaceous gland is obstructed and an accumulation of the secretion takes place, it does not always inflame; the secretion may go on until a large cystic tumour is formed. The orifice of the duct can still usually be found, and sometimes by mere pressure the contents can still be evacuated. They consist of inspissated sebum without, on the one hand, the pus which mingles with the secretion in inflamed acne, and without, on the other hand, the remarkable modified epithelial cells which will presently be described as characteristic of molluscum. When the watery parts are absorbed, the sebaceous secretion consists of the ordinary animal fats, palmitin, stearin, and olein, some butyric and caproic acids, either free or united with glycerin to form neutral fats, a small amount of albumen, or rather globulin, with a larger proportion of casein, epidermic cells, the flat tabular crystals of cholesterin, and earthy salts. Occasionally the fatty material appears to be absorbed as well as the water, and there remain behind only calcareous masses like those found in a diseased aorta or in the apex of the lung in a case of obsolete phthisis. These cutaneous calculi are, however, of very rare occurrence. The yellow, somewhat granular, half liquid, and half solid mass has been compared with putty and with mortar. It is exactly what the Greeks meant by atheroma, and although this term is now generally applied to the very similar products of chronic inflammation of the arteries, yet "atheromatous tumour" is still used by some writers as synonymous with what is otherwise called *Steatoma*, *meliceris*, or, perhaps better, a sebaceous cyst.

These tumours, called, when they attain a large size, *wens*, most frequently occur upon the scalp, where they are often multiple, and may grow to the size of a fist, or even bigger. They may also be seen upon the eyebrows, face, and neck, less frequently on the trunk, and most rarely upon the limbs.

Cysts with similar contents, but of very different origin, and, probably, (some of them certainly), congenital, occur on mucous membranes and in deeper parts of the body, especially about the root of the tongue and hyoid bone; and such *cholesteatomata* have also been described in the brain and in the bones. Many of them are true dermoid cysts and may be compared with those of the ovary, which contain not only sebaceous matter but hairs and sebaceous glands.

Sebaceous cysts are perfectly innocent, but occasionally require removal from their inconvenience or unsightliness. The plan usually adopted is to incise the tumour and tear or dissect out the secreting cyst wall, or, if this be difficult, to rub the interior with caustic.

MOLLUSCUM CONTAGIOSUM*.—This somewhat rare disease was first described by Bateman, who added it to the small group of Tubercula as defined by Willan. The case figured by him in his 61st plate, on the face and neck of a young woman, was a typical example of the disease, and Bateman traced the contagion from a nursling of this patient and two other children in the same family back to a fourth patient with the same affection. He also mentions a second case in an infant apparently contracted from an older child. It was from these facts that the epithet *contagiosum* was

* *Synonyms.*—Molluscum sebaceum—Epithelioma molluscum—Acne varioliformis.

applied by English physicians,* and also by von Bärensprung, Virchow, and Rindfleisch, and although the correctness of the epithet has often been doubted it is now satisfactorily proved.

Carswell, Rayer, and other writers recognised Bateman's disease, and Huguier, in 1846, had described it as a non-syphilitic affection of the vulva. Caillaux, in his 'Treatise on Diseases of the Skin in Children,' named it "acne molluscum."

The disease occurs in the form of small rounded tumours of a pink colour, sometimes sessile but more often pedunculated. They are scattered irregularly over the skin, which remains quite healthy between them. Their number varies from a single tumour to a countless multitude, and the size from that of a vetch, to use Bateman's comparison, or a large pin's head, to that of a marble or occasionally much larger dimensions. The colour also, though usually pink and waxy, is sometimes scarcely distinguishable from that of the skin, and at others it has a dead white or even yellowish tint. These last, however, are probably not uncomplicated examples of the disease. A minute dimple is to be found on each tumour, which is the orifice of a sebaceous duct. This disputed point, however, will be presently considered. The growth of the little tumours is very slow. They may retain a size not exceeding a pea while increasing in number during many months, and perhaps longer. As they grow bigger they become more rounded and the groove at their base becomes deeper until they may hold to the skin by only a slender pedicle, a condition which was formerly described as *acro-chordon*. The colour usually becomes paler and more translucent as they increase in size, but this is not constant.

No *symptoms* are produced in the most marked and typical cases; as before stated, the skin between the little tumours is perfectly normal, and they themselves are no more than a disfigurement.

Anatomy.—On incising the tumour, a white opaque thick material can be usually squeezed out, and a hollow sac remains flaccid behind. Herein a molluscum tumour resembles an ordinary sebaceous cyst or steatoma, but the contents are white instead of yellow, and to the naked eye have not the atheromatous appearance so characteristic of accumulated sebum. Moreover, tested chemically and microscopically, instead of fat, cholesterin and earthy salts and epithelial scales, the white material seems to be made up almost entirely of characteristic oval transparent bodies with a pearly lustre, without a nucleus and not readily staining with logwood. These have been described as "molluscum corpuscles." They were first recognised and well described by Wilson in 1842; they were rediscovered at St Louis and described as cryptogamic spores, the source of the contagion. This view, however, is certainly incorrect. Their size, their aspect, their re-

* Wilson, 'Diseases of the Skin,' 1842, p. 302; Paterson, 'Edinburgh Medical Journal,' vol. lvi, 1841, pp. 213, 240. Cases were also recorded by Alibert, Biett, Cazenave, Schedel, Gibert, and Jacobovitz, 'Le Molluscum: Recherches Critiques,' Paris, 1840. Most of the foreign cases, however, were examples not of Bateman's disease but of what will afterwards be described as fibroma molluscum.

The term molluscum, there is no doubt, was, as Mr Wilson suggested, taken by Bateman from the celebrated case of Tilesius, described by C. F. Ludwig, of Leipzic, in 1739. His words are, "Corpus tectum est verrucis mollibus sive molluscis." The word is obviously used as a synonym of mollis just as mollusca was first applied to the mollusca nuda et testacea, the *soft*-bodied animals or malacozoa. Alibert, followed by Cazenave, misinterpreted the meaning of the term. Bazin unfortunately described the disease under the term *Acne varioliformis*, and this has led to much confusion, especially since the same term has been employed for a singular variety of acne mentioned above (p. 947) before its misapplication to molluscum contagiosum had been forgotten.

action to potash and their inability to develop are conclusive against it. No doubt they are epidermic cells which have undergone a hyaline transformation. When, as is sometimes the case, along with these molluscum corpuscles there is found a certain amount of fatty sebaceous material, we must regard the condition as one complicated by the addition of the sebaceous secretion. In some well-marked cases there is an entire absence of sebum.

A section horizontal to the surface shows that each little tumour is made up of loculi more or less separated from each other by septa, and in most cases a central axis may be demonstrated, which is supposed to be the duct of the gland. The question remains whether the origin of the cyst is in the sebaceous gland, and whether the metamorphosed epithelial cells are derived from those lining its acini or from those of the duct, or whether the whole tumour is a new growth unconnected with the sebaceous apparatus and starting in the deeper epidermic cells. The latter is the opinion held by Virchow and supported by some excellent observations of Dr Sangster.*

Distribution.—Molluscum is most common on the face and neck, especially upon the eyelids and cheeks, and also upon the mammæ of women; but when a multitude of small tumours occur they may be found upon the arms, especially the thin skin of the flexor surface, as well as on the face.

Mr Hutchinson describes molluscum as not uncommon on the penis and scrotum of young adults, and in one case mentioned by Dr Paterson similar tumours were apparently caused by contagion upon the vulva of a woman whose husband was thus affected. He also refers to the molluscous growth occasionally suppurating, and points out that it may resemble an indurated chancre.

In the cases which the writer has seen of molluscum occurring on the male genitals, the tubercles have been yellow and not either pearly and translucent nor pink and waxy looking, and on incision or even on pressure without incision have yielded opaque yellow oily material, so that they should rather be called steatomata. In the Guy's Hospital museum, Model 496 shows the appearance of molluscum upon the thigh. Sometimes a large molluscous tumour will suppurate, burst, and thus cure itself. A case of this kind, which occurred under the late Dr Addison, in a little girl ten years old, was modelled by Mr Towne for the same museum.

Molluscum contagiosum is most common in infants and children, less so in women, and decidedly rare in men. In children it almost always affects the face, in women the mammæ, and in men the genitals. But this is only true of the larger and fewer tumours. All the cases of numerous very small molluscum simulating warts and sometimes described as molluscum verrucosum which the writer has seen in adults have been situated on the arms. Mr Hutchinson has observed similar cases in which the trunk or lower extremities have been so covered with little tumours as to resemble some papular eruption, as lichen.

Molluscum appears to be much more common in England than abroad,

* See papers by Drs Morrison, Crocker, and Thin in the 'Pathological Transactions' for 1881, p. 245, and also to a description by Dr Davies-Colley, 'Guy's Hospital Reports,' 3rd series, vol. xviii, 1870, p. 350 and figs. 1 and 2, p. 364. He describes most of the characteristic oval cells as nucleated. Virchow's original paper was published in 1865 in the 33rd volume of his 'Archiv,' p. 144.

though cases of the true disease are reported from France and Germany. It is well known in Scotland and also in America. The majority of cases occur in dirty neglected children, but it is often seen in those who are clean, rosy, plump, and in every respect healthy.

Ætiology.—There can be no doubt that the epithet contagious is rightly applied to this disorder, notwithstanding the frequent failure produced by inoculation and the incredulity of Hebra and other dermatologists. The subject is well discussed by Dr Duckworth in two interesting papers in the 'St Barth. Hospital Reports' for 1868 and 1872. No fungi and no bacteria have yet been observed.

Treatment.—Molluscum tumours beside, as above stated, sometimes suppurating, which appears particularly apt to occur when they are confluent, may also undergo passive involution by gradually shrinking and subsiding. This must be the case with many infants who have never come under medical treatment, but these modes of spontaneous cure are rare or slow and uncertain, while treatment is rapid and efficient. Each tumour should be removed either by being snipped off with a pair of sharp scissors curved on the flat, or by being incised and emptied. In either case the whole of the diseased structure must be removed. Where the tumours are very numerous, it may be better to apply nitric acid or the acid nitrate of mercury to each one in the early stage. When the growths are not larger than pins' heads, Mr Hutchinson believes that white precipitate and sulphur ointment in equal parts will cure the affection; but it is rare for an opportunity for this treatment to occur.

Sycosis.*—Closely allied to acne is a disease whith consists in pustular inflammation of the large hair-sacs of the beard. Anatomically it is difficult to draw a broad line between them, for although we speak of acne as inflammation of the sebaceous glands, yet since all these glands open into a hair-sac, obstruction of their duct and obstruction of the corresponding hair-sac are almost the same. The anatomical difference lies in this, that whereas on the general surface of the body the hairs are small with shallow sacs, and the sebaceous glands large, in a man's beard the hair-sacs are large and deep, and the glands comparatively small. Moreover, there is no doubt that in acne it is the gland which is first obstructed so as to form a comedo and which afterwards inflames, while in sycosis it is the hair-sac which is the primary seat of disturbance.

It was Bateman who first accurately defined the characters of sycosis in the modern sense of the word. He placed it under Willan's order Tuberculа on account of the swelling and induration which often surround the pustules.

Anatomy.—Hebra expressed the opinion, since supported by Liveing and other observers, that the immediate cause of a sycosis pustule is the presence of two or more hairs growing together in the same follicle.

* *Synonyms.*—Mentagra—Varus mentagra—Acne menti vel barbæ—Barber's itch.—*Fr.* Impétigo sycosiforme (Devergie), Adénotrichie (Hardy).—*Germ.* Bartfinnen.

The name *sycosis* was applied to this affection by the Greeks, from its supposed likeness in the worst cases to the inside of a ripe fig, the red pulp answering to the inflamed and swollen skin, the seeds to the little pustules. "Est etiam ulcus quod a fici similitudine σύκωσις a Græcis nominatur" ('Celsus,' lib. vi, cap. 3). The terms *mentagra* and *lichen menti* of Pliny and Martial, like the sycosis of the Greeks, were often applied to syphilitic affections of the lips and other parts, probably to what we now call mucous patches and condylomata; but the Latin *ficus*, like the Greek σῦκον, was certainly applied not only to condyloma ani but also to hæmorrhoids.

Wertheim, in 1861, published a paper in the 'Transactions' of the k. k. Ges. der Aertze of Vienna, in which he referred the origin of sycosis not to the growth of more than a single hair in the follicle, but to its being abnormally thick.

Whatever the immediate cause, suppurative inflammation takes place in the hair-sac, and the hair-bulb becomes loosened, but the shaft still blocks the sac. The drop of pus first formed is pent up and produces pain and fresh inflammation. By the time that the hair is at last detached, a small but deep cutaneous abscess has formed, and considerable congestion and œdema around it has produced what Willan called an inflammatory tumour or nodule. When the pus at last finds exit, it dries into a scab; and this is rendered much more adherent than that of impetigo by the numerous hairs which tether it to the skin. Fresh accumulations of pus take place beneath it, and thus in severe cases of sycosis a most repulsive and "malignant" aspect may be produced.

Course.—If left to itself, sycosis is a most obstinate disease. The hair-sacs are successively destroyed and cicatrices result which are sometimes deep and obvious. When at last the disease has worn itself out, the greater part of the beard is often permanently destroyed and the face disfigured by scars. The affected part is usually very tender, though, except when touched, there is rather tension and heat than severe local pain.

Excluding parasitic sycosis, which is of course contagious, Hebra and most German writers maintain that inflammation of the hair-sacs of the beard is non-contagious; but this seems to be very doubtful. As before stated (p. 896), pus is itself in many cases an extremely contagious product. We see that it is so in cases of contagious impetigo and of furunculi, and therefore it is wise to consider all cases of sycosis as more or less capable of spreading by inoculation of neighbouring hair-sacs in the same person, or even under favourable conditions, to another person.

Distribution.—As explained above, the peculiar kind of inflammation described can only occur in large and deep hair-sacs like those of the beard. The disease usually begins upon the chin, frequently on the upper lip, on the cheeks, or under the jaw. It may, however, occasionally be observed in the eyebrows and on the pubis, and still more rarely what perhaps may be fairly called sycosis has been observed upon the chest, thighs, and other hairy parts.

Bateman asserts that women are not altogether exempt from sycosis, and Wilson admits that in rare instances it has been seen in a female patient. This, however, is a question of diagnosis (or at least of definition) rather than of fact. The typical disease, excluding acne, inflamed ringworm, impetigo, and all syphilitic affections, is confined, if not absolutely, with the rarest exceptions, to the chin, lips, and cheeks of male adults. It is rare to see it in the comparatively soft beard which has never been shaved.

Diagnosis.—Sycosis must be distinguished from eczema of the face, for ordinary papular eczema and impetigo sometimes invade the cheeks and lips, and simulate sycosis. It is possible for the dermatitis thus produced to penetrate to the deep hair-sacs, and then a condition ensues which must be termed true secondary sycosis. But this is certainly very seldom the case; the superficial dermatitis as a rule preserves its superficial character, the pustules and crusts are those of impetigo, and when removed leave the surface but little affected; the treatment by ung. hydr. ammon. is

simple and rapidly successful; no scars are left behind, and no hairs are destroyed.

Treatment.—In cases of true sycosis which affects the hair-sacs, mercurial ointments must be combined with epilation. It is not, however, necessary in most cases to remove all the diseased hairs, and certainly not healthy ones. It is enough if those which are already loosened are extracted, so that the rule is for the patient to pluck out all the hairs which will come easily and without pain, that is, those which are already detached from their sacs. The first step in severe cases is to steam the face, and if necessary, to soften the crusts with poulticing, and sweet oil; then to remove the loose hairs with broad-pointed forceps. The beard should be cut short but not shaved. If there is much local pain and swelling the inflammation should first be subdued by lead lotions, lead and zinc ointments, or other soothing and astringent applications. When this is accomplished the treatment above advised should be begun and followed out day by day. In most cases the result is successful. When cure has resulted it is generally better for the patient not to shave for several months, but to allow the beard to grow naturally.

Not infrequently, however, sycosis proves very obstinate in spite of all care and diligence. The possible presence of a parasitic cause should in such cases be carefully looked for. In the most obstinate cases complete epilation on Plumbe's and Hebra's plan is no doubt the only effectual treatment, but it should be carried out piecemeal and with the help of previous application of potash-soap, and other remedies which soften and loosen the hair. During epilation dilute red oxide, or better, perhaps, the yellow oxide of mercury ointment should be rubbed into the surface.

Bateman recommended diluted unguentum hydrarg. nitr., or white precipitate ointment with an equal part of zinc or lead.

Parasitic sycosis.—Since Gruby, in 1847 ('Gazette Médicale,' No. 37), published an account of a cryptogamic plant which he discovered in cases of sycosis, French writers have generally described sycosis as a parasitic affection. Bazin named it Teigne Mentagre. Gruby's name for the fungus was *Microsporon mentagrophytes*, but Bazin and Rubet proved that it is identical with *Trichophyton tonsurans*, the name given to that of common ringworm in 1846 by the Swedish writer Malmsten. So also Köbner in 1864. Hardy followed Bazin and diverged from Cazenave and from Biett, who had placed sycosis among pustular dermatoses, for he practically maintained that all sycosis is parasitic. In his recent work, however, while regarding "true sycosis" as a teigne sycosique, he admits non-parasitic sycosis as an impetiginous inflammation of the hair-sacs.

Hebra, after reviewing the statements of previous authors, affirms that in more than 300 cases of sycosis or "follicular inflammation of the beard" he has never seen a single case accompanied with parasitic fungi.

There can, however, be no doubt of the existence of what the French writers call parasitic sycosis. The writer saw many cases at St Louis, and others, though much more rarely, in London. He never saw a case in Vienna, and those who have studied dermatology in several schools will probably agree that this is one of the instances in which we must admit local differences in the frequency of diseases. Parasitic sycosis certainly does occur and will be found if looked for in London, but it is far less common here than in Paris; and it must be added that the presence of the

fungus is often only to be ascertained after prolonged and repeated search. When detected, however, the case acquires at once a new character, and for practical as well as scientific reasons it is desirable to separate "parasitic sycosis" from the non-parasitic disease.

*Sycosis capillitii.**—This title of Willan's should perhaps be given to five remarkable cases of sycosis or pustular eczema of the hair-sacs, observed by Hebra on the occiput and nape of the neck.

This very rare condition was named by Kaposi *dermatitis papillomatosa capillitii.* Hans von Hebra ('Archiv. für Derm. und Syph.,' 1869, p. 382) describes them, under the name suggested by his father of *Sycosis frambœsiformis.* He describes it as beginning in very small somewhat red papules, each traversed by a hair, which grow together and form hard tumours resembling raspberries, and at last end in a long, tough, cheloid-looking band. The disease occurs on the nape of the neck where the hair is growing; the skin around is eczematous and red, and the place painful. The course is very slow, and as new papules arise they fill with thin pus. When raised flat papules have been formed, hairs are seen pushing out in bundles. This, with the hardness of the growth, its extreme slowness of development, and its locality are the characteristic points. It may, however, occasionally occur on the scalp. This same affection has been described by Dr Vérité as *Acné kéloïdique* (Académie de Médecine, 9 Mai, 1882). And Mr Morrant Baker has described and figured a case under the same title ('Pathological Transactions,' 1882).

The treatment of this remarkable disease consists in destruction with caustics or removal of the tumours while still small with a sharp spoon, by galvano-caustic, or by other means. When the disease has already gone far excision is the only remedy.

Histological sections show that there is true enlargement of the papillæ, very scanty exudation of leucocytes, and gradual formation of parallel and interlacing bundles of fibrous tissue, among which the sebaceous and sweat-glands are squeezed and atrophied.

FURUNCULI.—*Boils.*—Recognising the complete impossibility of any complete and satisfactory classification of skin affections which can set forth all their complicated mutual relations, it seems convenient to associate the troublesome and painful affection of boils with acne and sycosis; for although it is not possible to demonstrate that the seat of inflammation is always in a hair-sac, yet in many cases this may be readily ascertained and it is probably true of all. However, the depth of the inflammation, its pustular character, and the scars which it leaves behind, are points in which it is closely related to the affections described in this chapter and particularly to the deep and painful suppuration which affects the glands of the vibrissæ of the nostrils and the ears.

The characteristic pathological feature of furunculus is that the inflammation leads to the death of a minute portion of the deeper layer of the cutis. This slough or core of necrosed connective tissue is passed out by a

* *Synonyms.*—Sycosis frambœsiformis (Hebra)—Dermatitis papillomatosa capillitii (Kaposi)—Acne keloid. The sycosis capillitii of Willan, p. 66 of his 'Atlas,' is not unlike the curious affection described on p. 947 as *Acne varioliformis.* The *Pian ruboide* of Alibert (pl. 35) may have been the same disease, unless we suppose with Bateman that these were mismanaged porrigo favosa, or with Hebra that they were undiagnosed syphilis.

process of liquefaction and suppuration, and the abscess which is formed then slowly heals. In its early stage the disease appears as a pimple, distinguished by its excessive pain, a pain which resembles that felt from the plucking out of a hair, and in all probability depending upon inflammation of a hair-sac under somewhat different conditions from the comparatively painless pustules of acne and sycosis. The papule speedily shows a yellow spot at its pointed summit and this little pustule is never preceded by even a transient vesicle. Meantime, a bright, intensely injected halo appears around the pustule and considerable inflammatory œdema swells the whole skin into a conical elevation; the pain increases and becomes throbbing in character, while the dull constant aching and sense of tension is varied from time to time by sharp stabbing pains. When the abscess has ripened and is lanced or bursts of itself the core becomes visible, and is sometimes not expelled for a day or even longer. This stage is accompanied by a sharp pricking pain which is very characteristic; the pain rapidly subsides when the core is got rid of, a small scab forms, the redness and œdema disappear, and soon nothing but a minute scar remains.

Unfortunately, however, it is seldom that this process is confined to a single furuncle. Most often a second and a third appear before the first is completely healed, or a whole crop may spring up almost simultaneously. A succession of painful abscesses may thus be established and last for weeks or even months, until the patient's health seriously suffers from the pain and the discharge. Sleeplessness, loss of appetite, and much depression, both physical and mental, may be the result. When a crop of boils thus appears, they are generally found to vary in size and severity, from those which are so large and deep as to challenge the name of carbuncle, to small superficial pustules which the older dermatologists would have called ecthyma.

Distribution.—There is scarcely any part of the surface which may not be the seat of a furunculus, but the affection has nevertheless a decided predilection for the back of the trunk from the hair at the nape of the neck to the fold of the nates. The thick cutis, thin epidermis, and small but numerous hairs of the dorsal region appear to furnish the most favourable conditions for this kind of inflammation. The back of the neck, especially at the edge of the scalp, is perhaps the most frequent seat of all; the buttocks come next to the nape of the neck in liability to boils. Moreover, the friction of the collar of the dress in the latter situation, and that occasioned by riding or by rowing in the former, aggravate the misery of the complaint and probably keep it up.

Boils are far from unfrequent in the coarse skin of the outer part of the thighs which resembles that of the dorsal and scapular regions anatomically. They may also appear, though less frequently, on the leg below the knee, on the upper and forearm, on the wrist, and on the back of the hand. The chest and abdomen, and even the face, are not exempt, but boils very seldom occur on the scalp, and never on the palms, or the soles of the feet. They are rare on the male genital organs, but not unfrequently occur in the neighbourhood of the anus, in the perinæum, and on the vulva.

Ætiology.—The true cause of this form of inflammation is unknown. There is little ground for supposing that boils are the result of indigestion or of overwork or exhaustion from any cause; when numerous and long continued they produce, but are not the product of, anæmia and weakness.

Nor, on the other hand, do they come from plethora and over-richness any more than from poverty of the blood. In the practice of the water-cure it is customary to wrap patients who suffer from dyspepsia, paralysis, and most other chronic diseases in wet sheets, which are then surrounded with blankets; free perspiration is thus produced, and in many cases it is offensive in odour and accompanied by a copious crop of boils and pustules. This is supposed to indicate the efficiency of the plan in bringing out the poison from the system, but in reality it only means stimulation of the sudoriparous glands, possibly vicarious excretion of urinary or fæcal products, and certainly traumatic inflammation of the skin.

Age.—Boils are far the most frequent during youth. They are rare in infants, and not common in early childhood, but schoolboys are very liable to them, especially to the most characteristic form of successive crops on the neck and shoulders or on the nates. After thirty, liability to this painful affection becomes decidedly less, and it is comparatively rare to find boils in an elderly man. Women during the whole of life seem less liable than men, though some of the most severe cases occur in young women.

Furuncles are sometimes contagious from one patient to another; and almost an epidemic may sometimes run through a school; but more frequently they spread from place to place upon the same patient.

Treatment.—If this view be correct it furnishes an important indication for treatment. While the first furuncle is developing, no doubt a poultice gives great relief from its warmth and the relaxation of tissue it produces, but by making the skin sodden and softening the epidermis it predisposes it to inflammation and renders the access of the chemical or morphological contagium of the pus more easy. The constant application of poultices over large surfaces affected with boils very much tends to spread and continue the disease. A much better plan is to dispense as much as possible with poultices, using water dressing instead, and to apply to the skin immediately round the boil the lotio plumbi, Goulard wash, or a somewhat stronger lead lotion. Tannic acid may be used with the same object, or as each bulla appears a circle may be drawn around it with tincture of iodine or dilute solution of silver. Sometimes collodion, especially the flexible collodion applied in the same way, seems to act best both as an astringent and a protective. Meantime, the pustules should be covered with lint soaked in carbolic oil (one in ten) and the same antiseptic dressing should be continued after the pustule has burst. It was formerly the practice to open each boil successively with the lancet, but though the process is undoubtedly thus hastened, the pain and the dread of the pain are so severely felt, especially in young people, and when a sensitive part of the body is affected, that at least in such cases the furunculi may be left to ripen and burst of themselves.

There is no evidence of special advantage in the purges and alteratives which are the traditional treatment for persons affected with boils. Where only one or two exist no internal treatment is necessary; but where the crops are numerous and successive, the treatment above indicated by local astringents and antiseptics should be combined with the internal administration of wine or porter with the meals, and of either bark with mineral acids or tincture of steel in full doses. Small doses of Calcic sulphide are frequently prescribed for boils as well as for other chronic suppurative affections, and apparently with benefit. Thin delicate boys will often be

much benefited by cod-liver oil either alone or, if anæmia indicates it, in combination with steel. There is reason to believe that a stay at the sea-side is particularly useful during convalescence.

Carbuncle.—This term, as the diminutive of *carbo*, is the Latin translation of ἄνθραξ, a coal, and was applied to any red, angry, inflamed pustule. The word anthrax has in recent times been restricted to the disease known as splenic fever, accompanied with a characteristic boil or carbuncle of the skin, derived by contagion from cattle and associated with the presence of a specific bacillus, which has been fully described in the first volume (p. 367).

A carbuncle is pathologically identical with a boil, differing only in its severity and extent, but its natural history is sufficiently different to justify the old distinction being retained.

Anatomically, a carbuncle is the inflammation which accompanies a considerable cutaneous and subcutaneous slough. It differs from the larger and deeper boils by the affected tissue being so extensive that not a single opening forms, but several, giving a characteristic perforated aspect to the broad summit of the tumour. If left to itself this gradually opens by ulceration, and a deep and wide aperture is formed, through which the slough is at length extruded, often with considerable hæmorrhage. The surrounding redness is commonly deeper and more lurid in hue than that of a boil; the œdema also is more extensive. A carbuncle almost always occurs singly. Its most frequent seat is the nape of the neck and the shoulders; it may occur on any part of the trunk, but is rarely seen on the limbs or the buttocks. Occasionally it appears upon the face, and is then severe and often dangerous.

Affections of the Sweat-glands.—The sudoriparous glands are less liable to disease than the sebaceous, and their affections are less important from a local, though far more so from a symptomatic, point of view.

Anidrosis, or deficiency of sweat, is seen as a concomitant of many forms of pyrexia, and usually accompanies erythematous and roseolous eruptions. In most of the forms of superficial dermatitis, also in psoriasis and in pityriasis rubra, little or no sweat is secreted, and probably the same is true of eczema madidans. The skin of ichthyosis, including its slighter forms, which will afterwards be described as "xerodermia" (dry skin), is also marked by absence of sweat. That the function of this secretion is only supplementary to that of the kidneys and lungs as an excretion of water, and that its chief purpose is not excretory but regulative of temperature, is shown by the fact that patients with universal ichthyosis or pityriasis rubra show no symptoms of blood-poisoning from retained excreta.

Hyperidrosis.—General or profuse secretion of sweat takes place under two conditions. First, along with hyperæmia; this occurs in health during the natural sweating and warmth of skin induced by active exercise, and pathologically in rheumatism and the sweating stage of ague. Secondly, profuse cold perspirations take place with an anæmic state of the skin, as in the cold perspirations of terror, the night sweats of phthisis, and the cold perspiration which sometimes marks the approach of death. Modern physiology teaches that the vascular supply and the epithelial activity of sweat-glands, as of other secreting organs, are governed by two distinct sets of fibres, the former of which appear generally to belong to the ganglionic, the latter to the directly spinal system of nerves.

Local hyperidrosis, when it affects the hands and feet, is sometimes the source of considerable annoyance. Astringents are often useful, particularly tannin and alum. In a troublesome case profuse perspiration of the palms of the hands in a young lady was cured, after other treatment had failed, by the local application of belladonna. Internally the same drug is indicated by our knowledge of the physiological action of atropine upon the submaxillary gland; but the difficulty is in substituting for an occasional large dose the altered habit which may be produced by continuance of less than physiological doses for a considerable period.

Bromidrosis and *osmidrosis* are names given to fœtid perspiration, which is usually also excessive. This most frequently affects the feet, and may become a source of the utmost discomfort. The persons it affects are almost always young adults, and women more frequently than men. A horrible stench results from decomposition of the fatty matter which mingles with the sweat, particularly the fatty acids which belong to the formic acid series—butyric, caproic, and caprylic. Dr Thin has described and figured a bacterium to which this decomposition is probably due ('Proc. Royal Soc.,' 1880). Almost the only other seat of evil-smelling sweat beside the feet is the axilla.

The treatment of this distressing affection is often extremely difficult. The first step is to check the secretion by astringents, and to prevent its soaking into the clothing by absorbent powders, such as lycopodium; the next is, by frequent change of linen, to remove the products of excretion as rapidly as possible. Antiseptics like thymol and salicylic acid may be usefully applied, and the latter preparation, especially in the form of collodion or a salicylic plaster, has the further advantage of softening the accumulation of macerated cuticle, which helps to keep up the disease. With the same object Hebra used to recommend enveloping the foot and toes in strips of diachylon plaster, and many can testify to the efficiency of this treatment, the details of which will be found minutely given in the English edition of his work, vol. i, p. 89. Dr Thin found a saturated solution of boracic acid efficient ('Brit. Med. Jour.,' Sept. 18th, 1880).

Chromidrosis is the name given to the occasional secretion of coloured sweat. The sweat of the axillæ in some persons contains enough pigment to stain their linen of a reddish tint. The writer has met one well-marked case of this, and Hoffmann has recorded another in the 'Wiener med. Wochenschrift' for 1873, No. 13. Sometimes, however, a bluish pigment stains the sweat on the face or elsewhere. Cases of supposed chromidrosis occurring in young women should be watched. In most cases the apparently dark sweat is an *arte factum*. But although most supposed cases have proved to be factitious, there is no doubt that true chromidrosis does occasionally occur; and in some cases it has been proved to depend on indican being excreted in the sweat, and turning to blue indigo when oxidised by exposure to the air. Dr Foot published a case in the 'Dublin Quarterly Journal' for August, 1869, and collected no less than thirty-seven others. Another source of colour is the production of blue or greenish fungi in decomposing sweat. When coloured sweat affects the eyebrows it is usually of black colour, looking almost like soot. A remarkable case of red-coloured sweat was reported by Dr Wilks in the 'Guy's Hospital Reports' for 1872. In this case a chemical analysis by Dr Thomas Stevenson proved the presence of iron but the absence of hæmoglobin.

Uridrosis, or the excretion of urea in the sweat, probably only occurs

as a morbid phenomenon. The observations of Funke on the normal excretion of urea through the skin have not been confirmed, but in Bright's disease urea has been visibly discharged in the sweat.

Hæmatidrosis (or *hæmidrosis*), a bloody sweat, is an extremely rare but undoubted morbid condition. It does not appear to accompany purpura or other diseases in which one would anticipate such hæmorrhage from general changes either in the blood or the capillaries; and in some of the very few authentic cases on record it appeared during apparent health, as in that of a friend of Hebra, who observed the exudation of blood-stained sweat upon his hand while sitting at table.

Dysidrosis was the name given by the late Dr Tilbury Fox to a curious affection of the skin of the hands, since described as *chiro-pompholyx* by Mr Hutchinson ('Lancet,' 1876, vol. i). It consists in large vesicles without any surrounding inflammation, occurring in groups upon the palm and back of the hand and the fingers, especially near the web. These vesicles have been compared to sago grains, though they sometimes reach a much larger size. Rasori, who published a case in the 'Transactions' of the International Medical Congress for 1881, vol. iii, p. 146, calls it hydro-adenitis diffusa. Hans v. Hebra records a case in his 'Kr. Veränderungen der Haut,' p. 426. It affected the palms and soles of a woman forty-two years old, and some of the larger bullæ were surrounded with a red halo. Mr Hutchinson has observed relapses of this singular affection on several occasions. Whether it depends, as Dr Fox supposed, upon obstruction of the duct and accumulation of its contents is doubtful. Dr Liveing regards it as an inflammatory affection characterised by the symmetry of the parts attacked, its preference for the hands and feet, its large vesicles or blebs, and its tendency to recur. He has also noticed that the nails are sometimes undermined and broken near the root He notes a typical case observed in America by Dr Robinson, of New York. Dr Liveing believes that this chiro-pompholyx is rather allied to a bullous erythema than dependent on obstruction of sudoriparous glands.

Sudamina.—There is, however, a well-marked cutaneous affection which undoubtedly depends upon accumulation of sweat in little vesicles under the skin and has been known for centuries as sudamina or miliaria. It is only seen during the profuse sweating of rheumatism. The orifice of the duct becomes obstructed, and the horny cuticle is raised as a thin transparent layer enclosing a drop of transparent fluid (*miliaria crystallina*). This ruptures before it exceeds the size of a pin's head, but sometimes the contents become turbid and alkaline from slight inflammation. On the chest and back these sudamina are most common; they never occur on the face, and are rare on the thick skin of the palm, where they naturally attain larger dimensions before they burst, so as to resemble the "sago-grain" vesicle of chiro-pompholyx above described.

The profuse sweat which causes sudamina also produces, especially if not quickly removed, local irritation. This seldom goes beyond the stage of papules or erythematous redness except where it is aggravated by friction. The commonest seat for this *dermatitis a sudore* is the vertebral groove from between the shoulders to the sacrum, and the front of the chest. The more severe inflammation which occurs sometimes in the axillæ, often between the toes, and most frequently in the perinæum and between the cheeks of the nates, is known as *intertrigo*.

RINGWORM AND ITS ALLIES, WITH OTHER AFFECTIONS OF THE HAIR

RINGWORM.—(1) *Tinea tonsurans—Anatomy—Course—Events—Histology—Detection of the fungus—Ætiology—Prognosis—Treatment—Parasiticides—Irritants—Mode of application—Epilation—Precautions against contagion*—(2) *Tinea circinata—Form and locality—Burmese ringworm—Tinea marginata—Treatment of ringworm of the body*—(3) *Onychomycosis—Rarity and obstinacy.*

FAVUS.—*History—Anatomy—The fungus—Treatment.*

TINEA VERSICOLOR.—*Names—Parasitic nature—Appearance—Distribution—Diagnosis—Treatment—Tinea vel Pityriasis rosea.*

ALOPECIA.—(1) *Physiological*—(2) *Febrile and syphilitic*—(3) *Area—its appearance—locality—spread—question of its parasitic nature—prognosis—diagnosis*—(4) *Universal alopecia*—(5) *Congenital alopecia.*

TRICHOCLASIA *or brittleness of the hair.*

WE have seen in other parts of this work that, beside animal parasites, the human body is liable to the invasion of the lower forms of vegetable life. The Schizomycetes, often spoken of generally as *Bacteria*, are by far the most important of these, since they probably form and certainly convey the contagion of some, possibly of all specific fevers. They are described at p. 24 of the first volume.

Of far less practical importance are the fungi which are parasitic on the human body. Some of these affect the mucous membranes and have been already described in the present volume as *Oidium albicans* in the mouth, p. 301, and *sarcina* in the stomach, p. 363. We have now to consider diseases of the skin which depend upon the growth of similar microscopic fungi. In most cases the cryptogamic spores and mycelium lodge in the deep hair-sacs of the skin.

We will take, first, the most important of this group, then the remaining parasitic affections, and, lastly, it will be convenient to deal in this chapter with non-parasitic affections of the hair which need to be distinguished from ringworm.

RINGWORM OF THE SCALP.*—This troublesome disease, which our forefathers described as "ringworm," and rightly distinguished from "scald head," or impetigo of the scalp, was only proved to depend on the presence of a cryptogamic parasite in 1844 by Malmsten, the Swedish microscopist. He named the fungus *Trichophyton tonsurans.*

Origin and spread.—We seldom see the earliest stage of the disease, but the first effect of the entrance and growth of the fungus in the hair-sacs is for the affected hairs to lose their glossiness and colour and become dry, shrunken, and brittle. They break short and probably thus expose fresh

* *Synonyms.*—Tinea tonsurans, the Porrigo scutulata of Willan.—*Fr.* Teigne tondante.—*Germ.* Herpes tonsurans.

spores to spread the contagion. At the same time, the growth of mycelium in the hair-sac produces slight irritation, partly from the inflammation directly excited and partly from the patient's scratching. Moderate hyperæmia and corpuscular exudation follow, so that by the time a small bare patch appears, it is raised, slightly red and covered with a few scales. The process extends, partly by the spores being conveyed to fresh places, partly by their steady advance to the next adjacent hair-sacs. Thus, one, two, and often numerous round patches are developed, each of which closely resembles the other. The form is often geometrically circular, sometimes oval or irregular; the hair is replaced by a few broken, dark, and thick stumps, which can be recognised by the naked eye, while their characters are still more obvious under a lens; the surface is usually covered with greyish-yellow desquamation composed of epithelial cells and sebaceous material mixed with broken hairs, spores, and mycelium. It has a uniform, granular, closely adherent appearance which is almost decisive to a practised eye. At the edge of the circle a little redness may sometimes be observed, occasionally a few papules, and still more rarely, a vesicle or two. In the immediate neighbourhood individual hairs may be found by the aid of the microscope to be already affected by the spreading evil.*

A ringworm patch may increase to several inches in diameter without materially altering its appearance, but more often it is modified as it expands.

Either from scratching or from the effect of the fungus on the naturally irritable scalp, or as the result of irritant applications, more or less of ordinary superficial dermatitis appears, so that many cases of ringworm appear as impetigo capitis, and their true nature is not manifest until the scabs and crusts have been removed. In neglected cases, moreover, pediculi are not unlikely to breed and further aggravate and confuse the condition. Such horrible masses of felted hair, mingled with inflammatory products, vegetable and animal parasites, and all kinds of filth, constitute the *plica polonica* of Eastern Europe, which may still be sometimes seen at Vienna.

On the other hand, if the hair is kept short and the head clean, and if the skin is not naturally irritable, the fungus, while spreading at the edges of the patch, appears to exhaust the soil in the centre and dies away like the larger cryptogamic fungi which form fairy rings upon the grass. The result is that the middle of the patch is more or less completely bald, with only a few short stumps or thin, feebly-growing hairs, while the circumference is occupied by a zone of flat brownish scales, granular desquamation, papules and broken hairs. This is the most typical form of traditional ringworm and probably suggested the specific title *scutulata*. When growing patches of the disease meet, they form figures of 8 or dumb-bell-shaped patches, and as they still grow and unite with others, irregular gyrate figures like those of old-standing psoriasis, of erythema marginatum, or of syphilodermia. At last almost the whole scalp may be invaded, and reduced to baldness. There is, however, never a perfectly smooth clear skin left as in alopecia areata, but a few ill-developed, thin, pale, scattered hairs are always to be found. Moreover. the process is seldom or never

* They may be more easily detected by the naked eye if, as Sir Dyce Duckworth suggests, chloroform be first applied ('Brit. Med. Journ.,' November, 1873). This gives the affected hairs a dry, pale, brittle look, like that of burnt-up hay, apparently owing to its solvent power on the oily constituents of the hair. But this reaction is far from decisive alone.

quite universal: on one side or other about the temples or the occiput more or less unaffected portions of hair remain.

Events.—The disease does not spread continuously beyond the scalp, but fresh patches arise, sometimes in the eyebrows, occasionally in the beard, more frequently on the skin of the neck and shoulders, and even on more distant parts. If left to itself, the course of the disease is extremely chronic, and shows little or no tendency to recovery—if the patient is a child—until the period of puberty is reached. It must not, however, be supposed that among neglected children in a village or a school, where ringworm has invaded the community and scarcely a child has escaped, the disease constantly assumes the severe and inveterate character above described. A single bald patch may remain for months or years, or it may more or less completely recover and fresh patches go through the same series of changes; or, what is still more important to notice, the spores falling upon an unfavourable soil continue to multiply, and are thus a fresh source of contagion, but yet do not sufficiently interfere with the nutrition of the hair to produce obvious bald patches. In a family or school in which ringworm has appeared one may find evidence of its presence in the heads of children who are entirely without the characteristic bald patches.

Histology.—If one of the broken stumps of a ringworm patch be extracted with forceps and placed in a drop of liquor potassæ under a quarter-inch objective, it may often be at once recognised by its opacity. When less densely packed with spores or when soaking in potash has cleared it, the condition is equally manifest by the complete destruction of all the normal histological characters of human hair. The cortex and medulla are undistinguishable, the surface is rough, the pigment no longer normally distributed, and the free end, instead of tapering to a point or being transversely cut off, is broken, slightly bulbous, ragged, or split into a sheaf of fibres. A less degree of infection is recognised by a few spores in nucleus-like chains or a little branch of mycelium, in the substance of an apparently healthy hair. Dr Frederick Taylor has pointed out that the parasitic fungus does not invade the cutis itself, nor even the follicle, and only slightly affects the epidermis adjacent. The inner root sheath is full of spores, the outer root sheath free. (Compare his paper, 'Med.-Chir. Trans.,' lxii, with Dr Thin's, ibid., vol. lxi.)

The spores differ from oil drops, with which they are often confounded, in the following particulars: first, they are uniform in size; secondly, they do not run together; thirdly, they are not perfectly spherical, but some at least perceptibly spheroidal or oval; next, they do not refract light so strongly, and though glistening and having a well-marked outline, the centre is not so bright nor the circumference so broad and black; they occur in little groups or in chains; lastly, potash, instead of dissolving them by forming a soap as it does with oil drops, is powerless to affect their protoplasm, which is protected by a cell wall: thus it only serves to bring them out clearly by making the surrounding keratin and oily matter transparent. Ether is also without effect. Carmine and other staining agents act slowly, but in the end stain the cell. Often the most characteristic objects are not the extracted hairs, but short broken fragments which are conveyed to the glass-slip with scales and *débris*.

Diagnosis.—The recognition of ringworm is in most cases sufficiently easy after a little experience, but we must remember that it may be masked by secondary impetigo as above described; also, that when of long standing

it may produce patches of almost bald skin which may simulate the atrophic patches to be described (p. 975) as alopecia areata; and, thirdly, that the trichophyton may exist in hair which, as explained above, does not show the ordinary signs of ringworm which are visible without a microscope. In all doubtful cases, therefore, we must depend upon careful microscopical observation. This is particularly important when we have to decide whether the disease is cured or not. It is only by taking numerous specimens that we can assure ourselves of the fact. We must sometimes, where to the naked eye the ringworm has disappeared, hunt through a dozen slides without finding a single diseased hair, until in the last we may find unmistakeable evidence of the ringworm being still incompletely cured.

Ætiology.—The only efficient cause of ringworm is the growth of the *Trichophyton tonsurans;* and its almost universal spread under favourable circumstances shows that individual difference of soil has but little to do with it. All that we see are differences in the luxuriance of its growth, in the irritation it occasions, and in the obstinacy with which it clings to the affected scalp.

It has often been stated that ringworm occurs chiefly in pale thin children, who are called "scrofulous" or "strumous," without enlarged glands or any sign of tubercle. There is little evidence for this opinion, nor for its supposed predilection for light-haired, "lymphatic" children. One often sees ringworm in those who are the picture of rosy health. It occurs more frequently in light-haired children than others because most children in England have light hair, but it is common enough in those with brown hair, black hair, or red hair.

What is really important in its ætiology is that it is most frequent between the ages of three or four and nine or ten. It is not very common in infants, and when present is usually cured without difficulty. This probably depends upon the less development of hair. Why ringworm of the scalp is so seldom met with in adults is difficult to say. Not only do mothers and nurses rarely take the disease from their children, but when it does occur it is far more readily cured. In children above ten or twelve years old it is easier of cure than in younger ones, and about fourteen or sixteen years of age its treatment seldom gives trouble, and it sometimes disappears spontaneously.

Ringworm is equally common in boys and girls.

It must be remembered that the lower animals are liable to this disease, and the source of contagion may sometimes be a cat or a horse.

Prognosis.—In infants and in adults ringworm of the scalp is a very manageable disease; in children, though the majority of cases may with care and attention be cured, it often proves obstinate, and now and then, in spite of the best available treatment, may persist for years, and at last yield to advancing age alone. In a school or a family, from a third to a half of the cases will be cured in from three to nine weeks, a few of them by three or four days' application of the remedy. The majority of the rest will yield to persevering treatment in from three to six or eight months. A few only out of a large number will last beyond this time, and some of these are pretty sure to prove inveterate.

Treatment.—The principle of treatment is the same as that of scabies. In both cases we know the cause of the disease; we know the natural history of the invading organism and the means of checking or destroying it. The difficulty in the case of ringworm is that most frequently before the case comes

under our observation the fungus has already fixed itself deeply in the hair-sacs of the scalp, and it is extremely difficult to apply remedies to reach it. It is moreover protected by the epithelial scales which closely surround the hair-bulb, and by the sebaceous and other products which block its mouth. We shall see that when the same parasitic growth invades the surface of the body its cure is easy.

So great are the practical difficulties of treating ringworm of the scalp that, although with perseverance and skill we can cure the vast majority of cases, and some of them rapidly as well as safely, yet everyone who has much experience in this disease must have met with cases which are so intractable that when after many months or even years they at last get well, it is to time and the increasing age of the patient that the cure is due.

Preparations of *mercury* are poisonous to all cryptogamic plants, to bacteria as well as to fungi, and probably the most poisonous is corrosive sublimate. A solution of perchloride of mercury in alcohol, two grains to the ounce, is sometimes rapidly effectual in curing recent cases of ringworm. It should, however, only be applied to separate patches, since there is at least one case on record in which its free use over a child's scalp produced (by some unusual accident in the application, or possibly some idiosyncrasy in the patient) absorption of the drug, and death by mercurial poisoning. Lotions, however, have the disadvantage of being repelled by the oily sebaceous infiltration of the natural and diseased structures of the scalp. We therefore usually prefer lard or vaseline as a vehicle, and in early cases of ringworm the white precipitate ointment (*ung. hydrarg. ammon.*) is often completely successful. It should be well rubbed into each patch morning and evening after thorough cleansing with hot soap and water and flannel. Instead of white precipitate ointment the *oleate of mercury*,* of the strength of one in twenty or one in thirty-five, is effectual, and by many preferred to the older preparation. The 10 per cent. oleate is too strong unless applied to a very small patch in an elder child.

Another parasiticide which has become popular is tincture of *iodine*. This also is sometimes effectual with recent cases.

There is, however, another method of destroying the fungus which is often found to be practically more efficacious. It consists in setting up a local inflammation, the products of which destroy the parasite. This plan is most applicable to the first stage of the disorder. If a mercurial application does not prove effectual within a few days, then, with elder children, and especially on the first appearance in the family, it is probably better, after isolating the infected member, to attempt the immediate destruction of the fungus by exciting local inflammation. A stronger solution of iodine acts in this manner, but probably the most effectual and least painful application is the blistering fluid made of *cantharides*. The affected spot should be first shaved, including half an inch around it, and a circle of oil be drawn round the margin to prevent the blistering fluid from spreading. The pain of its application does not last long, and in many cases success is immediate and complete.

Too often, however, the fungus has already spread too far to be treated in this decisive manner, which is scarcely applicable except to recent cases with only a single diseased patch.

We will suppose that a child is brought to us with the disease established

* Oleates were introduced by Dr Shoemaker, of Philadelphia (see 'Brit. Med. Journ.,' October, 1884).

for several weeks, with numerous rings, and perhaps with crusts and pustules from attempts to cure by various irritant applications. The first step is to have the hair cut quite short over the whole of the scalp. Scabs and crusts must then be removed with the help of poultices, and the whole surface made as clean as possible. We then see the real extent of the primary disease. It is often much less than it at first appears; the secondary superficial dermatitis is readily cured, and the diseased patches are soon ready for treatment. Sometimes we find no impetiginous crusts and little active inflammation, but scattered over the whole scalp small spots of ringworm, while the apparently healthy hair between often furnishes evidence of infection. Under these circumstances the shortest and most effectual way is not merely to cut the hair short but to shave it completely off. In inveterate cases it is much better to wait until the hair is removed, the crusts or scales got rid of, and the inflamed glands reduced, before beginning active treatment. Meantime, the whole scalp should be well anointed morning and evening with carbolic oil, one in fifteen or one in twenty, and the child's head covered with a linen cap both by night and by day. In this way no time is lost, and the spread of the infection to other children is prevented. If, without much active inflammation, there is found considerable accumulation of dead epithelium, and especially when it takes the granular adherent character above described, this must be removed with potash-soap, or other alkaline applications. Dr Foulis ('Brit. Med. Journ.,' 1885, vol. i, p. 536) has recommended for this purpose spirits of turpentine rubbed in until the child begins to feel it tingle, and then washed off with abundant warm water and carbolic soap. This is sometimes a rapid and effectual treatment, but it is only applicable when comparatively small patches are affected, and should not be used in the case of young children.

When the way has thus been cleared for parasiticides, we may in the slighter cases obtain good results by rubbing into each patch the white precipitate ointment as above recommended, anointing the intermediate surface with *carbolic oil*. In many cases, however, this proves inadequate, and we must then use stronger applications, although if the disease is extensive they must be applied only to a limited portion at a time. Equal parts of unguentum hydrarg. nitratis and sulphur ointment form an efficient and usually not too severe application. Dr Alder Smith, whose experience of ringworm at Christ's Hospital has been very large, recommends in obstinate cases a mixture of carbolic acid one part, citrine ointment one part, sulphur ointment one part. With children under ten, two or three instead of one part of the sulphur ointment should be used and it will then cause no pain. Instead of carbolic oil (1 in 10 or 1 in 5) the carbolic glycerine of the British Pharmacopœia (1 in 4 or diluted to 1 in 8) is often preferred. It is preferable where lotions are being used. Another plan is to use carbolic oil (1 in 10) to the generally diseased surfaces and carbolic acid lotion to successive portions; but this is apt to produce more pain and less certain curative effects than the compound ointments.

Some writers recommend *chrysophanic acid*, which is the efficient constituent of Goa powder, much used in the East Indies.* As stated in the chapter on psoriasis, it is sometimes an extremely severe irritant, and always stains both the skin and linen unpleasantly. Chrysophanic acid has been tried, dissolved in chloroform, by Dr Alder Smith (seven grains

* Chrysarobine is the trade name of Goa powder used at Bombay. The native name is araroba.

to the ounce); and he recommends it in recent cases with only one or two spots as more successful than blistering. At the same time he uses a lotion of hyposulphite of soda (two drachms to the ounce) or of liquor sodæ chlorinatæ (one part in eight). Dr Crocker ('Lancet,' January 27th, 1877) reports careful and impartial trial of Goa powder in twenty cases of ringworm. Only eleven were slightly improved after three months' treatment and only two were cured. Another objection to chrysophanic acid is that it is apt to get into a child's eyes, especially during the night.

A better application in every way is the ointment of *pyrogallic acid*, which is much used against ringworm in Vienna.

Among the more severe applications is one introduced by Dr Coster, of Hanwell Central London Schools, and afterwards published in the 'Medical Times and Gazette,' vol. i, 1867, p. 34. This *Coster's paste* consists of two drachms of iodine dissolved in an ounce of colourless oil of tar, obtained by distillation from common tar and known as light oil of wood-tar or rectified spirit of tar, of sp. gr. 853 to 867. It is applied with a brush to the affected parts and forms a cake which separates at the end of a week or fortnight. (See a letter by Mr Martindale, in the 'British Medical Journal,' January 19th, 1880.) This was used by Dr Ringer at University College with success. Mr Morrant Baker, at St Bartholomew's, preferred iodine in the same proportion with creasote.

The most severe application is *croton oil*, which produces an artificial pustular dermatitis known as "kerion." A favourite ointment both in Germany and in France is that which is also used in the cure of scabies, a combination of sulphur with an alkali (Wilkinson's and Vlemincqx's ointment). Hardy gives the formula: Carbonate of potash a quarter to half a gramme, sulphur one to one and a half gramme, lard thirty grammes.*

Instead of ointments or aqueous solutions the cure of ringworm has often been attempted with alcoholic lotions, but without marked success. Lately, however, Dr Cavafy ('Brit. Med. Jour.,' June 24th, 1882) recommended a lotion composed of boracic acid, alcohol, and ether, in the following proportions: boracic acid twenty grains, ether one drachm, spiritus vini rectif. one ounce. The object, of course, is to dissolve the sebaceous material in the hair-sac and thus enable the boracic acid in solution to soak down to the spores which lurk there. This plan has been adopted and recommended by several dermatologists of experience. The writer has found that this lotion, rubbed into the patches not less than four times a day, has proved cleanly and painless. It sometimes effects speedy cure, but, like all other applications, it not unfrequently disappoints us.

Salicylic acid has also been employed dissolved in alcohol, ether, or chloroform; and corrosive sublimate may be used in alcoholic solution—two grains to the ounce.

If watery lotions are preferred, sulphurous acid gas in solution (*Acidum sulphurosum* of the British Pharmacopœia) is one of the best parasiticides. It must be applied on pieces of rag to each patch. Or the hyposulphite of soda (two drachms to an ounce of water) may be used.

Thymol is another unirritating parasiticide which may be employed. It is soluble in alcohol and ether.

Dr Alder Smith recommends Barff's *boro-glyceride* as one of the best applications if the scalp is tender and sore, especially if impetigo is present.

Oleate of copper is an imitation of the old verdigris ointment, as that

was of pennies laid in vinegar. It is of a bright green colour, and said to be not ineffectual.

With oleates, frequent washing is unnecessary and even undesirable. With solutions, whether in water, alcohol, chloroform, or ether, constant cleansing with common or soft soap is absolutely necessary.

Is it desirable to aid the action of parasiticides by removing diseased hairs? This plan of *epilation* is generally carried out both at Paris and Vienna, and is adopted by many English physicians. Others believe that it is ineffectual. The fact is that to pull out all the diseased hairs over an extensive surface affected with ringworm is impossible even by a skilled manipulator. A certain number are sure to break off in the forceps, and still more are too short to be laid hold of. Moreover, the attempt is extremely tedious and painful, and the result insignificant. Where, however, a very small patch is for the first time seen, it is well to pull out at once all the hairs not only from the obviously diseased skin, but from a small circle around, before applying acetic acid, blistering fluid, or any other agent by which we hope to destroy the parasite at once. Again, in chronic and extensive cases, removing loose hairs helps to prevent contagion, to clear the scalp, and also to ensure minute observation and care on the part of the nurse. It is therefore well to give her a pair of broad-tipped, well-roughened and weak-springed forceps, and to instruct her to remove every morning after washing the head as many hairs as seem to be loose, but not so as to cause the child pain.

Contagion.—While ringworm is under treatment the whole of the child's hair should be kept short, cut in fact as close to the head as may be; and this is probably as effectual as shaving. With girls a fringe of hair may be left round the forehead and behind the ears, so that a cap may be worn during the day, and the child's appearance attract no attention out of doors. At night a linen cap should be used. Impervious coverings of gutta percha or oiled silk make the scalp hot, and are unnecessary.

There is no need for a quantity of ointment to be left on the scalp at night. The free application of carbolic oil, or carbolic glycerine, or oleate of mercury to the head is best undertaken in the morning. Although mothers and nurses very rarely suffer from the most assiduous dressing of ringworm, it is well to instruct them to anoint their hands with carbolic oil each time they touch the child's scalp.

With these precautions, and scrupulous avoidance of contact with caps, brushes, &c., it is possible for a child with ringworm to be treated and cured without removal from the family. But if the infected member or members cannot be separated from those who are healthy, they should sleep in separate bedrooms, and, if possible, meet only out of doors. It generally happens that in a family, while most of the cases are cured quickly, there remain one, or perhaps two, extremely obstinate. These may, if necessary, be removed for the sake of treatment, but practically when the child has once been cured it is little liable to take the disease again, especially if the hair is kept short, if carbolic oil is used as a pomade, and if the nurse (who if at all intelligent will by this time be able to recognise the disorder) is careful to wash and inspect the scalp every week.

It is obviously wrong for a child suffering from ringworm to be sent to school, for other children to be admitted to the house, or for its hair to be cut except by its own nurse.

The only proof of complete cure is the careful microscopical examination

of the hairs, not only from the previously diseased spots, but from the surrounding scalp. When the skin is itself healthy, and the hair which grows on it is soft and downy, when no broken stumps and black points are seen under a lens, and when these good signs are associated with an absence of spores in the hairs examined, we may pronounce the child to be cured. It should, however, not be sent back to school for at least a fortnight after this, and should then be carefully examined again before the risk of relapse can be considered past.

Ringworm of the Body.—*Tinea circinata.**—This affection occurs in the form of small rings with a red, papular, vesicular, or scaly margin. They are mostly confined to the face and neck, but are sometimes seen elsewhere on the trunk. They produce very little irritation.

The disease is contagious, and if scrapings from the ring are placed in potash under the microscope, the mycelium and spores are apparent. There is more of the former in proportion to the latter than in ringworm of the scalp, and the fungus is not so readily seen, but when thoroughly soaked in potash it can always be discovered.

It often appears in children along with common ringworm, but may also be seen when the scalp is quite free from disease. It occasionally occurs in adults, especially in the form which will presently be described.

Burmese ringworm is the name given to what is described by the late Dr Tilbury Fox as nothing but a somewhat severe and troublesome form of *Tinea circinata* (see his account of this and other exotic forms of ringworm in his work on 'Skin Diseases,' p. 541).

Tinea marginata.—There is a form of tinea only observed in adults, and of which the parasitic nature was first recognised by Köbner. It was formerly called *eczema marginatum.*

Its distribution is very characteristic. Unlike all other forms of ringworm, it is symmetrical, and occurs only on the thighs, abdomen, perinæum, and buttocks. It begins, probably in all cases, with minute spots, which rapidly form rings; but, as these extend and coalesce, they produce gyrate figures, as above explained in the case of psoriasis, erythema, and other disorders which spread at the edge. When a case comes before us it has usually already assumed its characteristic aspect of a somewhat sinuous, broad, yellowish or brownish red, more or less inflamed band, which runs over the upper and inner part of each thigh, passes back to the fold of the nates, or even as high as the sacrum, and returns over the lower part of the abdomen or the groin to the pubes. This curious distribution no doubt depends upon the mutual contact of the parts, and is aided by the warmth and perspiration which favour the growth of the fungus. The centrifugal spread is that of all forms of tinea, but the central parts are sooner free from the disease, the margin is more inflamed, and the duration more prolonged than in tinea of other parts of the body.

This curious affection is apparently confined to the male sex and adult age. It is most common in those whose occupation necessitates a sitting position for a long time; thus it is most frequent in cobblers and cavalry soldiers. There is generally much irritation and discomfort; and, like all long-continued forms of dermatitis, it produces pigmentation, not only in the growing margin, but also upon the inner exhausted surface.

The microscope demonstrates the same mycelium as is found in *Tinea*

* *Synonyms.*—Herpes circinatus—Vesicular ringworm—Trichophytie circinée.

circinata; but the disease may last for ten years or more, and when of very long standing it is often difficult, and sometimes perhaps impossible, to discover the parasite. Fortunately the aspect and locality are sufficiently characteristic.

Treatment.—Tinea circinata is very easy of cure. White precipitate ointment or oleate of mercury, verdigris ointment (subacetate of copper two scruples, benzoated lard one ounce), tincture of iodine, boro-glyceride, sulphurous acid in solution—may each be employed with a certainty of speedy cure, in striking contrast with their action in ringworm of the scalp. In England there is no need for resorting to the more severe parasiticides, but in India Goa powder (chrysarobine, chrysophanic acid) was first introduced for so-called Burmese ringworm. It should certainly never be employed in the cases which come before us in this country.

Ringworm of the body is of course contagious, and may not only propagate itself, but may lead to the development of tinea in the scalp. Its easy cure, however, renders precautions by isolation almost unnecessary.

Tinea marginata, however, is, as above stated, very obstinate and difficult of cure. Sulphurous acid of the British Pharmacopœia freshly made, hyposulphite of soda (a drachm to the ounce), boracic acid (ten grains to an ounce of spirit), and corrosive sublimate (two grains to an ounce of water), may each be used with good effect.

In one very obstinate case, in which the patient, there seemed no doubt, had contracted the disease from a pair of knickerbockers which had been mended by a village tailor in Switzerland, most of these remedies were tried ineffectually for some months. At last the effect of pyrogallic acid ointment (half a drachm to the ounce of benzoated lard) was so rapid and unmistakeable that the patient complained of this cure not having been used at first.

Onychomycosis.—It is happily very rare for tinea to attack the nails. When present, ringworm of the nails is usually a complication of ringworm of the scalp. Cases were recorded by Meissner in Vierordt's 'Archiv,' by Virchow and by Bazin, as early as 1853. It was carefully described and the microscopic appearance figured by Neumann ('Hautkrankheiten,' p. 347, figs. 48 and 49), by Dr Purser ('Dubl. Quart. Jour.,' Nov., 1865), and by Dr Fagge ('Guy's Hosp. Rep.,' 3rd series, vol. xv, p. 553, and 'Clin. Trans.,' vol. i, p. 77). The nails become yellowish and brittle, but not rough as when affected by eczema or psoriasis. The fungus is occasionally that of favus (*achorion*, to be next described), but more often that of common ringworm (*trichophyton*). Good models of onychomycosis due to each of these parasites will be found in the Guy's Hospital museum, Nos. 536 and 537.

It is the most obstinate of all forms of ringworm, and will often persist during the whole of childhood, and only disappear after puberty.

The *treatment* recommended is scraping the affected nail, softening it with alkalies, and when other means fail, complete removal, together with the sedulous application of sulphurous acid or hyposulphite of soda; but in one case an eminent dermatologist adopted this method, in addition to every other possible parasiticide treatment, without curing the disease.

FAVUS.*—This is a rare affection of the scalp and body, due to the presence of a fungus named *Achorion Schönleini.*

The disease was recognised and named by Bateman, and was figured by

* *Synonyms.*—Tinea favosa—Porrigo lupinosa.—*Fr.* Teigne faveuse.—*Germ.* Erbgrind.

Alibert. But it was not till 1839 that Schönlein published in Müller's 'Archiv' the discovery that the yellow crusts of favus were neither pustular nor sebaceous, but were composed of the mycelium and conidia of a parasitic fungus. This discovery preceded that of Malmsten above mentioned (p. 962), and therefore to Schönlein belongs the merit of opening the whole chapter of cutaneous mycology.

In its earliest stage favus is probably undistinguishable from common ringworm, but very soon a characteristic flat, round, yellow object is seen, depressed in the middle, opaque, adherent, and perfectly dry. Its colour has been compared to a honeycomb (*favus*), and its shape to the disc of a lupine seed. The sight of a single case of the disease of such models as Nos. 523—527 in the Guy's Hospital museum, or even of a well-executed drawing, is sufficient to enable anyone to recognise favus.

The individual crusts grow, coalesce, and form thick, rugged, porous, yellowish masses, resembling the rind of old worm-eaten cheese. They have a characteristic mouldy odour like that of mice.

The disease may affect any part of the body, but is particularly severe upon the scalp, where it destroys the hair-sacs and often produces complete baldness. It is, as above stated, extremely rare in England, but is less so in Germany, and comparatively common in France. It appears also to be not very infrequent in Scotland. Mr Hutchinson published forty-four cases with instructive remarks upon the disease in the 'Med. Times and Gazette' for 1859 (vol. ii, p. 553).

Favus has been recorded by Dr Purser, of Dublin, in a cat (1866), and by St Cyr in rabbits and mice ('Ann. de Dermatologie et Syphilis,' 1869), quoted by Dr Fox ('Skin Diseases,' p. 431).

The *treatment* is unsatisfactory. Ordinary parasiticides produce improvement, and if perseveringly employed, apparent cure; but relapse is almost sure to occur. The old French treatment of epilation by a cap of pitch-plaster, applied to the head and then forcibly pulled off, is no more effectual than less barbarous methods; but epilation is probably necessary for even a temporary cure. Several cases of this remarkable disease are described by Dr Fagge in the 'Guy's Reports' for 1870 (p. 354), where more than one apparent cure by epilation is recorded.

Tinea Versicolor.—This affection, described by Willan as *Pityriasis versicolor*, was formerly named *maculæ hepaticæ*, a translation of the vernacular German name *Leberflechte* (liver spots, *chaleur de foie*). Another name still often applied is *chloasma*, but this is better reserved for true maculæ produced by pigment.

In 1846 Eichstädt published in Froriep's 'Journal' the discovery that this affection is due to the presence of a fungus. It is worth noticing, now that its real nature is understood, Bateman's remark, that "the causes of this pityriasis are not well ascertained; fruit, mushrooms, sudden alternations of heat and cold, violent exercise with flannel next to the skin, have been mentioned as probable causes: the most extensive eruption I have seen occurred in a Custom-house officer after drinking spirits freely during a day of fasting on the Thames."

Tinea versicolor occurs as yellowish-brown spots of various shades, scarcely rising above the level of the skin, and yielding a branny or furfuraceous desquamation when scratched. The spots vary from a pin's head to several inches in diameter. As they multiply and coalesce, they

form larger patches and then rings, which when united produce the gyrate or serpentine outline before described as the result of this mode of development of an eruption. It is rare, however, to see such perfect rings as in tinea circinata, and the central parts seldom completely recover and remain more or less discoloured.

The *distribution* of this affection is very characteristic. In the great majority of cases it occupies the chest, it often spreads to the abdomen, and is frequently seen on the back, especially between the shoulders. It may overspread the whole trunk, but rarely descends below the waist or ascends above the neck. Occasionally a patch or two may be also found on the border of the axilla and on the soft skin of the inner part of the arm and on the bend of the elbow. Even when the abdomen is not affected, it is common to find this form of tinea on the inner side of the thigh, whence in males it is apt to spread to the scrotum. We may say, therefore, that the affection never occurs upon parts which are exposed to the air, and that its favourite seat is on skin which is the most protected and the most constantly warm and moist.

On scraping some of the surface and putting the scales in a drop of potash under a microscope, both spores and mycelium can be seen without difficulty. The spores of the fungus, *Microsporon furfurans*, are somewhat larger than those of *Trichophyton tonsurans* and occur in heaps, which are surrounded by mycelium threads.

The presence of the fungus is of course the decisive point of diagnosis; but with a little experience the colour, the branny desquamation, and the locality of this affection are sufficiently characteristic.

Sometimes patients complain of the irritation occasioned by tinea versicolor, and it may be accompanied, especially in hot weather, by slight erythematous dermatitis or urticaria, as the result of scratching. Most frequently, however, it produces no symptoms whatever and is either discovered accidentally or is only regarded as a disfigurement. The superficial layers of the epidermis are alone affected by the parasite.

The *cause* of this curious affection, or rather of the fungous growth on which it depends, is quite unknown. It is remarkable that it seldom, if ever, occurs in children, and is rare after middle age. It is most often seen in men between twenty and forty, but may also be observed in women, especially under the fold of the mamma. Although the fungus has been proved by experiment to be capable of transmission by direct inoculation, the disease is not practically contagious, or if at all, to a very small degree.

Treatment.—If left alone tinea versicolor continues indefinitely, but it may be readily removed by any of the milder parasiticides. After thorough washing with hot soap and water, or, if a rapid cure is desired, with soft soap, the affected parts must be well rubbed with oleate of mercury or unguentum æruginis (℥ij ad ℥j); or, if preferred, sulphurous acid or hyposulphite of soda (℥j ad ℥j) may be applied in watery solution.

Tinea rosea (?)—Pityriasis rosea.—This affection was first described by Gibert as an acute centrifugal erythema of the trunk. Bazin called it *P rubra maculata et circinata*, Behrend, *Roseola furfuracea herpetiformis.* Vidal regards it as parasitic ('Trans. Intern. Med. Congr.,' vol. iii, p. 133). Most German writers agree in this view and class it as a variety of ringworm of the body. But Dr Liveing, who formerly looked on it as a variety of ringworm of the body described by Hebra as *Herpes tonsurans maculosus*,

is now satisfied that it is not parasitic but a form of erythema, and calls it *Roseola circinata*. Its course is rapid, and the bright red spots or circles leave slight branny desquamation and faint pigmentation behind. It needs no treatment, but it is important to distinguish it from an early syphilide.

It will be convenient to consider in this chapter the remaining affections of the hair, some of which were formerly confounded with ringworm and are still liable to be mistaken for it.

ALOPECIA.*—Baldness, or loss of hair, when not the result of the presence of *Trichophyton tonsurans*, is the immediate consequence of atrophy of the hair-bulbs, which occasions the premature fall of the hairs from the follicles. When this is only partial and followed by fresh growth of weak hair, the result is thinness or partial baldness, but when the hair-sac is no longer capable of producing a fresh hair, complete alopecia results.

Although a senile change, baldness cannot be considered strictly physiological; for it is often absent even to advanced age in men, it is usually absent and rarely complete in women at any age, and it sometimes occurs very early without any other signs of senile decay. In these cases it is frequently hereditary, but by no means constantly so.

The atrophy of the hair-sacs certainly does not depend upon general deficiency of healthy nutrition, nor upon locally deficient supply of blood. It is not accompanied by anæsthesia, by numbness, or by any other evidence whatever of nervous disorder, so that to ascribe alopecia, whether premature or not, to "vascular" or "neurotrophic disturbance," is an arbitrary hypothesis. It has been asserted that adhesions of the pericranium, and particularly want of mobility of the aponeurosis of the occipito-frontalis, produces alopecia, but many instances disprove the assertion. Neither wearing tight hats, nor going without hats, nor wearing turbans indoors, nor exposure to the sun—nor gout, nor scrofula, nor intemperance, nor abstinence—none of these will in the least explain either senile or premature baldness, for each supposed cause fails on examination.

Alopecia of this quasi-physiological character begins usually in the frontal region, sometimes at the central point at the back of the head from which the hair falls forward, backward, and laterally; and not unfrequently, in both regions at once. There is often seborrhœa sicca or a slight degree of pityriasis which precedes and accompanies baldness; but if this is the cause, the thinness of hair can be cured by restoring the skin to a healthy condition, and even if neglected it does not go on to complete alopecia. Moreover, in many cases both of senile and premature baldness the skin is healthy throughout. When the hair has fallen off from the mid region of the scalp, the process almost always ceases, and that on the temples, behind the ears and on the occiput persists without change. This ordinary alopecia, moreover, never affects the beard, the eyebrows, or other parts of the body.

Many attempts are from time to time made by physicians, as well as by hair dressers, to check the loss of hair or to restore it. They consist either in applying stimulating lotions of which cantharides is usually the basis, or in shampooing and manipulating the scalp. It is very rarely that these

* *Synonym.*—*Calvities*, or more frequently in classical Latin *calvitium*. *Atrichia* is a modern name. *Alopecia*, *ἀλωπηκία*, fr. *ἀλώπηξ*, "quod vulpes hoc malo sæpe corripiatur," is the real Greek name. Celsus distinguishes it by occurring in patches and only in adults from *ὄφιασις*, which spread in a serpentine form on the back of the head in children.

attempts have even partial success. A process introduced a few years ago by Dr Pincus, of Berlin, promised better, but has not in the sequel fulfilled the expectations of its author.

Alopecia as the result of febrile diseases.—Although this often proves the first step of ordinary baldness, yet it is distinguished therefrom by its affecting both sexes and all ages, by the fall of hair not being confined to any region of the scalp, and by its thinning rather than completely stripping the surface affected. Moreover, it is not only secondary in origin, but usually passes away of itself after convalescence, instead of being practically incurable, either by nature or by art.

Syphilitic baldness agrees in these characters, and its frequency, apart from any other affection of the scalp, as well as its early appearance, likewise point to its ætiology as a febrile alopecia.

AREA.*—So peculiar is the appearance of this disease, that it is less needful to insist upon its distinction from other kinds of alopecia than upon the fact that it is a true alopecia, anatomically identical with the other forms of atrophy of the hair, though differing in its origin and course. It is independent of the presence of a fungus.

Hebra believed at one time in the statement of Gruby that the disease was parasitic, but before long changed his opinion, so that one can only share in the surprise expressed by Dr Kaposi, that Hebra is associated with Bazin as a supporter of the parasitic nature of area by his disciple Dr Neumann. It is possible that the single observation of Gruby in 1843 ('Comptes Rendus,' xvii), which gave rise to the question, was made upon a case of true ringworm. Neumann, who has no doubt that area is not parasitic, once, like the writer, found some spores in a case of the disease, but doubts rather the significance of a single observation than the accumulated testimony of his own and others' experience. In fact M. Bazin's statements (and those made recently by Malassez and by Eichhorst) are the only ones which rest on large experience and assert the presence of a fungus. The French dermatologists call many cases "pelade" or "teigne pelade," which in England or Germany would be regarded as true ringworm in its later stages. In M. Hardy's lectures it is not difficult to recognise in the swelling, irritation, and desquamation of the skin, which he describes in pelade, the characters of ringworm.

Apart from the microscopic evidence, the naked-eye appearance and natural history of the disease would almost disprove the parasitic hypothesis. The hairs around the affected spot are not swollen at the root, nor brittle in the shaft, but are merely atrophied like normal hairs which are ready to fall out. There is no evidence of local irritation in the hair-sac. The dis-

* *Synonyms.*—Alopecia areata (Sauvages)—Area Celsi—Porrigo decalvans (Willan)—Teigne pelade (Bazin)—Tinea decalvans.—The first of these titles appears to be best, since it is distinctive and is generally accepted. Celsus did not particularly describe this variety of baldness, but applied the word "area" ("a bare space," *locus sine ædificio*) to any form of baldness, distinguishing *ἀλωπηκία* and *ὀφίασις* as varieties. The "porrigo" of Willan meant any eruption of the scalp, including true ringworm and impetigo or pustular dermatitis, and the term is now almost out of use. The appellation Tinea or Teigne depends upon the erroneous doctrine of the parasitic nature of the disease.

I have many times sought for a fungus and have never found the smallest evidence of its presence, with one single exception. This occurred nearly twenty years ago when working under the late Professor Hebra. In one of his patients suffering from area discovered some spores and scanty mycelium in one of the neighbouring hairs. I showed it to the professor and he told me that he had never seen it before. He doubted whether its occurrence was more than accidental, and with my present experience I doubt it also.

ease, above all, is not contagious, at least as we observe it in England, and it is not curable by anti-parasitic treatment.

Dr Thin ('Proc. R. Soc.,' 1881, No. 217) has figured minute schizomycetes, which he calls *Bacterium decalvans*, but which are rounded rather than rod-like, and probably identical with those described by Dr v. Sehlen in 'Virchow's Archiv.' Even if this were of ætiological importance, it would not make area a true tinea.

Another theory is that area is a tropho-neurosis, for which there is no sufficient evidence. The subject was discussed in the International Medical Congress of 1881 by Hardy and Vidal, Kaposi, Liveing, and others (vol. iii, p. 158).

Area is certainly more common in children and young adults than after thirty.

Among 101 patients under the writer's care 41 were children from four to fifteen, 45 were young men or women from sixteen to thirty, and 41 were above thirty, one being forty-seven and one fifty-eight. In the last case area supervened after ordinary senile alopecia had begun to appear, and the two affections were perfectly distinct.

Of these 101 cases of area, 68 occurred in male and 33 in female patients.

There were several cases of recurrence of the affection in the same patient, and three of its appearance in two or more children in the same family (see Dr Tyson's case, 'Clin. Trans.,' 1886).

In many cases area probably would pass away of itself, but recovery is often hastened if not brought about by treatment. This consists in local irritants, and, when necessary, internal corroborants. We may begin with a lotion containing ʒss to ʒij of acetum cantharidis to a pint of water. This will often cause slight erythema in children, but in adults and in many children we may increase the strength to two, three, or four ounces with advantage, letting the irritation subside whenever it goes beyond redness. A mild and often efficient application is *linimentum myristicæ* (one part of the expressed oil to three of olive oil). With brown hair the *unguentum iodi* of the Pharmacopœia is a useful application.

Area occurs in persons of all degrees of health, complexion, and temperament; but if the patient is pale and thin, steel is certainly useful, and bark or cod-liver oil may be prescribed when indicated by some other symptom than the bald patches.

A second or third attack of area sometimes follows after the first had been completely cured and an interval of time had elapsed.

Universal alopecia.—There are some cases of complete and rapid loss of hair which are neither senile, syphilitic, nor febrile, and which cannot be classed as examples of area. They are distinguished first by the hair falling off almost simultaneously from the whole of the scalp, not gradually from certain regions as in ordinary baldness, nor by the confluence of separate patches as in area; secondly, by the baldness not being confined to the scalp (nor even to the scalp and beard or eyebrows as is occasionally the case in area), but affecting the whole of the body; thirdly, by its not following an illness.

In one case of this kind the patient was a young man in robust health, and wearing a full beard. Without any assignable cause he lost the whole of the hair of his body in a very short space of time.

This universal alopecia occurs in both sexes, always beginning in adult life, and always in young adults. It is quite incurable.

We may at present distinguish these somewhat rare cases of *alopecia universalis acquisita* from the still rarer cases of *congenital alopecia.* In these the nails, as well as the hair, are affected; and like other deficiencies of development, the condition may be hereditary. Such cases are comparable with congenital ichthyosis, especially in such marked examples as the "porcupine boy;" and still more closely with the "hairy family" of Burma, and the blue and hairless horse exhibited a few years ago in this country.

A striking series of examples of this form of baldness occurred five years ago in this hospital under Dr Fagge. It is remarkable that the development, both of hair and nails, was tardy and imperfect, but not absolutely deficient. The italic letters denote the female sex, as in Mr. Galton's nomenclature.

F. Born without hair or nails. Hair began to grow when he was about twenty-three years of age, and at thirty he had a full head of hair. The finger-nails also grew after puberty, but were always ill formed, and he never had toe-nails. *F.* Normal.

B. 1. Born without nails or hair; the former appeared while teething, the latter when she was ten years old. *n.* Born without hair and nails; none yet grown.

B. 2. Born with hair but without nails; died, aged seven. B. 3. Born without hair or nails; died, aged five months. B. *B.*, 4—9. Born with normal hair and nails.

B. 10. Born partly bald with ill-formed nails; he is now twenty-two and has a fair head of hair, but his nails are not good.

The patient herself, then nineteen years old, the eleventh and youngest of this large family, was born without hair or nails. She had, in 1876, only thin lanugo on the scalp and imperfect nails.

Trichoclasia (*Wilson*).—A singular disease of the hair which has been described under this title, and also as *fragilitas crinium* and *Trichorrhexis nodosa* (Kaposi), is characterised by each hair dilating at intervals and breaking at these enlarged points. The dilated node consists of separated cortical fibres which look very much like the splitting and enlargement of a cane when broken across, and the air which enters between the fibres makes them appear white by reflected light. They have thus a superficial resemblance to the ova of pediculi.

It is almost always confined to the beard, is non-contagious and non-parasitic. It was described by Devergie as "trichoptylose," and subsequently by Beigel and by Wilks. The writer has seen three or four cases of it, one of which he figured in the 'Pathological Transactions' for 1879 (p. 439), where references will be found to the scanty literature of the subject.*

This is apparently quite distinct from a parasitic affection of the hair known as "Piedra" from its stony hardness, which occurs in the hair of the scalp, among women only, in Central and South America. This has been described by several French writers and by Mr Malcolm Morris in the same volume of the 'Pathological Transactions' (p. 441).

* See also a valuable paper, with fuller references, by Dr T. C. Fox, in the 'Lancet,' Dec. 7th, 1878, and Hans v. Hebra, loc. cit., p. 391.

CHRONIC DEEP INFLAMMATIONS AND HYPERTROPHIES

Deep and chronic dermatitis—its definition—its relation to eczema and other forms of superficial dermatitis—to hypertrophy—and to new growths.

GUTTA ROSEA.—*Origin in recurrent erythema — Development — Localities—Causes and pathology—Relation to dyspepsia—to drink—to ovarian irritation—Treatment.*

EPIDERMIC AND PAPILLARY HYPERTROPHIES.—*Callosities and corns—Leucoplacia lingualis et buccalis—Warts—Condylomata and mucous patches.*

ICHTHYOSIS.—*Anatomy—Varieties—Xerodermia—Prognosis and treatment—Ichthyosis intra-uterina—Horns.*

SCLERODERMIA.—*History—Description—Distribution—Histology—Diagnosis—Prognosis and treatment—Sclerema neonatorum—Linear atrophy.*

ELEPHANTIASIS.—*Nomenclature—Anatomy—Pathology—Relation to chyluria and filaria sanguinis—Clinical characters—Dermatolysis.*

XANTHELASMA.—*History — Course and symptoms — Histology — Relation to jaundice.*

As stated in the introductory chapter, the great majority of affections of the skin consist pathologically in superficial inflammation, that is to say, inflammation which affects only the papillary layer of the cutis and the Malpighian layer of the epidermis, with the resulting change in the cuticle. In no form of this superficial dermatitis are the papillæ destroyed; and no scars result. We have now to speak of a far less frequent kind of inflammation of the skin which involves, if it does not originate in, the deep layer of the cutis, which destroys the papillæ, which spreads from the skin proper to the subcutaneous connective or adipose tissue, and which after recovery leaves scars behind. Eczema, psoriasis, and their allies, scabies, the erythematous eruptions, and the parasitic affections—are all, in the sense in which the word is here used, superficial; and however severe and protracted their course, when cured, they leave either no trace behind or only a pigment spot.

It is true that when inflammation occupies the deep sacs of the hairs and the sebaceous glands a cicatrix is not unfrequently the result.

Thus acne in its severe forms leaves scars behind, varying from white spots, very slightly depressed and otherwise inconspicuous, slight local atrophies, up to the hypertrophied scars which sometimes simulate cheloid. The same applies to sycosis, though obvious scarring is less frequent. Some other pustular diseases destroy the papillæ and thus produce scars. This never occurs with true impetigo (which is one of its pathological as well as diagnostic characters) nor with the pustules of scabies or bullæ of pemphigus; but variola, when unmodified by vaccination, almost always leaves indelible traces of its presence—either deep-pitted, depressed, white scars or more extensive and hypertrophied puckering. The same is true, though less constantly, of varicella, and the deeply pitted cicatrix is the well-known mark of successful vaccination. Lastly, the pustules of zona

very often (though by no means constantly) leave more or less marked cicatrices and sometimes, especially upon the forehead; these are deep and indelible.

But beside these deep pustules, we meet with inflammation of the skin which, uniformly and over large surfaces, penetrates below the papillæ and affects the whole thickness of the integument, together with the subcutaneous tissue. Such *deep dermatitis* is usually chronic in course; or, if it shows acute characters, they are repeated again and again, without any tendency for the malady to come to a natural end. Such recurrent subacute diseases become practically chronic, as we see in the case of inflammations of the bronchial tubes, of the eye, and of the colon.

Like other chronic inflammations, those of the skin show in many cases little of the classical signs of the process and are unattended with fever; moreover, the exudation is never purulent, but if œdematous gradually assumes the characters of *œdema durum*, if congestive, those of hypertrophy. The inflammatory corpuscles, instead of dying and undergoing transformation into pus-cells, become organised into connective-tissue corpuscles, and gradually form fibres. Thus chronic inflammations are closely related to, and often undistinguishable from, *hypertrophy* in the humbler stages of that process, hyperplasia of the connective tissues.

Again, chronic inflammation is apt to lose the uniform and characteristic qualities which distinguish the catarrhal, adhesive, and suppurative forms of acute inflammation as described in the first volume (p. 56). Thus chronic catarrhal broncho-pneumonia is apt to assume a *caseous* form and ultimately to lead to the new growths which we call tubercle. Thus chronic inflammation of the urinary tract often ends by becoming caseous. Thus also, chronic deep dermatitis not infrequently acquires a tubercular character.

Moreover, the continued irritation which gives rise to inflammation and thickening of the mucous membrane of the tongue, the lips, the pylorus, or the rectum may in time, by almost imperceptible stages, pass into a *new growth*, perhaps of the most markedly "heterologous" and malignant kind. Warts and other innocent growths, condylomata and syphilitic nodes also arise from and are complicated with chronic dermatitis and cutaneous hypertrophy.

It is therefore pathologically justifiable to associate with *chronic deep inflammations* of the skin, *hypertrophies*, *tubercle* and *new growths*; and this arrangement we propose to follow.

The only important instance of *acute deep dermatitis* is that afforded by erysipelas, which has been already treated as a specific disease in the first volume. The deep and acute inflammations which result from burns and other injuries are best studied in surgical text-books.

Gutta rosea.*—This affection in its more obvious forms is well known beyond professional circles. A classical instance of it provoked the well-known descriptions by the English knight and the Welsh squire: "the lanthorn in the poop," "an everlasting bonfire light;" "his face all bubucles and whelkes and knobs and flames of fire," "sometimes blue and sometimes red."

Short of Bardolph's degree, it is not uncommon to see gutta rosea, and it is far from being always the result of intemperance.

* *Synonyms.*—Acne rosea—Couperose—Acne congestive (Hardy)—Erythema angiectaticum (Auspitz).

Course.—The affection begins with slight erythematous redness, usually of the tip of the nose, occurring after food and combined with local irritation; the heat and itching are felt by the patient, the redness and even slight swelling are visible. It passes off quickly and perhaps may not return for days or weeks, but gradually becomes more frequent, until it is at last habitual.

The next step is for the congestive vessels to fail to recover themselves in the intervals between the successive states of hyperæmia. What was a recurrent subacute erythema becomes a chronic dermatitis with exacerbations. Frequently-recurrent œdema has moreover ended in hypertrophy, so that the skin and subcutaneous tissue of the affected part are swollen and thickened. Some of the veins from habitual distension become varicose and remain visible as red tortuous lines. The sebaceous glands are apt to be obstructed or to inflame without obstruction, and pustules resembling those of inflamed acne result. Hence the common name "acne rosacea." But there are no precedent comedones, and the distribution, ætiology, and entire natural history of the disease are distinct from those of acne.

Hypertrophy may go on until great pendulous masses of thick skin, with the scars of past pustules and abundant fibrous tissue, form hideous excrescences upon the nose, growing either from the tip, from the alæ, or from the septum.

Distribution.—By far the most frequent seat of gutta rosea is the nose, but it is not the only one. In persons in whom this feature is characteristically affected we usually find large red pimples with inflamed base and chronic course upon the cheeks, the chin, and other parts of the face. When the nose is only slightly affected and the rest of the face decidedly, the appearance is very different from that of the hypertrophied form above described when confined to the nose, but the anatomical condition is essentially the same, and every gradation between the two forms may be observed. Beyond the face, similar recurrent erythema, with more or less of hypertrophy, may be seen in the lobes of the ears, although here it is very rare to see pimples or pustules. We never find a corresponding condition of the shoulders or chest, as we do in acne.

Causes and pathology.—Gutta rosea is no less distinctive in its ætiology than in its anatomy and distribution. It is essentially an erythema, or rather the result of frequently-recurring erythema. At p. 669 was described the congestive erythema with œdema and hypertrophy which affects the extremities when from any cause the circulation in them is inactive. The difference between chilblains and their allies and gutta rosea is anatomically that the former is a passive cold venous congestion, and the latter an active, hot, arterial hyperæmia. Like other erythemata, gutta rosea is symptomatic (p. 668); it is never the result of local irritants, it always depends upon reflex inhibition of vaso-motor nerves causing active congestion. We saw that the origin of this reflex action is in some of the most marked forms of erythema irritation of the primæ viæ by poisons, drugs, or food. Gutta rosea is no exception to this rule; almost always in men, and most frequently in women it is the result of gastric irritation.

Common notoriety affixes the stigma of drink to the possession of a nose like Bardolph's, but it would be no less unjust than uncharitable to assume this as the necessary cause. No doubt the excessive use of alcohol produces most frequently and most readily the gastric irritation which leads to gutta rosea; but marked examples of the disease may be seen in

persons of habitual temperance, and even in total abstainers. In women, especially at the period of the menopause, there is apt to be a form of dyspepsia which leads to flushings of the face, not only after every meal, but in the worst cases, upon putting the first morsel of food into the stomach; these flushings are felt by the patient and cause great distress. The frequently-recurring hyperæmia leads to habitual congestion, pimples, and at last more or less hypertrophy, although in these cases the type is more often that of diffused redness with pimples scattered over the face than of marked local hypertrophy. This, however, is occasionally seen, just as the diffused pimply redness is often the result of tippling.

The only cause for gutta rosea, beside alcoholic or non-alcoholic dyspepsia, is uterine or rather ovarian disturbance. The frequency with which the disease occurs when menstruation is becoming irregular before it finally ceases seems to make this probable. In many cases of gutta rosea the affection is decidedly worse at the menstrual periods, and is associated with dysmenorrhœa. Considering, however, the great number of cases of menstrual disturbance in which no such effect is produced, and the extreme rarity of gutta rosea even in the worst cases of dysmenorrhœa in young women, as well as the frequency of what may be called climacteric dyspepsia, it seems probable that in almost every case gastric irritation is the exciting cause of the disease, and that the monthly exacerbations which undoubtedly occur in certain cases are due to direct physiological vascular excitation at that time of the whole surface rather than to morbid irritation of a reflex kind from the ovaries.

Gutta rosea is not produced by the most frequent kind of dyspepsia, that of young adults; it is rare before the age of forty, even in persons who drink freely. It is often combined with acute dyspepsia, and with gout, but is the result of dyspepsia which, like the gout, is produced by overfeeding and over-drinking rather than directly connected with excess of urate of soda. Gutta rosea is very far from being confined to the male sex, though the most typical cases from alcohol are of course more frequent in men.

Treatment.—The rational treatment of this, as of every other disease, depends upon recognising its pathology and origin. The first indication is to remove the gastric irritation which is almost always present, to discover if possible its cause, whether in excess of food, in imperfect and hasty mastication, or in some particular article which acts as a poison. Salt meat, spices, pickles, melted fats, and sauces, any of these may prove to be the offender; but most frequent of all are wine, beer, or spirits. If we fail to discover the cause of the dyspepsia, we may yet do good, apart from diet, by the exhibition of small doses of soda with rhubarb and calumba, or when gastralgia is marked, by ten grains of subnitrate of bismuth as a powder either before or after meals, to which five grains of carbonate of soda may be added if there is obvious acidity. Gentle laxatives are often desirable and occasional doses of blue pill. In some cases euonymin is particularly valuable, taken in doses of two or three grains every other night; in others a dinner-pill of colchicum and nux vomica with extract of aloes is found useful. Locally, astringent washes, like Goulard lotion, are useful and pleasant; flexible collodion may also be painted over the congested parts at bedtime. In advanced cases, scarification by innumerable punctures with a lancet is sometimes efficacious; and very successful cases have been reported by Mr Squire and Dr Stowers. When the hypertrophied masses are considerable they can only be removed by the knife.

Epidermic Hypertrophies.—It was observed by John Hunter that internal pressure produces atrophy, as when a tumour or aneurysm presses upon a vertebra; but that external pressure produces hypertrophy, as in pressure upon the skin of a labourer's hand. It is better put by Paget, that continuous pressure produces absorption and atrophy, intermittent pressure produces hypertrophy.

When pressure is continuously applied, as to a lady's foot in China while still growing, atrophy takes place with only moderate distortion of the bones and without thickening of the skin; but when it is applied only while walking, as by the narrow-toed and high-heeled shoes of a European lady, there ensues, along with a certain amount of distortion, hypertrophy or thickening of the prominent parts of the skin. This is usually accompanied with a chronic deep dermatitis whereby the papillæ are affected and a new growth forms or occasionally a deep bursa results. These products, in which chronic inflammation, hypertrophy and tumour are seen at their point of junction, we know by the names of corn and bunion.

When without unnatural pressure or distortion, the hand or foot or any other part is exposed to intermittent pressure, the result is something short of this. It is a pure hypertrophy affecting only the epidermis (*callositas, tyloma*). Such is the case in the thickened skin of the ball of the foot and the heel in adults, and of the palm of the hand in all who do manual labour. In children the thickening is but very slight, probably an inherited character, since we find it in all plantigrade animals. In adults the degree of it varies with the habits of the individual. This most purely physiological form is seen in those races who go barefoot, for wherever shoes are worn there is a chance of corns appearing even on the sole of the foot. Precisely similar callosities appear in the middle of the palm in workmen who use screwdrivers, gimlets, and augers, in the cleft between the finger and the thumb in shoemakers, and others who habitually pass a strap or cord in this position, over the patella in those who frequently kneel, and on the back of the neck, especially over the sixth vertebra, in those who carry burdens on their shoulders, as may be often observed in railway porters.

In such a callosity it will be found on section that the horny layer or cuticle is enormously increased, the Malpighian layer slightly, if at all, and the cutis vera quite unaffected. Hence these callosities appear lighter than the rest of the skin in negroes.

The corn (*clavus*), as was first shown by the anatomical researches of Gustav Simon, consists of a diseased growth of the horny cuticle into the subjacent living Malpighian and papillary layers. The horny downgrowth is of a more or less conical shape, and causes atrophy of the immediately adjacent papillæ, but at the same time a thickened layer of cutis forms around by true chronic inflammation. Here the cuticle is but slightly thickened, and not hard as in the central part, and the papillæ become gradually hypertrophied. Occasionally the original central hardening appears never to take place, especially in the soft parts of the skin between the toes, which are continually in contact and moistened with perspiration: the result is what is known as a "soft corn." There may be a mere horny plug pressing on the skin beneath, without exciting inflammation around, as occurs most frequently on the naturally thickened skin of the ball of the great toe or heel. In this case the resulting pain is that of an occasional sharp prick, when the sharp, hard, horny plug is suddenly driven home by accidental pressure—is very different from the

continual, tender, wearing, and disabling pain of a clavus mollis. The commonest kind of corn, partaking of both characters, combines the discomfort of each.

When a cyst or bursa forms beneath the corn and increases so as to become obvious it is called a bunion. A small cyst is often to be found beneath an ordinary corn of old standing and large dimensions, but the large cysts seldom form except over the metatarso-pharyngeal joint of the great toe, when this has been rendered artificially prominent by the distortion of short, narrow-toed, and high-heeled shoes. The bursa from time to time inflames, and the tension then occasions severe pain, although suppuration is rare.

Treatment.—The proper treatment of corns is prevention. Children's shoes should be made low in the heel, broad in the tread, straight on the inner side, and each shoe markedly unsymmetrical. In measuring for shoes, or for making a last, one should not sit but stand, so that the weight of the body may expand the foot into the natural shape and size which it then assumes. In a perfect covering for the foot similar expansion is afforded by the elasticity of the upper leather, and the yielding of a thick and soft stocking, but the sole should slightly project, so as to equal the largest length and width of the foot. Even in adult life the trouble of insisting upon boots being properly made is well repaid by the increased comfort and ability to walk, and the disappearance of acquired corns and distortions. In bad cases it is well for the patient to wear stockings with divided toes like a glove, and to have a stout vertical piece of leather fixed so as to separate the great toe from the rest, and to press it outwards into its natural position, a plan devised by the late Aston Key.

Beside removing the thickened epidermis and extracting the conical plug of hard keratin from time to time, relief may be obtained by treatment with salicylic acid, 2 per cent., mixed either with mutton suet (the ointment in use in the German army), or in the stronger proportion of five or ten grains to the ounce of vaseline, or as a plaster, as recommended by Dr Unna. See Dr Thin's paper ('Clin. Trans.,' xvii). Where, in addition to soft corns, the whole foot is tender and painful, the remedy consists, first, in large and low shoes, so as to diminish the heat and moisture; secondly, in thick and loosely knitted stockings, which are at once absorbent and pervious; and, thirdly, in soaking the foot night and morning in alum lotions, or brine, or solution of tannin. Thread and cotton coverings for the foot should never be worn. When wool or merino-mixture cannot be borne, silk is the only proper substitute.

Until comparatively lately the shoes supplied to the English army were symmetrical, that is to say, there was no difference between right and left. This is now happily corrected, owing to the efforts of Dr Parkes and other medical reformers; at present our soldiers are probably better shod than the French with their shoes and gaiters, or the Prussians with their high boots. The best foot covering of all is, perhaps, a kind of sandal worn by the Spanish infantry. In the handsome and serviceable costume of the Hungarian army, an excellent laced boot is worn, much like that of our own troops, but somewhat higher, like a shooting-boot, without the addition of a leather legging. The importance of anatomical knowledge in army clothing is conspicuous in this instance; but in civil life, apart from artistic considerations, the misery and ill-temper produced by ill-fitting shoes render the subject one of serious importance.

*Leucoplacia buccalis.**—Closely allied anatomically to corns and callosities, consisting, like them, in hypertrophy of epithelium, are the milk-white patches or corns upon the prominent parts of the heart, both auricle and ventricle, and the thick gristle-like white fibrous patches on the surface of the spleen, with similar conditions less frequently met with in the pleura and the peritoneum.

Still more closely connected with corns are the white patches upon the mucous membrane of the tongue and inner lining of the cheeks. These patches, which have been described as "psoriasis of the tongue," ichthyosis, are of much diagnostic interest. They sometimes occur as the result of irritation from a rough tooth. They also are produced, or at least aggravated, by smoking, not by the chemical action of nicotin, but by heat or by friction, or by the two combined. Very similar patches may be the result of syphilis, but these may generally be recognised by their being unsymmetrical, and not confined to the mucous membrane but dipping beneath it; moreover, in most cases there is either an ulcer on the patch, or more or less contraction around it from previous loss of substance. The diagnosis from syphilis, however, is sometimes difficult.

These white patches may be the seat of subsequent cancer: they are not its first stage, for they may last many years before malignant action appears, but they are the seat of irritative proliferation of cells, which only needs a determining condition, whatever it be, to produce carcinoma.†

WARTS.—*Verrucæ-papillomata.*—These are small cutaneous tumours consisting in overgrowth of the papillæ of the cutis.

A vertical section shows that the horny layer of epidermis is unaffected or is somewhat thinner than usual. The Malpighian layer is sometimes slightly thickened, and in many cases is the seat of more abundant pigment than usual. There is seldom or never any evidence of inflammation, the process is one of hypertrophy and new growth.

Warts are very rarely painful, but their removal is desired from their unsightliness and also because of their inconvenience, or sometimes the pain occasioned when they are accidentally pressed upon. They are sometimes single, more often multiple, and in rare instances occur in innumerable multitudes. They appear to be never congenital, but are most common in children, and are comparatively rare after early adult life. We can sometimes trace their origin to certain definite sources, usually some form of local irritation.

The most common seat of warts is on the hands, not the palm, but the fingers, the dorsum, and the wrist. They may also occur on the arms, the face, not unfrequently on the scalp, and more rarely on the trunk or lower extremities. They are decidedly rare on the feet, but are not uncommon on the penis and vulva, around the anus, and at the orifice of the lips and on the mucous membrane of the mouth. A similar condition occurs also in the œsophagus, especially in certain cases where pressure has produced irritation of the mucous membrane, and also where chronic cardiac disease has led to its habitual congestion. Warts are usually of a rounded, hemispherical, or pointed shape, but sometimes are flat at the surface, and by growth

* *Synonyms.*—Ichthyosis linguæ—Psoriasis linguæ—Leucoma—Tyloma—Keratosis linguæ et oris.

† See the discussion on this affection at the International Medical Congress, 1881 (vol. iii, p. 171), introduced by Dr Schimmer, of Buda-Pesth.

or coalescence a large flat warty mass may be formed which is called a condyloma.

Pathologically we may recognise the following varieties of papillomata:

1. The innocent and painless warts of youth, chiefly occurring on the hands, easily removed, not recurrent, and if left alone probably disappearing of themselves. When one of these appears others quickly follow, and their prevalence among children of the same age has led to the popular belief that they are contagious. They are almost always found upon the hands.

2. Small multiple warts, usually of a pinkish colour, and thus differing from the pale or yellowish tint of those first described. Beside their small size and colour they differ in their often occurring in large numbers so as to simulate papular dermatitis, or, again, if rather large, discrete, and somewhat flat they may simulate molluscum, a variety of which has been named "verrucosum" from this resemblance. These multiple warts are usually seen covering the arms, but may also be met with on the neck, face, and forehead. In one case, in a girl of eighteen, they covered the back of both hands, in another, of a healthy woman of twenty-eight, they closely resembled lichen planus. In a third, a young man of twenty, they occupied the neck and left side of the nose, where we counted more than three dozen. He had also warts, though less numerous, on both hands and forearms. They all came in six months, starting with one large one on the pomum Adami. This patient had had warts on his thumbs when a boy.

3. Warts of old age (*Verruca senilis pigmentosa*), usually single or few in number, large and deeply pigmented. They are apt to occur around the orifices of the body, on the eyelids, the lips, the genitals, and around the anus. Similar papillomata occur on the tongue and mucous membrane of the mouth. They are very liable to degeneration and are often the seat of subsequent epithelial cancer after having existed for months or years without showing the slightest malignancy.

4. *Condylomata and mucous patches.*—The composite warts, known as condylomata, hard condylomata, Spitzcondylom (*C. acutum*), are true papillomata in structure, but are always local, never scattered about as true warts are. They occur most frequently about the anus and genital organs. They are certainly not always syphilitic. They may follow the irritation caused by the discharge of a soft chancre or a gonorrhœa, in the latter case being identical in all but size with gonorrhœal warts. They may also occur in the cleft of the nates as the result of friction from riding, when there is not the least probability of other than mechanical origin.

On the other hand, the soft condylomata (*plaques muqueuses* or mucous patches) are believed to be always syphilitic, and they almost alone of secondary lesions have the power of transmitting the virus. They occur on the lips, on the mucous membrane of the tongue, cheeks, palate, and tonsils, occasionally on the eyelid, sometimes on the female mamma, and frequently around the anus and vulva. Here they may grow to great hypertrophic masses, the tertiary *syphilis vegetans* of authors. Their treatment will be found mentioned in the account of syphilitic affections in the first volume.

Ichthyosis.—This is a very remarkable and in its fully developed form a rare affection. It is an example rather of hypertrophy and malformation than of chronic inflammation. It was classed by Willan among his squamæ, and its name, "the fish skin disease," was given for the same reason. The

scales of ichthyosis are, however, very different from the branny desquamation which follows all superficial dermatitis, from the large pearly coherent scales of psoriasis and from the thin squames of pityriasis rubra. In the most marked cases the surface of the skin rather resembles the rough, dark, and scaly surface of the bark of a tree, or it may be compared to the rugged hide of an elephant. Sometimes the roughness and horny excrescences are so marked that they rather resemble the prickly skin of certain sharks, so that persons affected with an extreme degree of ichthyosis have been exhibited as "porcupine men" (*Ichthyosis hystrix*).

The disease is congenital, but does not appear until infancy is past, although the mother will generally admit that the infant's skin was from the first more smooth, dry, hard, and shining than that of other children.

Ichthyosis is most marked upon the limbs, but its characteristic feature is that it is practically universal. In a fully developed case no portion of the whole body is absolutely healthy. The parts least affected are the scalp, face, palms, soles, genital organs, and the flexures of the joints, in other words, the thinnest portions of the skin. The greatest accumulation of horny epidermis is on the outer side of the arms and legs, and especially about the elbows and knees, but the back, the buttocks, and the whole of the trunk are often scarcely less affected. The scales are not large and are more adherent than those of psoriasis, so that considering the thickness and extent of the mass there is less free desquamation than one expects. The surface is dry as well as rough; there is almost complete absence of perspiration; the sebaceous glands, instead of their natural lubricating oil, secrete a thick material (seborrhœa sicca) which helps to form the bulk of the crusts and gives them more power of attracting and retaining dirt. The difficulty of keeping the rough scaly skin clean is extreme, so that children affected with it have a dingy appearance, which in some cases and in the worst parts becomes almost black. The name "ichthyosis nigra" has been very unnecessarily applied to this condition. There is no deposition of pigment, and mere friction will sometimes rub off the superficial dirty scales from the most exposed parts, and leave a grey abraded surface which is characteristic and hideous enough. The thick dry skin is apt to crack in a somewhat regular square fashion like the skin of certain kinds of armadillo, and these cracks may penetrate to the cutis and become painful bleeding rhagades.

Except for this accident, ichthyosis is completely painless and apparently does not affect the general health. One or two children under the writer's care affected with it have been remarkably plump, rosy, and in other respects well developed and healthy.

As above remarked, ichthyosis is a congenital disease or rather malformation, and it is not unfrequently seen in families, as in the famous case of John and Richard Lambert, two brothers who were exhibited as the porcupine men, and whose father is said to have had a similar state of skin.

Histology.—On vertical section the diseased masses are seen to consist of beautifully arranged wavy layers of horny scales exactly like those of the thicker parts of the cuticle. A section of the skin shows that the Malpighian layer of epidermis is proportionately small, and that the ridge-and-furrow cells ("prickle cells") have more or less completely disappeared; in other words, the keratinous transformation of epithelium is here more rapid than usual. The cutis is completely unaffected. Contrary to the state-

ments of earlier writers, the independent observations of Fagge and of Esoff first showed that although the papillæ are often elongated, this is a secondary change,—that they are really atrophied and not hypertrophied. The sweat-glands have disappeared or only exist as cysts, and the sebaceous glands are smaller and less numerous than usual. The hair-sacs are thickened by overgrowth of epidermis, and the hairs are atrophied, tufted at the root, and easily shed.

If the above account of ichthyosis be correct, there is no need for the distinction which Erasmus Wilson attempted to make between true and false ichthyosis. His "false ichthyosis," the *ichthyosis sebacea* of other authors, is the seborrhœa sicca above described (p. 949). In true ichthyosis there is no doubt a certain amount of sebum, which is mixed with the epidermic masses, and can be extracted by ether in the form of stearin and cholesterin; but this is not the essential part of the disease.

Nor is there any need to continue the distinctions of Devergie and other writers into *Ichthyosis alba, I. brunnea vel nigra*, and *I. hystrix*, or Alibert's of "Ichthyose nacrée and I. cornée."

In some cases of ichthyosis hystrix there are papillomata mingled with the epidermic lesions, as in a boy in Philip Ward in 1887, where the horny warts were arranged in long stripes down his arms and legs.

Xerodermia.—We must recognise as true ichthyosis, though of a much milder form, that affection of the skin which was named by Wilson "xeroderma."* This dryness of the skin is accompanied by roughness, to be felt rather than seen, which chiefly affects the outside of the arms and legs. There is but little desquamation, and the morbid change is so slight that it is difficult to believe it can be essentially the same as that which produced the porcupine-men. But of this there is no doubt, for we meet with every gradation between the two conditions. On the one hand, such an extreme degree of the affection as the *ichthyosis hystrix* of Tilesius is extremely rare, and on the other, even the slighter forms of xerodermia are more extensive, obstinate, and clinically important, than they at first sight appear. At the same time we may admit two groups of the affection, the more severe, which corresponds with the classical description of ichthyosis, and the milder forms, for which the term xerodermia might be used, if it had not since been unluckily conveyed to a totally different and malignant form of cutaneous disease (*v. infra*, p. 1018). Each case has its own characters from an early period, and when once established in the second or third year of life, does not usually become much worse. Both alike are congenital malformations, both have the same distribution and probably the same histology.

The chief importance of this remarkable disease, even in its mildest form and quite apart from the hideous deformity of the worst kinds, is that the dry, harsh, unlubricated skin is extremely disposed to superficial dermatitis; or, as it is usually put, ichthyosis and xerodermia are often complicated by eczema.

Treatment.—The first indication is to cure the inflamed, red or weeping patches, and the deep painful fissures by the same methods which have been above described for the treatment of eczema rubrum, madidans, and rimosum. The second indication is to supply the deficient natural lubricant of

* In this, as in other similar compounds, the name of the disease, the condition of the derma, should be spelt with *i*. So sclerodermia, pachydermia, &c., words analogous in formation to anæmia, and anuria.

the skin by oils or ointment; suppleness is thus restored, the characteristic dryness is removed, and the liability to dermatitis reduced to normal limits. In the more severe cases, however, it is necessary before this can be done to remove the products of disease; and for this purpose warm baths, alkaline baths, friction with soap and water, and above all with soft soap, are the measures which are necessary. The only caution is not to be too vigorous in softening and removing the diseased epidermis, until local inflammation has been relieved. From time to time the process of cure may have to be interrupted and the tender skin soothed by zinc or lead ointments or olive oil. Dr Fagge, as also Dr Liveing, recommend glycerine of starch, but often oil is more soothing than glycerine in any form.

It is astonishing what excellent results may be obtained, even in the worst cases of ichthyosis, when treated with perseverance and with an intelligent appreciation of the object in view. Within a few weeks children, whose portraits would almost go side by side with that of the porcupine men, present an appearance which it requires the scrutiny of an experienced eye to recognise as more than "a little roughness of the skin."

The disease, however, is relieved, not cured. As soon as the patient is neglected it returns as before, and he can only maintain his skin in bearable condition by constant attention to cleanliness, by frequent warm baths, and continual inunction. Dr Fagge recommended antimonial wine ('Guy's Hosp. Rep.,' 1870), and many physicians administer cod-liver oil.

The term *ichthyosis congenita* has been applied to a rare and remarkable form of disease described by Lebert, in 1864, as *keratosis diffusa intrauterina*. It affects the whole of the skin with thickening of the epidermis, which is too small for the body, so that the child is literally hide-bound. Numerous and deep fissures result, and the appearance which ensues has been described as the "harlequin fœtus."

The horny layer is greatly thickened, the papillæ and the rest of the cutis unaffected, the sebaceous glands are atrophied, and the ducts of the sweat-glands enormously stretched.

Cases of this curious and very rare affection have been described by several authors. The best account of it is that given by Hans von Hebra in his 'Krankhaften Veränderungen der Haut,' p. 348. Mr J. B. Sutton believes that it consists essentially in a perverted secretion of the hernia caseosa.

Cornu cutaneum (*ichthyosis cornea* of Willan and Bateman*) is the name applied to those remarkable cases of horny growths which have been figured as "freaks of nature." They are occasionally seen in old women, less often in old men, and very rarely indeed in early life.

Lebert collected 109 cases. Most often they spring from a sebaceous cyst. They may occur anywhere, often on the lip or the glans penis, and sometimes are followed by cancer.

The growth can always be readily removed, and shows no tendency to return; although, as Bateman remarks, if merely sawn or broken off, they invariably sprout again, like hair or nails.

* Bateman objected to calling them horns on the ground that they have no connection with the bones or other part beneath, and are of purely cuticular growth. But this is the only ground on which we call them true horns and not exostoses or antlers. What he meant was that they have no bony core as the horns of ruminants; but they are exactly identical in structure with that of the rhinosceros.

Two remarkable cases of cutaneous horns, one on the neck and the other on the hand, were modelled by the late Mr Towne for the Guy's Hospital Museum (Nos. 333 to 339).

SCLERODERMIA.*—This is a rare but interesting disease of the skin which, pathologically, is a chronic deep indurating dermatitis, followed by atrophy, and often ending in complete involution.

One of the best contributions of the lamented author of the present work to dermatology was his masterly account of this disease in the 'Guy's Hospital Reports' for 1867, in which he conclusively proved the essential identity of the diffused sclerodermia of authors with the circumscribed sclerodermia which was also known as *Addison's keloid*, and is synonymous with many cases described as *morphœa* by older writers. See also his second paper (ibid., vol. xv, p. 297).

Course.—The disease begins very gradually in a hardening of the deeper layers of the skin. The epidermis is unaffected, the surface smooth, and the colour unaltered; but the patient finds that the affected spot is stiff, and on feeling it a more or less marked induration is recognised, the skin cannot be pinched up into folds as in health, and instead of the natural elastic softness of the integument a characteristic hardness appears. In the circumscribed form the edges are well defined, so that it feels as if a disc of hard, smooth leather were let into the skin. In the diffused form the stiffness and induration become gradually less and less, until they are lost in the natural softness of the skin; but even then one may generally find some directions in which the sclerosed patch has a more definite edge. Sooner or later the local appearances become more marked, the affected skin becomes white, or assumes a sallow, yellow tint, or becomes pigmented with a pale yellowish brown, which is usually most marked towards the borders, and is never uniformly diffused over the entire patch. In the early stage a slight rosy circle may be observed around the patch, occasionally forming a distinct ring in the circumscribed form, or a more ill-defined and irregular blush in the diffused form (*scleriasis*). The smooth white patch, with its colour heightened by the pink margin, has been often compared to an ivory disc.

The patch of morphœa may go on increasing until a disc several inches in diameter is formed, or it may lose its distinctive characters and pass into the diffused variety.

Diffused scleriasis usually has its own characters from the beginning, and extends with no definite margin until it involves a considerable part of one limb, or one side of the neck or half the trunk. After a time contraction begins to appear, and scar-like bands vary the surface of the disease. This, together with increasing pigmentation, gives some resemblance to the contracted cicatrices from a scald or burn, and explains Addison's application of the term keloid to the affection.

Locality.—A patch of morphœa most often develops on the trunk, particularly on the skin of the female mamma, where such parchment-like *plaques*, or ivory indurations, like the skin frozen by an ether spray, have been sometimes called "vitiligo." Diffused sclerodermia may be seen on the scalp, the forehead, the chin, or other parts of the face; and the expressionless, mask-like aspect it gives to the features is very striking, particularly since the immo-

* *Synonyms.*—Scleriasis cutanea—Sclerème des adults (Thirial): including "Addison's keloid" and Morphœa. Vitiligo (in part).

bility is not uniform, but affects one side or certain features only. Scleriasis is also frequent in the arms, hands, and fingers, which become contracted and useless, and on the side of the neck, where a distortion may be produced, which resembles torticollis; or it may invade extensive regions of the trunk or lower extremities. In the well-marked case of a young and healthy soldier, reported by Dr. Curran ('Edin. Med. Journ.,' 1871), the disease covered the whole surface of the body. It is unsymmetrical. Occasionally, but only as an exception, it may be traced in the course of a cutaneous nerve.

Cases have been described in which it or a similar affection involves the mucous membrane of the mouth.

Symptoms.—Sometimes patients complain of a good deal of pain as well as stiffness in the affected parts, but this is often completely absent. There is no itching, and, as a rule, no accompanying inflammation. Sometimes, however, deep and very intractable circumscribed ulcers form on the sclerotic patches, as in a remarkable case brought by Mr Morant Baker before the Patholological Society (vol. xxxii, p. 261). There is no hyperæsthesia, nor true anæsthesia—at least, as a rule; but patients may complain that they do not feel as distintly as on the normal skin.

Histology.—There is scanty evidence of a true inflammatory process in this singular disease, nor does there seem to be anything which can fairly be called a new growth; the epidermis is unaffected, the papillæ atrophied only in the later stages of the affection, the hair-sacs, sebaceous and sweat glands normal, as also are the unstriped muscles of the cutis. The seat is primarily in the deeper layer of the cutis and the subcutaneous tissue. Here the fibrous bundles become thicker and the fat between the meshes is absorbed, while increased pigment is gradually deposited both in the papillæ and in the cutis. No cell proliferation is to be seen, according to the careful observations of Chiari, in the 'Vierteljahresschrift f. Derm. u. Syph.,' 1868. This process of mingled hypertrophy and atrophy leads to the characteristic results, both of the earlier and later stages of the disease. In the earlier stage, by compressing the blood-vessels, the peculiar pallor is produced; and by the increase of fibrous tissue and disappearance of fat the scleriasis of the later stages. Beside the pigmentation, the affected parts sink below the level of the healthy skin, instead of being, as at first, on the same level, and the contraction leads to the cicatrix-like bands which crumple the fingers or deform the face or breast.*

Prognosis.—Dr Fagge made the remarkable discovery that sclerodermia, both in its circumscribed and diffused forms, is liable to spontaneous involution. He tracked one of the most marked cases described by Addison, and found that the patient's skin had recovered its normal condition. The same thing has been repeatedly observed since, although it is too much to say that complete recovery is an invariable or even a frequent result. The disease, at all events, shows no tendency to develop into any active or malignant form, and beyond the disfigurement and disablement due to contractions and the occasional pain, the most serious result is the rare one of ulceration, as above noticed.

Sclerodermia is more common in women than in men—30 to 10 of

* See a valuable paper on "Sclerodermia" by Dr Rasmussen, of Copenhagen ('Edin. Med. Journ.,' Sept., 1867); and another by Dr Van Harlinger, of Philadelphia, with a full list of references ('American Journ. of Syphilis and Dermatology,' October, 1873). See also an account of the Histology of a Morphœa Patch, by Dr Crocker, 'Path. Trans.,' 1880.

Rasmussen's collected cases. It has been observed at all ages, including children under six and adults up to seventy.

Efficient *treatment* is completely unknown. Emollient oils, warm douches, and manipulation have been tried with some apparent benefit. Electricity has also been employed, either in the form of continuous galvanism to the affected patches, or by interrupted galvanism to the neck in the somewhat vague hope of stimulating the cervical sympathetic, and the equally vague expectation that occasional stimulation of the cervical sympathetic would have any effect upon the disease.

Sclerema neonatorum.—This affection, sometimes called Thirial's disease, is best named as he called it, *sclerème*, in distinction from the sclerodermia or scleriasis just described. It is the condition which is known as "hide-bound" in newborn children, affecting the whole of the surface, and characterised, not only by hardness, want of elasticity, and pallor, but also by œdema. The temperature is lowered and the child generally dies within a fortnight.

Linear atrophy.—Somewhat resembling sclerodermia in appearance, and perhaps also in pathology, is a curious affection of the skin, which takes the form of long streaks, generally broader in the middle than at the ends, or less frequently of round, more or less regular patches: in both cases it appears like a scar, for there is loss of pigment and atrophy of the cutis vera. It was first described by Dr Wilks ('Guy's Hosp. Reports,' 3rd series, vol. vii, p. 298) as an idiopathic affection which exactly resembles the cicatricial marks caused by overstretching of the skin and rupture of its deeper layers—well known under the name of lineæ gravidarum as a result of abdominal distension from pregnancy, but also seen in ascites or whenever the abdominal skin is similarly stretched, and over joints which have enlarged and stretched the skin. The spots are palpably depressed below the level of the healthy surface, and on a microscopical section, which was carried out by Kaposi at Vienna, the papillæ were found atrophied or vanished, the epidermis in both its layers thinned, and the subcutaneous tissue and glands atrophied. This curious affection, which may be idiopathic, has been seen upon the hips, the leg, the knee, the ankle, and the hand. In the early stage the marks are somewhat pink, but there are no signs of inflammation, no pain, or any other symptoms. In a case described by Dr Liveing the maculæ were at first slightly red and raised above the skin; they occupied the upper part of the sternum and neck, and after passing into the atrophic stage above described, ultimately underwent gradual involution.

In a girl who lay ill with renal dropsy in Miriam Ward in 1886, there was during the illness and after the recovery the most remarkable zebra-like development of atrophic stripes on the forearms, loins, and hips.

There are some good models of these striæ atrophicæ in the Guy's Hospital museum, Nos. 340—347.

ELEPHANTIASIS.*—This among many other names has been given to a curious form of hypertrophy of the skin chiefly met with in tropical climates. It is not necessary to enter upon the tangled labyrinth in which this, like so many other names of cutaneous diseases, is involved. It will suffice to say that the word elephantiasis was used by Aretæus and by Celsus for the very different disease known to the Greeks as lepra and to

* *Synonyms.*—Elephantiasis Arabum or Elephas—Bucnemia or Bouknemia—Arabic, dal fyl—Pachydermia—Barbadoes leg.

English readers as leprosy. They used it because of the magnitude and monstrosity of the disease. Unfortunately the comparison was supposed to be between the appearance of the disease and that of an elephant's hide, and since the legs affected with pachydermia have some resemblance to the thick and shapeless limbs of an elephant, the two diseases and their names were long hopelessly confused.

The most important pathological fact about elephantiasis, using the term as applied by the Arabian translators of the Greek authors, is that it is hypertrophy dependent upon recurrent deep dermatitis. All observers in countries where the disease is endemic agree that it begins and is accompanied by recurrent attacks of what has been called erysipelas, each attack leaving the tissues more thickened and infiltrated. Inflammatory œdema of the skin and subcutaneous tissue is the characteristic lesion. This gradually becomes œdema durum and no longer yields to pressure, the infiltrated tissues undergo hypertrophy, and masses of fibrous tissue are thus produced, which may be described as a diffused new growth. The skin itself appears at first to be unaffected, at least in its papillary and epidermic layers; but after a time it also hypertrophies, the papillæ becoming enlarged and the surface coarse, thick, scaly, and pigmented.

Histology.—On section, the hypertrophy of the deep layer of cutis, and the massive fibrous bands of white and elastic tissue, with œdematous connective and adipose tissue, are very characteristic; the lymph-spaces of the cutis are enlarged, and the lymphatic vessels are frequently found dilated and varicose. Occasionally an ulcer will accidentally open one of these enlarged lymphatics; and a discharge of normal lymph, more or less milky if it has passed through several lymph-glands, is poured out.

The disease does not spread to the deeper fasciæ or bones, and it never affects internal organs or leads to any but local results.

Such a condition is occasionally seen as the result of long-continued inflammatory dropsy of one limb. An example in a case of old dermatitis of originally syphilitic origin is figured in the 'Guy's Hospital Reports' for 1877, pl. ii. A similar result may also be seen in cases of enormous obesity and general hypertrophy of fat and subcutaneous tissues. Or, again, it may be the result of local pressure upon the veins and lymphatics, as by enlarged inguinal glands or other tumours. But in many hot countries, particularly the West India Islands, in Cape Colony, Egypt, South America, in China and Japan, and in the Pacific Islands—elephantiasis is idiopathic and endemic. Dr Turner, of Samoa, has made numerous photographs of this disease, which presents exactly the same features there as in the other races and climates where it is found.

The *distribution* of elephantiasis is almost limited to the legs and scrotum. Sometimes only one foot is affected, sometimes the thigh remains free. One leg may entirely escape while the other forms a huge tumour, and the scrotum may be diseased independently or along with the legs. The size of these scrotal tumours is sometimes enormous, the mass reaches to the ground, the penis is completely lost within it, and the whole weight exceeds that of the rest of the patient. As above stated, the organs involved in this monstrous tumour are, when dissected out, found perfectly normal, except that the tunica vaginalis is often the seat of hydrocele.

Ulceration of the unwieldy mass of flesh often occurs, and the pain and discharge of the ulcers may produce a certain amount of cachexia.

The *cause* of the disease was until lately perfectly unknown, but owing

to the remarkable discoveries made by Dr Lewis, Dr Manson, and other observers, it is now known that a certain proportion of cases of elephantiasis, particularly when it affects the scrotum (known as "lymph scrotum"), coincide with chyluria and the presence of a parasitic worm in the blood. (*Filaria hæmatobia*, supra, pp. 459, 700). It is supposed that the lymph-channels are mechanically blocked by the parasites; this leads to œdema and inflammation on the one hand, and, when rupture into the urinary tract occurs, to chyluria on the other.

There is no doubt, however, that many cases of elephantiasis have been observed in which no filariæ could be detected in the blood. See a case with discharge of milky lymph recorded by Mr Wagstaffe ('Path. Trans.,' 1875, p. 215), and in the same volume, one with great lymphatic dilatation figured by Mr Stewart, as well as a third case of ordinary pachydermia with histological details by Mr Butlin.

The *treatment* of this disease is purely surgical. There appears to be little or no power of restraining its course until the tumour is sufficiently large to be removed. From the famous cases of Clot Bey in Egypt to those of Dr Turner in Samoa and other medical missionaries, the removal of these frightful masses of flesh has been one of the most brilliant benefits conferred by European surgery.

Closely allied to elephantiasis are the curious cases described as *cutis pendula* or *dermatolysis* in which the skin hangs in great folds like garments. A classical case was recorded and figured by à Meek'ren in a young Spaniard who could bring the skin of his chest up to his eyes and down to his knees (1657).

XANTHELASMA*—This remarkable affection was originally described by Addison and Gull under the name of Vitiligoidea. See 'Guy's Hosp. Rep.' for 1851 (plates); ibid., 1866 (plates); ibid., 1877, with thirty-eight tabulated cases; and 'Path. Trans.,' 1866, p. 277 (plates); ibid., 1868, p. 436; ibid., 1882, p. 376, with thirty-six cases of multiple xanthelasma.

It shows itself in two distinct forms. There are in the first place flat cream-coloured patches (*Vitiligoidea plana* of Addison and Gull), which cannot be detected by the finger, although they look raised and have defined margins. The first indication of the development of xanthelasma is the appearance of such a yellow patch in one of the upper eyelids, just above the internal canthus. Afterwards other patches come out in the same neighbourhood, and these may ultimately coalesce, so as to form a broad ring surrounding the eyes. Similar flat patches may show themselves elsewhere; on the surface of the body, on the backs of the hands, on the scrotum, and also on the palms and soles, where they either present a peculiar dotted appearance, or form long streaks following the creases of the skin. This plane variety of xanthelasma may affect mucous membranes as well as the skin. It occurs in the gums and palate, and in the larynx and trachea; Dr Legg has seen it on the side of the tongue, and in two cases Dr Fagge found it in the lining of the bile-ducts.

The second and rarer form of xanthelasma consists of raised solid nodules,

* *Synonyms.*—Plaques jaunatres des paupières (Rayer)—Vitiligoidea plana et tuberosa (Addison and Gull)—Xanthelasma, *i. e.* Yellow laminæ from ξανθός and ἔλασμα (Wilson). —Molluscum lipomatodes (Wilson). The term Xanthoma (yellow tumour) was suggested by Dr Frank Smith in 1869, and has been generally adopted in Germany.

or tumours (*Vitiligoidea tuberosa*). These make their appearance later than the flat patches. They occur on the ears and on the limbs, especially on the extensor surfaces; they form aggregated tubera on the olecranon; and swellings on the knuckles not unlike those of gout. They are occasionally found, not in the skin itself, but in the adjacent tendons of the extensor muscles of the fingers. Two cases are reported in 'Virchow's Archiv' (1883, vol. xci, and 1885, vol. cii), in which the eyelids were unaffected, but nodules and tumours occupied the elbows, fingers, knees, and buttocks, and two others in the 'Path. Trans.,' 1884, by Mr Startin.

Xanthelasma is important, not only because of its pathological interest, but because it is often attended with much suffering to the patient. The parts affected with it are sometimes exceedingly tender. A patient of Dr Fagge's was unable to stand or even to sit with comfort, on account of the pain produced by the slightest pressure on the xanthelasmic patches, and for a similar reason she could not use her needle. In this case the affection became much less marked under internal treatment, most of the raised tubera disappeared, and the pains were in great part removed.

Similar involution has been observed in other cases, *e. g.* a remarkable one associated with icterus from atrophy of the liver recorded by the late Dr Frank Smith, of Sheffield ('Path. Trans.,' xxviii, 236).

Microscopical examination shows that xanthelasma is essentially a chronic deep dermatitis with early fatty degeneration, the yellow colour depending upon the presence of innumerable fatty granules in the tissue. In the nodules there is also present a dense fibrous tissue, and even in the plane variety a few ill-formed cells have been detected. The minute structure of xanthelasma is thus identical with that of atheroma in an artery.

The multiple and tuberous form of the disease is most frequently seen in chronic cases of jaundice from whatever cause it may arise, although it may also be found, particularly in children, in cases entirely free from icterus. Of eight cases of infantile xanthelasma multiplex not one was associated with jaundice; and in these the eyelids were not affected, as they almost always are in adults (see 'Path. Trans.,' vol. xxxiii, p. 383). In most cases it does not make its appearance until the patient has been jaundiced for a year or more, but in one case it began within six months after the jaundice, or perhaps even earlier.* The more common *plane* form of xanthelasma which is confined to the eyelids, has been shown by Mr Hutchinson to occur frequently in those who have suffered from sick headaches (see vol. i, p. 784).

Xanthelasma has been observed more frequently in women than in men, in the proportion of three to two. It occurs, like atheroma, most often in adults over forty years of age. Several examples are recorded in children, and two congenital cases by Dr Stephen Mackenzie and Dr Barlow ('Path. Trans.,' 1882 and 1884). Dr Church met with six cases of the affection in the same family ('St. Barth. Hosp. Rep.,' vol. x).

No plan of treatment is known, but it is only in exceptional cases that symptoms arise, and there is some reason to hope for spontaneous recovery.

* Some cases of xanthoma associated with diabetes (the second of Addison's and Gull's original list), and others by Dr Bristowe ('Path. Trans.,' 1868), M. Gendre ('Thèse de Paris,' 1880), and Mr. Malcolm Morris ('Path. Trans.,' 1883), are probably of a different nature, and rather resemble lichen.

LUPUS

Definition, history, and nomenclature—Anatomy, histology and course—Locality—Symptoms—Age, &c.—Diagnosis from cancer, rodent ulcer, and syphilis—Pathology—Relation to tubercle and scrofula—Prognosis—Treatment.
Lupus erythematosus—Pathology—Locality—Course—Histology—Treatment.
Disseminated erythematous lupus—Rhinoscleroma.

Of all diseases which affect the skin alone, lupus is the most destructive. Unlike syphilis, leprosy, and malignant growths, it is a purely cutaneous disease. It affects the deep layer of the cutis, and the epidermis is only involved subsequently. It also spreads to the subcutaneous tissue and occasionally to adjacent mucous membranes, but rarely to the cartilages or fascia propria beneath the skin; and it never attacks muscles, bones, or other deep structures. Lupus is at once a chronic deep dermatitis of a special kind and a new growth in the modern sense of the word. Virchow included it with tubercle, leprosy, and syphilis among the Granulomata, or new growths which consist of the same corpuscular elements which form the granulations of a healing ulcer. By the French writers it is almost universally assumed to be tubercular in nature, and forms the type of a supposed natural family of Scrofulides. In earlier times lupus was confounded with ordinary chronic ulcers, with cancer, with leprosy, and most of all with the later forms of syphilis. The lupus exedens of older writers was in most cases tertiary syphilis, *e. g.* the woodcut given as a type of the disease in Druitt's 'Vade Mecum.'*

Lupus was first carefully defined and described by Willan and was figured by Bateman in his sixty-seventh plate. He placed it among Tubercula and noticed its characteristic preference for the face. The distinctions subsequently introduced:—*Lupus exedens* and *L. non exedens* (Rayer), the lupus "qui détruit en surface—en profondeur—avec hypertrophie" (Cazenave and Schedel)—*Lupus serpiginosus*, *syphiliticus* and *vulgaris*—with many others—are unnecessary. Only one aberrant form, or rather allied disease, *Lupus erythematosus*, need be separately named.

Anatomy.—Lupus begins by the formation of minute nodules of granulation-tissue in the deeper layer of the cutis. These can be felt like shot in the skin, although without hardness, and show as reddish spots which mark the "macular" stage. When exposed, they are found to be vascular; and when several are seen together, a yellowish tint is sometimes observable, which has been compared to that of apple-jelly.

Histologically, they consist of small nucleated exudation-cells with very scanty stroma. As the disease goes on, these granulation-nodules unite, and undergo changes in two directions. The intercellular substance may become a stroma of delicate connective tissue. This may increase and

* *Syn.—Noli me tangere, Tentigo prava, Impetigo rodens,* and *Herpes exedens.* The word "lupus," which has been traced back by Virchow to the school of Salerno in the thirteenth century ('Archiv,' vol. xxxii, 1865), expresses the destructive ravages of the disease. It is applied to an incurable ulcer by Alexis of Piedmont (1578).

acquire firmness until it becomes connective tissue with spindle-shaped corpuscles; and finally may form a firm, fibrous, contracted, or œdematous tissue, which resembles an atrophic or hypertrophic cicatrix. More frequently, however, either universally or with only a certain amount of the fibrous transformation just described, the new-formed lupus tissue breaks down, the nodules become confluent, the cells undergo fatty degeneration, ulceration destroys the new growth, the epidermis gives way, and an ulcer results. The floor of this ulcer is formed by lupous nodules which can be distinguished by the naked eye from the healthy granulations of a healing sore. The edges are somewhat raised and can generally be felt to consist of nodules which have not yet softened. The pus secreted is usually thin and scanty. While fresh deposition of nodules and fresh softening and ulceration ensue, there is usually some effort at repair by the fibrous transformation above described.

The skin around, though red and slightly swollen, does not feel hot, and the redness is of a venous tint.

The whole process is strikingly similar to that which occurs in the lungs during the course of phthisis. There also we have minute nodules of granulation tissue, which have been described both as new growths and as inflammatory. There also the nodules undergo softening and ulceration; the ulceration spreads, with chronic inflammation and continual deposit of fresh "tubercles." There also the ulcerative process is rarely unaccompanied by some amount of fibrous transformation, which in favourable cases leads to the involution of the disease and the formation of a cicatrix. The bearing of this resemblance on the theory of lupus will be presently seen.

The process above described is extremely slow. We may watch lupus for more than a year before it ulcerates. It usually begins at a single spot and spreads irregularly therefrom, sometimes in a serpiginous form and comparatively swiftly, more often with an irregular rounded shape. It is rare for two independent foci of lupus to be seen, but this may sometimes occur. Whether in separate patches or as a single spreading surface it is decidedly unsymmetrical, unless it happens to begin in the median line.

The epidermis is usually more or less thickened, particularly the deepest layer, but the original seat of the process is in the papillary layer of the cutis, whence it spreads downwards as well as upwards, until the whole thickness of the true skin is infiltrated. According to Auspitz, the sebaceous glands are destroyed, the sweat-glands are unchanged, the hair-follicles disappear or are transformed into cysts. Rindfleisch asserts that lupus begins in the sebaceous glands, not the erythematous or so-called sebaceous form but lupus vulgaris; and therefore calls it an adenoma. But this no doubt is a mistake: any inflammatory disease will show most exudation in the more vascular parts of an organ, and the most vascular parts of the skin are the papillæ and the sebaceous glands; but the glands themselves are not involved except as a secondary result in the disease. Neither Neumann nor subsequent histologists agree with Rindfleisch.

Giant-cells are frequently observed (Friedländer in 'Virchow's Archiv,' 1874; and Thin, 'Med.-Chir. Trans.,' vol. lxii). Bacilli either identical with those of tubercle or closely resembling them were discovered by Doutrelepont in 1883. The characteristic bacilli, though no doubt always present, are very few in number, so that it takes much time and many sections to discover them.

Locality.—Lupus by preference attacks the face, particularly the alæ of

the nose, the edges of the lips, the cheeks, the eyelids, and the conjunctiva. It also occurs upon the ears and spreads to the neck. It is rarely seen on the scalp and is not common on the trunk and limbs; but there is probably no part of the body on which lupus has not been observed, and although, as just stated, it is rare to see two lupous ulcers at once, it often, after appearing and being cured upon the face, reappears in another region. In Vienna the trunk and the buttocks are said to be more often the seat of lupus than the arms and legs; the hands and feet are almost exempt.

Lupus also affects the mucous membrane of the nose, the lips, the hard and soft palate, and the larynx (cf. vol. i, p. 813). It very rarely affects the tongue or the deeper mucous membranes. According to Hebra, however, there is no doubt of the cartilages of nose and ears being occasionally affected, and even tendons and ligaments of joints. Lupus vaginæ has been described by Dr Matthews Duncan.

Symptoms.—As the progress of the disease is slow and its local signs torpid, so its symptoms are but slight. It is astonishing how little pain is felt even when extensive tracts of the skin are deeply ulcerated; as we shall presently see, the remedy is far more painful than the disease. The general health is also unaffected; so that, except for the disfigurement, lupus would be one of the most easily borne of all serious and destructive diseases.

Ætiology.—Apart from the question of its relation to tubercle, which will be presently discussed, we have no knowledge whatever of the cause of lupus.

It is probably equally common in both sexes. With respect to *age*, it is, as commonly seen, a disease of young adult life. But it has then lasted in most cases for several years, and it may occur in very young children. We have observed it in those not above four or five years old, but it is more common after puberty and usually begins at from fourteen or fifteen to twenty. After thirty it is certainly rare for it to begin, though cases of undoubted lupus may be occasionally observed to begin even later.

Moreover, in young subjects it shows an almost constant tendency to spread, even when repair, as generally happens, is to some extent occurring at the same time; but after thirty lupus tends to undergo involution, and if left to itself will in most cases end in cicatrices, disfiguring or disabling the patient, but no longer active.

According to Hebra and his disciples, it always begins before puberty, occasionally in infancy, but usually from the fifth to the ninth or tenth year. Lupus is not hereditary, and has never been seen at birth.

Devergie noticed that lupus is more often seen amongst hospital patients than in private practice. It is more common in Vienna than in London.

Diagnosis.—The fact of lupus being a deep inflammation of the skin and leaving scars, at once distinguishes it from eczema and all the superficial forms of dermatitis enumerated in the earlier chapters of this section; nor is there much practical difficulty in distinguishing it from varicose and other traumatic or accidental ulcers. The real difficulties of diagnosis arise between lupus, syphilis, rodent ulcer, and cancer of the skin.

Lupus is distinguished from *cancer* by the absence of pain, by its slow progress, by its beginning early in life, by the presence of granulations, the absence of hæmorrhage, and the nodulated, but not uniformly and densely infiltrated, edge. Invasion of deeper structures and secondary enlargement of the corresponding lymph-glands decides the case to be cancerous, but our object is to make diagnosis long before this point has been reached.

Rodent ulcer is covered by an adherent reddish-brown scab, which when

present is characteristic, but it may often have been removed by accident, by poulticing, or by other remedies before the lesion is seen. The edges are neither thick, hard, and infiltrated like those of epithelial cancer, nor do they contain little nodules as in lupus. Granulations are absent, the ulcer being of the kind known as "indolent," while that of lupus is what is called "weak." Like lupus, the face is its favourite seat, but the neighbourhood of the eyes rather than the cheeks and nose; it is always single and never extends so widely as lupus; it makes no attempt at spontaneous cicatrisation; and it only occurs in those who are past middle life.

Syphilitic ulcers have an undermined, not an infiltrated or nodular edge; the colour of the surrounding skin is brownish or yellowish, whereas that of lupus is of a more venous, *i.e.* purplish red. In both there may be considerable crusts, forming what is described as "rupia" and "ecthyma" in the one case and as "lupus pustulosus et crustaceus" in the other; but when these are removed, the more characteristic ulcerated surface beneath will be seen. The scars which result when a syphilitic ulcer is healed often resemble those of lupus, but they are less apt to be hypertrophied, they are much more pigmented, and seldom present the pink aspect and enlarged veins which are often seen after lupus is healed.

Moreover, tertiary syphilitic ulcers begin as a rule in the formation of a gumma in or under the skin, deeper than the nodules of lupus and less early affecting the epithelium. In the nose this distinction is most applicable,—lupus begins at the edge of the nostril and slowly creeps on, only affecting the cartilages (if it does at all) in its latest stages; whereas syphilis begins in the perichondrium or periosteum and has already destroyed much underlying tissue before the ulcer on the skin appears. The nose which has lost its tip or alæ has usually been affected by lupus; that which has lost its bridge by syphilis. Extensive disease of the skin, with the cartilages and septum intact, is most likely lupus; a small ulcer, with a deep foul cavity beneath it, and exposed bone and cartilage, is almost certainly syphilis. The frightful cases of destruction of the greater part of the face and opening of the orbit, pharynx, and posterior nares, which figured in museums and plates as lupus exedens, were tertiary syphilis, neglected or ill-treated, cases which the better diagnosis and improved therapeutics of modern times have happily banished from civilized countries.

Apart from the local characters, diagnosis between syphilis and lupus will be much helped by syphilitic ulcers being frequently multiple, lupus being very rarely so; by secondary implication of lymph-glands, with characteristic induration, being common in syphilis,—rare and only as the accidental consequence of temporary inflammation, which renders them soft and painful, in lupus. The syphilitic ulcer is usually accompanied by other cutaneous lesions; the lupous ulcer has no complication. Syphilis begins after puberty, often long after; lupus before puberty or shortly after. Lastly, lupus is a disease of the skin and nothing else, whereas syphilitic gummata and ulcers will be generally accompanied by other signs of lues in the bones, glands, tongue, or viscera.

Of the cutaneous lesions of congenital syphilis, the early coppery rashes have no resemblance to lupus, and the later gummatous ulcers are exactly like those of tertiary acquired syphilis.

Pathology.—Although it is scarcely possible to confound lupus with the ordinary lesions of congenital syphilis, it has been supposed by respectable authors that lupus is the result of inherited syphilis which does not

show itself in the ordinary form. Hebra himself was led to this opinion by a striking case in his own practice, where a syphilitic father had born to him —first, a stillborn child; secondly, one which died in a few months, with the ordinary marks of inherited syphilis; a third who survived, after suffering from congenital syphilis of the skin and bones; and the last who was born apparently healthy, remained so for some years, but became before puberty the subject of typical lupus. The case is no doubt striking, and is supposed to show that lupus is the feeblest and most diluted effect of transmitted syphilitic virus. But no one pretends that a child with a syphilitic father or brothers is thereby protected from becoming the subject of lupus, and if so the two diseases must occasionally occur in the same family or the same individual. Persons affected with lupus may acquire syphilis, and persons who inherit syphilis may be attacked by lupus. Owing to Mr Hutchinson's classical observations, we can now recognise congenital syphilis not only in infants but in later life. Such persons are not more liable to lupus than others; and one ought to be very sceptical in admitting that congenital syphilis is present when it shows itself by none of its unequivocal characters.

The assumption that there is such a thing as "syphilitic lupus," a kind of hybrid between two diatheses, is also unjustified; and, like similar diagnoses of "rheumatic gout" or hybrids of scarlatina and measles, is practically mischievous. At the same time the diagnosis between syphilis and lupus is often difficult, and even with care and experience one may mistake the one for the other.

Is lupus, as the French school assert, a "scrofulide"? This word was invented by Bazin and Hardy in imitation of the Syphilides of Biett and Alibert. Bazin includes among "Scrofulides bénignes," chilblains, erythema, strophulus, prurigo, lichen, eczema, impetigo, and some forms of acne. These are justly excluded by the sounder judgment of M. Hardy, who precisely defines his scrofulides as depending exclusively upon scrofula as the syphilides do upon syphilis, never developing without it, and diagnostic of its presence. He would place under this definition lupus, which Biett had, with his usual good sense, separated from all other diseases without inventing an ordinal name for it, while Alibert had lumped it with eczema and psoriasis among the dartres. Cazenave described four species of scrofulide under lupus: erythematous, tubercular, ulcerous, and hypertrophic. Bazin's division of "Scrofulides malignes" was into erythematous, tubercular, and *scrofulide crustacée ulcereuse;* and Hardy addresses the same reproach to the third of Bazin's as to the last two of Cazenave's species. He himself describes five varieties of scrofulides:

1. *Scr. erythemateuse.*—This corresponds to erythematous lupus, an undoubtedly distinct form, which will be described below.

2. *Scr. cornée et acnéique.*—This is not what other French writers describe as acné cornée, a curious affection of the sebaceous glands unaccompanied with inflammation ("ichthyosis follicularis") very rare, and in the cases which the writer has seen without the slightest claim to the epithet scrofulous (p. 948). Groups of comedones are described by Hardy as placed on a purplish-red patch, and as followed by depressed cicatrices unpreceded by ulceration. It appears upon the face, has a very slow course, and occurs (we may presume) only in persons who for some other reason are entitled to the epithet scrofulous. It corresponds to Devergie's *Herpès crétacé*, and to Chausit's *Acné atrophique.*

In London, and probably in Vienna, this affection would probably be called lupus erythematosus or lupus sebaceus indifferently or conjointly.

3. *Scr. pustuleuse.*—This is the most frequent variety; it begins either by a number of pin's-head pustules grouped on a small red patch, lasting from a week to a fortnight and leaving a yellow scab, or else with a large pustule, like that of ecthyma, which when ruptured gives place to a dark prominent crust that the older dermatologists would have named rupia. That part usually affected is the nose, the course is very slow and unaccompanied by itching or pain, but the most characteristic point is that when the crusts, which are very adherent, are removed, ulceration is found beneath. This ulceration is not deep, the surface is pale and sometimes presents little hard, dry, rough, warty nodules, which led Hardy originally to describe the variety as "scrofulide verruqueuse."

This form would by German, English, and American dermatologists be recognised as typical lupus (*Impetigo rodens*), which, as above described, frequently begins in pustules and is accompanied by large scabs. The slow course of the disease makes the subsequent ulcerated stage much more familiar, but even if watched from the beginning, cases of lupus with pustules and large prominent crusts would be seen in London, in a minority not less perhaps than a third or a fourth of the whole.

4. *Scr. tuberculeuse* is divided again into a superficial and a deep variety, and the former distinguished as sometimes disseminated over various parts of the body and sometimes localised, sometimes inconspicuous and ending in a light atrophic scar, sometimes hypertrophied, especially when it affects the genital organs. M. Hardy speaks of these deeper tuberculous scrofulides as producing "ces vastes destructions, ces plaies épouvantables et hideuses qu'on ne rencontre que trop souvent à la face," and as occasionally proving fatal, with profuse suppuration, cachexia, and hectic. This form, according to the eminent author quoted, produces enormous cicatrices on the eyelids, the lips, the neck, the ears, the nostrils, like those produced by severe burns.

This is obviously lupus exedens in its severest and most destructive form, but not differing from the slighter forms accompanied with true ulceration, except in degree. Moreover, even when untreated, the ravages of lupus, however hideous, are more remarkable for their contrast with the deeper destruction of syphilis and cancer than for their extent and severity considered as a disease of the skin.

5. *Scr. phlegmoneuse.*—This is a superficial ulcer which begins in a phlegmon as big as an almond or a nut; this gradually softens, fluctuates, acquires a purplish-red colour, and at last discharges a little thin pus; a scab forms, and this process may be repeated and become chronic until a large surface becomes ulcerated. The disease appears chiefly on the face, but also on the trunk and limbs. It always leaves a scar, at first violet-coloured, afterwards pale, irregular, and reticulated.

This somewhat rare variety will be recognised as what older surgeons, and especially the late Mr Hilton, used to describe as "scrofulous ulcer." The variety no doubt deserves mention, but whether regarded histologically, or from the point of view of pathology or of treatment, it also may be included under lupus. When, however, it occurs on other parts than the face, the primary abscess is often due to suppuration of a tubercular lymph-gland, of which there are not a few too small to be recognised by the anatomist, but apparent when enlarged by the hypertrophy of Hodgkin's disease or by caseous inflammation.

A review of these varieties of the scrofulides defined by the most experienced and the most rational of the successors of Biett, and described in his admirable 'Leçons' * with the clinical acumen and skill characteristic of Professor Hardy, shows that the only diseases of the skin which have any title to be called scrofulous are those which Willan and Bateman, with their successors in England, and Hebra, with his disciples in Germany, would agree in calling lupus. It is remarkable that the rare papular affection of the skin described by Hebra as lichen scrofulosorum (p. 900), and also the dry, harsh, un-oiled condition called xerodermia (p. 949), and pityriasis tabescentium, are not included in the above account of scrofulides.

What ground then is there for ascribing lupus to Scrofula ? This raises the question of the meaning which we attach to that much-abused word.

Scrofula originally denoted a swollen neck, which in some children makes the head pass into the shoulders with scarcely any constriction, as it does in a pig (*scrofa*). It is found that this usually depends upon a chronic caseous enlargement with characteristic suppuration and subsequent cicatrices of the cervical lymph-glands. The word *struma* also meant a swollen neck, and while in England used as a more or less vague synonym of scrofula, which had better be discarded, in Germany it is applied to another cause of a chronic swollen neck, namely, bronchocele or goitre.

The mere form of degeneration is, as Virchow long ago pointed out, not characteristic ; for it may occur in a traumatic abscess and in atheroma, in the middle of tumours and even of cancers. Moreover, most cases of caseous disease of the lympharia would, on careful examination, be found not to be idiopathic but secondary to mucous or cutaneous irritation. If indurated lympharia are discovered, we at once seek for a primary affection in a chancre, if cancerous in a primary tumour on an epithelial surface, if suppurating in a primary wound or inflammation of the skin or mucous membaane. In the same way caseous lymph-glands can generally be traced to chronic inflammation of the surface from which they receive their lymph. In the neck most frequently it is traceable to the throat with its tonsils and other lymphatic organs, more rarely to the scalp, the teeth, or the ear ; bronchial lymph-glands become caseous in consequence of chronic or repeated subacute bronchitis and broncho-pneumonia ; mesenteric lympharia in consequence of chronic or subacute enteritis and diarrhœa. These three groups of lympharia in the neck, the thorax, and the abdomen are the principal seats of so-called scrofula, and the reason is probably because the mucous membrane of the fauces, the bronchial tubes, and the small intestine is pre-eminently rich in adenoid or lymphatic tissue. According to the more rational believers in scrofula as a diathesis, disposition, or general pathological tendency, caseous disease of lymph-glands is clinically found connected with caries of the bones, with chronic inflammations of the articular ends or of the synovial membranes in joints, and with certain forms of catarrhal ophthalmia. On these points we do not presume to speak with authority ; but a physician certainly sees many children with caries of bones or chronic inflammation of several joints, who have no affection whatever of their lymphatic organs ; and there are many children, and some adults, with caseous inflammation of cervical and other lymph-glands, which lasts for years without their bones, their joints, or their conjunctiva ever being effected. Watson's classical account of the two types of scrofulous

* The later volume ('Maladies de la Peau') published in 1886, does not in this subject deviate from that which was taught in 1864.

children left one sceptical of the same morbid disposition showing itself in such opposite ways; and it is now clear that "pretty scrofula" was in most cases tuberculosis and "ugly scrofula" inherited syphilis.

This brings us to the question of the relation between scrofula and tubercle, and the relation of lupus to each. After phthisis and scrofulous pneumonia had been long assumed to be pathologically identical with scrofulous glands and joints and bones, Virchow introduced the critical light of histology into the confused mass of doctrine on this difficult subject. Regarding miliary tubercle as the type of that condition and as essentially a granuloma or new growth of an adenoid or lymphatic type, he defined the scrofulous diathesis merely as "vulnerability," that is, inability of the organism to recover from slight injuries. Subsequently, however, scrofula in the only definite anatomical sense of the term, has again approached tubercle; for, first, the giant-cells of Schüppel, which were imagined to be characteristic of tubercle, were found also in scrofulous lymph-glands by Friedländer; and now the still more famous *bacillus* of Koch has been discovered in these same organs.

The nodules and granules of lupus contain a minute bacillus which in form, size, and reaction to staining agents is indistinguishable from that found in phthisical sputum.

Nevertheless, no physician with clinical experience is prepared to admit that the uniform presence of a bacillus, any more than of a histological element or of a chemical product, can settle the true affinities of morbid processes, which must be judged of ultimately by their natural history and physiology, not by their anatomy, chemistry or mycology. In this as in other matters, to use Hebra's dictum, where the pathologist and the clinical physician differ, clinical knowledge must be the master: "Wo der Patholog und der Kliniker im Streite sind, muss der Kliniker Meister sein." Admitting, then, that the *bacillus lupi* is constant, and that the same organism occurs in tubercle, in scrofulous lymph-glands, and in lupus, the question still remains whether lupus occurs in persons who have definite signs of scrofula or who are subject to tubercular diseases.

To the latter question we must answer, No. It is extremely rare to see lupus among the countless victims of phthisis, *i. e.* of chronic tubercular inflammation of both lungs, beginning at the apex, travelling down, ulcerating, and destroying the tissue, and associated with laryngitis, enteritis, and tubercles in the viscera. Nor, looking at the question from the opposite point of view, has the writer found among patients with lupus, either in hospital practice in London or in the large numbers under Hebra's own treatment, any considerable number of cases of phthisis; and yet phthisis is remarkably common both in England and in Vienna, so that it has been regarded by English writers as the characteristic scourge of this country, and by Austrian writers as so peculiar to Vienna that its prevalence has been explained by the geological condition of the soil.

Nevertheless, we do sometimes meet with caseous glands or scrofulous scars in patients with lupus. Perhaps the occurrence is not more frequent than mere coincidence would explain, when we remember that both lupus and scrofula principally affect children and young adults. This very predilection, however, for a certain period of life may be fairly brought forward as an argument for a relation between the two diseases. A more powerful argument is the considerable resemblance in the mode of treatment which is found effectual for both.

On the whole, we conclude that lupus has a certain pathological relation to caseous or tubercular disease of the cervical lymph-glands, independent of its histology and of the presence of a bacillus; and that apart from this relation it has no connection with phthisis or with general tuberculosis. In fact, the cliuical relation between phthisis and general tuberculosis on the one hand and tubercular or caseous lymph-glands on the other, is a slight and uncertain one. It is also clear that with the unimportant exceptions of so-called lichen scrofulosus and pityriasis tabescentium, all the diseases of the skin which have any true connection with scrofula or tubercle may be comprised under the name of lupus. Lastly, notwithstanding these concessions, we must maintain that it is not justifiable to forsake the old, well-understood, short, and expressive term of lupus, one merit of which is that it expresses no theory and begs no question. In many cases of lupus, those who believe in tendencies and diatheses may call the patient scrofulous, just as in many cases of eczema they may call him gouty, and in many cases of erythema, rheumatic. Few would deny that lupus may be called scrofulous if the patient shows scars which prove that he has had caseous lymph-glands, that eczema may be called gouty when it occurs in a patient who has tophi in his ears or in his joints, and that erythema may be called rheumatic when it occurs in a patient who has suffered or is suffering from rheumatic fever. But in the great majority of cases of lupus, as seen by the writer in Vienna, since then in London, and also in Paris, there was nothing which an unbiassed observer would have called a sign of scrofula excepting the disease of the skin.

Auspitz—who has widely departed from Hebra's classification—puts lupus among what he styles "chorio-blastosen," or anomalies of growth of the corium and subcutaneous tissue. He subdivides this group into simple hypertrophic (macrosomia) and paratypical or abnormal growths, which includes the granulomata. Here lupus finds a place side by side with leprosy, scrofuloderma papulosum (or lichen scrofulosus), and scrofuloderma pustulosum (or acne cachecticorum), scrofuloderma ulcerosum (or scrofulous ulcers of the skin), tuberculosis cutis (as a separate condition), syphilis, and, lastly, rhinoscleroma.

This is practically following Virchow's arrangement of tubercle, lupus, and leprosy among the granulomata in his primary group of "new growths which are framed on the type of connective tissue."

In 'Ziemssen's Handbook' Neisser places lupus close to tuberculosis of the skin and scrofulodermia, and makes them one division of a group of chronic infectious diseases of the skin, which includes in addition leprosy, syphilis, glanders, rhinoscleroma and frambrœsia. He, however, excludes erythematous lupus from the group. Writing in 1883 he admits that no one has established the constant presence of Koch's bacillus tuberculosis in lupus, while Schüller has found only micrococci.

Kaposi and Baumgarten both oppose the recognition of lupus as a tubercular disease. Auspitz and Frederic Lander, Neumann, and other modern dermatologists in Germany admit it, and even Baumgarten allows a possibility of a genetic relation between the two. Plumbe spoke of lupus as a strumous affection. Erasmus Wilson maintained the same relation, Dr Fagge says "it is apt to occur in scrofulous persons," and Dr Liveing that "it belongs rather to the scrofulous diathesis." The late Dr Tilbury Fox "could not subscribe to the view that lupus is an evidence of the strumous

diathesis, and was more inclined to regard it as having a predilection for tubercular subjects." *

Clinical course and prognosis.—Lupus is one of the most chronic of diseases. It creeps on, usually with an imperfect attempt at healing, sometimes retreating until it almost disappears, and then again advancing with a persistence and rapidity foreign to its usual character. In the end, if left to itself, it probably heals, leaving, however, indelible marks of its presence in hideous scars, contracted limbs, distorted features, or obliterated orifices. It is singularly free both from pain and from irritation, and never affects internal organs. Whatever its true pathology may be, it does not produce secondary caseous inflammation of the lymph-glands which correspond to the affected skin, and never leads to general tuberculosis of the internal organs. Happily it is amenable to the efficient treatment which has been established within the last twenty or thirty years, so that the prognosis almost entirely depends upon the early recognition of the disease by a skilled practitioner.

Treatment.—Bateman remarks that he knows "no medicine which has been of any essential service in the cure of lupus," and that "it requires the constant assistance of the surgeon." Wilson, in the first edition of his treatise (1842), by a remarkable omission mentions neither the disease nor the name; in the later ones, he recommends caustic applications and a prolonged course of liquor arsen. et hydrarg. iodid., *i.e.* Donovan's solution. The usual practice of the earlier English dermatologists appears to have been to use arsenic and so-called tonics. It was Hebra who, regarding lupus, like most other diseases of the skin, as a purely local lesion, resolutely attacked the diseased tissue, and by destroying it produced a healthy inflammation which ended in cure. The determination, sometimes, with which he carried out this method, led to the most remarkable success. Tilbury Fox introduced the Vienna treatment into England, and maintained that the real treatment of lupus consists in destruction of the diseased tissue by caustics. Even Hardy, though he begins with general treatment of lupus as a scrofulide, admits that in certain cases local measures are also necessary, that emollient applications are unimportant, and stimulating lotions seldom useful. He recommends iodine—one part dissolved in thirty of water with the help of three of iodide of potassium. Even this he admits is useless in most cases, and recourse must then be had to stronger caustics, as *chloride of zinc*, *potassa fusa*, and particularly *binoxide of mercury*.

Often less severe measures suffice, and Hebra himself accomplished admirable results with the solid *lunar caustic*. A strong solution of the same silver-salt (a drachm to the ounce) may sometimes be substituted with good effect. The acid nitrate of mercury may also be applied, especially to small and comparatively superficial spots. But the most satisfactory method of treating most cases of ulcerative lupus is *scraping* by the sharp spoon introduced by Volkmann, of Halle.† Chloroform should be given and the whole of the diseased surface scraped away. It is astonishing how boldly a skilful surgeon can use this instrument or an analogous one, employing enough force to remove all the diseased tissue without injuring the more resistant healthy cutis which surrounds it. Indeed, Hebra's use of the pointed nitrate of silver pencil almost converted it into a scraping or

* On this subject the reader is referred to the summary for and against the tubercular nature of lupus in Dr Payne's recently published 'Manual of General Pathology' (p. 500).

† See his paper on "Lupus and its Treatment," translated for the Sydenham Society in 'German Clinical Lectures,' 1876.

mechanically destructive as well as a chemically destructive agent. The hæmorrhage produced by these operations is less than would be supposed. While it is almost always necessary to repeat the application of a caustic, one advantage of the scraping is that it is sometimes sufficient after a single sitting and seldom requires more than two or three. The saving of time as well as of pain to the patient is certainly remarkable.

Caustic potash, applied as it used to be in stick, is not only extremely painful, but even with the greatest care will destroy healthy as well as diseased tissue. Hebra's *arsenical paste* is less destructive, but causes great inflammation as well as pain, and is every way inferior to scraping.

The *pyrogallic acid* introduced by Järisch is probably the best local application. It should be used as an ointment of 10 per cent., which is better than solution or plasters. It causes, however, considerable pain.

Another plan of treatment, also introduced by Volkmann, and carried out by Vidal and Besnier in France, by Mr Squire and Dr Stowers in this country, is *scarification* or, as the operation is now performed, minute stabs with a lancet or an instrument made for the purpose. This section of immense numbers of blood-vessels produces temporary hæmorrhage, but afterwards obliteration of their channels and anæmia of the lupus spots.

The *galvanic cautery*, though sometimes applicable and less painful than would have been supposed, has the same drawback as caustic potash and sulphuric acid, that is, it destroys diseased and healthy tissues alike.

It is of paramount importance, for the successful treatment of lupus, to recognise its character as a new growth which must be destroyed. So long as any of the granulations remain, it is liable to return. Once rooted out, it is rare for this to happen or even for it to appear in another part of the skin. Occasionally the knife may be employed to excise part of the tip of an ear or some other circumscribed piece of skin, but scraping and caustic, or the two combined, are in a great majority of cases as effectual and the results are better. Indeed when early and thoroughly treated, lupus becomes a manageable disease, and the cicatrices which result are often surprisingly slight.

In the more superficial forms of lupus, and especially in the variety to be described as lupus erythematosus, such vigorous means are generally unnecessary, though wherever ulcers or granulations are seen, their destruction by some means or other is the only thorough method of cure. The milder applications which have been recommended, such as tincture of iodine, iodoform ointment (half a drachm to an ounce), pyrogallic acid ointment (a drachm to the ounce), and strong solution of nitrate of silver (a drachm to an ounce), may probably stop the disease at an early stage. They certainly check its progress, and may be usefully employed whenever more decisive treatment is counterindicated or postponed. For erythematous lupus especially, pyrogallic acid or iodoform are excellent applications.

Although local treatment is essential for lupus and is often sufficient without any other methods, many dermatologists strongly recommend the internal administration of cod-liver oil. Even Hebra admits its value and used to apply it locally to the sores as well as internally. It is unwise to trust to this remedy without attempting local measures as well; but wherever swollen glands or phthisical symptoms are present, or even when want of weight and flabby muscles show malnutrition, oleum morrhuæ ought undoubtedly to be given. Syrup of phosphate of iron, steel wine, or tincture of steel, are indicated by pallor. Arsenic is of doubtful service.

Some of the most scaly forms of lupus are said to be cured by arsenic, but if we cannot recognise such transition forms as are called psoriasis-lupus, we may suspect these cases of being really psoriasis and not lupus at all; just as serpiginous lupus is often extremely difficult to distinguish from syphilodermia, and owing to this difficulty has sometimes been supposed to be cured by iodide of potassium.

*Lupus erythematosus.**—The essential nature of this somewhat rare disease still admits of doubt. There is no question that the sebaceous glands are much affected by it; it is equally certain that a slow chronic dermatitis, accompanied with a violet or rose-tinted erythematous blush, is always present. But it is seldom that one fails to discover evidence of a destructive process of the papillary layer in more or less well-marked cicatrices, and in many instances of undoubted lupus erythematosus the scars are obvious. It is therefore rightly associated with the ordinary disease known as lupus, the two forms being distinguished when necessary as *lupus vulgaris*, *lupus exedens*, or *lupus exulcerans* on the one hand, and *lupus erythematosus*, *erythematodes*, *sebaceus* or *non-exedens* on the other.†

The *locality* of this affection is very characteristic. It almost always occupies the face and usually the bridge of the nose, together with both cheeks; for, in contradistinction to ordinary lupus, it is remarkably symmetrical. The figure produced by this distribution has been compared to a butterfly, a bat, or the sphenoid bone, and when once seen is easily recognised. Lupus erythematosus is also found on the ears, and sometimes on the scalp. The hair is then destroyed, a sufficient proof that lupus erythematosus is not, as it is classed in 'Ziemssen's Cyclopædia,' a superficial dermatitis. It occasionally appears upon the limbs or trunk, sometimes preserving its symmetry, but sometimes being confined to one arm, and most often to the hand. On the trunk and legs it is certainly rare, but in one patient of the writer's it spread over the shoulders and buttocks.

It is seldom that we see the first beginning of this disease. It shows itself as an erythematous patch, not unlike that left after impetigo or an early stage of tinea circinata. It spreads at the edge (whence Biett's epithet *centrifuge*), which is marked by injection, swelling, and desquamation, while the centre becomes pale, smooth, and slightly depressed. The sebaceous glands are enlarged, sometimes prominent, resembling acne punctata, sometimes forming black comedones within the affected surface. It thus spreads until it has attained the form and dimensions above described as characteristic. Sometimes, however, fresh spots occur at a distance, and this is decidedly more frequent than with ordinary lupus. The dry whitish scales, formed chiefly of sebum, suggested the epithet *herpes crétacé* to Devergie and *seborrhœa congestiva* to Hebra and others.

Its course is extremely slow, and like ordinary lupus it is accompanied by neither pain nor itching. It has no claim to be called scrofulous.

* *Synonyms.*—This curious affection was first described by Biett and named *Érythème centrifuge*. It was called by Hebra *Seborrhœa congestiva* (1845), and the same view of its nature has led to the titles *Lupus sebaceus* and *Lupus acnéique* (Hardy). It has also been named *Scrofulide erythémateuse* and *Lupus de Cazenave*. It is, however, more generally recognised by Cazenave's name, Lupus erythematosus (1850).

† In favour of this view see Mr Hutchinson's 23rd lecture ('On certain Rare Diseases of the Skin'). For arguments in favour of a more complete severance of lupus erythematosus from true lupus see Kaposi's and Veiel's papers ('Trans. Intern. Med. Congr.,' vol. iii, pp. 162, 167) with comments by Schwimmer and Thin; also Dr Payne's remarks ('St Thomas's Hosp. Rep.,' vol. xiii).

On making a microscopic section of the diseased skin, infiltration of the cutis with leucocytes and dilated blood-vessels is obvious; and the congestion and proliferation is most abundant around the sebaceous glands. These cells never become caseous or soften down so as to form the granulations and pus of an ulcer. They gradually become transformed into connective-tissue corpuscles; and as the fibres thus formed take their place, the papillæ atrophy and the glands shrink and disappear (Neumann, Geddings, Thin). These histological characters appear to show that no sharp line of distinction can be drawn between chronic deep-seated inflammation with hypertrophy and consecutive atrophy on the one hand, and development of such simpler forms of new growth as lupus, tubercle, and syphilis. On the other hand, there is a clearly marked line between deep inflammations with destruction and atrophy on the one hand, and superficial inflammations which do not destroy the papillæ and are never followed by ulceration or cicatrices on the other.

Lupus erythematosus occurs chiefly in adults, but sometimes in children. Veiel says that most cases occur between twenty and forty, and the average age of this affection is certainly later than that of ordinary lupus. It is perhaps commoner in women than in men.

Lupus erythematosus in some cases simulates nævus. See the account of patients under the care of Mr MacCarthy and Mr Higgins given by Mr Hutchinson in his 'Lectures on Clinical Surgery,' vol. i, p. 284.

The *treatment* of erythematous lupus is that of the milder forms of lupus vulgaris. Alteratives in the sense described above (p. 884) of a stimulant kind take the place of destructive applications. Hebra's diachylon ointment or solution of soft soap (spiritus saponis alkalinus) sometimes appears to be sufficient. Iodide of mercury ointment (one to fifteen) was recommended by Cazenave; the pyrogallic ointment above described is also useful, but the effects of iodoform are often the most satisfactory of any. In some cases very mild applications can alone be borne, such as unguentum metallorum, yellow oxide of mercury, or unguentum hydrargyri ammoniati. In others again, the true nature of the disease is shown by the treatment which dermatologists, whatever name they give it, are led to adopt—scarification, and even scraping or the galvanic cautery.

For the local treatment of erythematous lupus Mr Hutchinson strongly recommends the continued use of an ointment consisting of half a drachm of liquor carbonis detergens to an ounce of petroleum gelatum. The ung. liq. carbonis deterg. of the Guy's Pharmacopœia is stronger, and is also a very useful local remedy.

Lupus erythematosus disseminatus.—Kaposi has named lupus erythematosus as above described *discoid*, in order to distinguish it from a rare and remarkable form of disease, which he probably rightly regards as a form of lupus, and which he has named the "disseminated" or "aggregated" variety of lupus erythematosus. Here the patches do not grow by the enlargement of the circumference, but by fresh ones appearing. Moreover, the disease is not confined to the face, but is seen upon the trunk, the course is sometimes acute, and the whole character of the disease is far more severe than that of ordinary erythematous lupus, or even of lupus exedens; there is considerable pain, and sometimes synovitis; there is high temperature, nervous symptoms which sometimes end in coma, and in not a few cases the result has been fatal. Cæsar Boeck saw two well-marked cases of this curious disease in Norway.

The acute form is, however, the exception. More often the disease persists with more or less frequent exacerbations, the face appearing as if affected with constant erysipelas. Here also the end is usually death, either from marasmus, or from an intercurrent disease.

The writer has only seen one example of this remarkable affection, which occurred in the practice of Dr Cavafy at St George's Hospital. The patient was a woman between thirty and forty; the affection occupied not only the face, head, and neck, but the greater part of the back and trunk. It looked like erythema of a somewhat gyrate form, and there was unquestionable scarring. The patient succumbed to pneumonia.

Rhinoscleroma.—This uncouth epithet was applied in 1870 by Hebra and Kaposi to a newly-recognised form of disease—a hard, smooth infiltration or new growth of the septum of the nose and the adjacent tissues of the alæ nasi and of the upper lip. It has a general resemblance both to lupus and to syphilis, but differs from both in not being prone to ulceration, a characteristic which also at once distinguishes it from epithelial cancer. Mr Hutchinson has not seen any case which corresponds with the fourteen or fifteen seen in Vienna, but thinks he has observed cases of lupus which by their unusual hardness and other characters approached rhinoscleroma. A few additional cases have been published in Germany, reference to which will be found at p. 496 of Hans von Hebra's 'Krankhafte Veränd. d. Haut.' The writer saw a very remarkable case in a patient of Dr Payne's, a young man from South America. Dr S. Davies has recorded a well-marked instance from Egypt ('Brit. Med. Journ.,' May 29th, 1886).

The ivory-like induration, the singular locality, and the absence of ulceration seem to separate it from ordinary lupus. Frisch has discovered a bacterium ('Ziemssen's Hbdh.,' xiv, 713), and since then Cornil and Alvarez have also seen it ('Ann. de derm. et de Syph.,' vi, No. 4, abstracted in the 'Lond. Med. Rec.,' August, 1885, p. 345). See it figured in Dr Payne's 'Manual of General Pathology,' and his remarks (*ibid.*, p. 672).

The histological characters are not distinctive, for Kaposi found only infiltration of the cutis with very minute leucocytes. Geber recognised giant-cells and spindle-cells ('Arch. f. Derm. u. Syph.,' 1872). In a doubtful case, brought by Mr Morrant Baker before the Pathological Society in 1881, Mr Hutchinson, Dr Cavafy and the writer were appointed a committee, and drew up a report, which will be found at p. 262 of the 'Transactions' for that year. A figure is given at p. 458 of 'Ziemssen's Handbuch' by Schwimmer and Babes.

On the whole, rhinoscleroma appears to be more nearly related to lupus than to any other disease.

It has returned after removal in cases reported from Germany and from Italy; but is said to have been favourably influenced by salicylic acid, applied in the belief that it would act as a germicide.

LEPROSY*

History and terminology—Geographical distribution—Anatomical lesions and course—Histology—The bacillus leprœ—Symptoms and event—Ætiology—Treatment—Other exotic diseases.

THIS disease, interesting from an historical point of view, is still of practical importance in many parts of the world; but we have only space here for a very brief account of it, referring the reader for further information to the elaborate article by Kaposi, in Hebra's great work, and to Dr Liveing's Gulstonian Lectures for 1873.

Nomenclature.—The names given to the disease by the Greeks were *lepra* and *elephantiasis;* it was divided into *alphos*, *melas*, and *leukos*.

The Egyptian leprosy of Lucretius was quite as likely to be what we now know as elephantiasis (sc. Arabum):

> "Est elephas morbus qui propter flumina Nili
> Gignitur Egypto in media, neque præterea usquam."
> DE RERUM NAT., lib. vi, 1112.

Celsus, however, who describes *alphos*, *melas*, and *leuce* as species of *Vitiligo* (lib. v, cap. xxviii, § 19), clearly and distinctly portrays leprosy as a disease affecting the bones and the whole body, almost unknown in Italy, "*quem ἐλεφαντίασιν Græci vocant*" (lib. iii, cap. 25).†

The term applied by Willan and Bateman to leprosy was "elephantiasis Græcorum," while they unfortunately used "lepra" for part of the white scaly disease which the ancients would probably have recognised as *alphos*, and which all modern dermatologists call psoriasis.

No doubt many other cutaneous affections, obstinate chronic eczema, syphilis, lupus, and perhaps psoriasis, were confounded with leprosy in ancient times; but there is no question that one and the same destructive form of disease has existed in Palestine under the Mosaic Law, in Western Europe during the Middle Ages, and at the present day in many parts of the globe; and this is best named by its historical title, leprosy.

Distribution.—Norway is the only European country in which leprosy is still common; it is there known as *Spedalskhed*. It is also found here and there in Sicily and Malta, in certain parts of Portugal, in the Levant, in the Crimea, and at Astrakan; it is more common in Persia, Bengal, S. India,

* *Synonyms.*—Lepra vera—Lepra Arabum—Elephantiasis Græcorum—Leontiasis—Satyriasis—Morbus Herculeus.—*Fr.* La lèpre.—*Germ.* Aussatz.

Leprosy appears to have been rare in ancient Greece, and it seems to be not quite certain that the Septuagint translators were correct in rendering *zaraath* of the Hebrew Scriptures by the Greek word λέπρα. The latter term, however, is universally applied to leprosy in the New Testament. It refers to the scaly surface often seen. The Arabic name of true leprosy, according to Dr Greenhill, is *Judzam* (= *lepra Arabum*). *Barat* (= *leuce* = *vitiligo*), or "white leprosy" is nothing but leucodermia. In the Middle Ages leprosy was known to the school of Salerno as *mal morto* and *mal di San Lazaro*.

† It must be remembered that the terms Elephas and Elephantiasis do not refer to rough skin or huge and shapeless limbs, but to the magnitude of the disease. "Elephantiasis a magnitudine et diuturnitate nomen accepit" (Aetius). "Est lepræ species elephantiasisque vocatur, Quæ cunctis morbis major sic esse videtur, Ut major cunctis elephas animantibus extat" (Macer). So also Aretæus.

Burma, and Siam; in Japan and in China, where it is said to have been known for ages; in Egypt, Nubia, the Soudan, the Cape Colony (where it coexists with elephantiasis Arabum), and most parts of the African coast (though apparently it is rare in the interior); in Madagascar and the Mauritius, St Helena, the Canary Islands, and the Azores; in New Brunswick, Mexico and the West Indies (especially Trinidad); Central America, Ecuador, British Guiana and Surinam, Bahia and the coast of Brazil; in New Zealand, the Sandwich Islands, and some other parts of the Pacific.

Accounts of the disease from many of these places will be found in a report on leprosy by the College of Physicians prepared for the Colonial Office, and issued as a Blue-book in 1867. In 1874 Dr Vandyke Carter published an official report upon leprosy in India.

Varieties.—Leprosy is essentially one and the same disease, but one of two forms is usually predominant—the *nodular* or "tubercular," and the *anæsthetic*. The two, however, are often combined. Either may be preceded or accompanied by pigment-spots, which have led to a third species being formed—*lepra maculosa*. All end in an ulcerative stage, and all may lead to loss of members—*lepra mutilans*. "Black leprosy" is the only genuine form; "white leprosy" is not leprosy at all, but leucodermia.

Origin and anatomy.—The disease begins insidiously, but in some cases with an outbreak of bullæ resembling those of pemphigus. There follows the appearance of red or violet patches, varying from a finger-nail to the palm of the hand in size, which gradually become darker in colour. At the same places, or independently, appear flat, firm, raised nodules, consisting of an infiltration of the deeper parts of the skin. The lymph-glands at the same time enlarge. These nodules of tubercular leprosy may shrink and be absorbed, leaving atrophied and sometimes pigmented spots; but more often they soften and ulcerate. The leprous ulcers secrete but little pus, and show few and feeble granulations. They slowly increase both in extent and depth.

The leprous spots usually appear first upon the limbs, and afterwards on the trunk and face. When fully developed in the face, the disease produces a singular deformity, which the ancients described as *leontiasis* and *satyriasis*, and which, once seen, even in a drawing, is never forgotten. The disease also affects the neck, shoulders, back, chest, and abdomen, but is most frequent in the extremities, especially on the extensor surface. Nodules occasionally occur, even upon the palm and sole. The hands and feet are swollen and distorted, with thickened and rough skin; the ulcers burrow deeply and affect tendons, bones, and fibrous tissues, until at last toes, fingers, or the entire hand or foot undergo gradual necrosis and fall off.

The mucous membranes are also affected, particularly those of the mouth, nostrils, and larynx, also the conjunctivæ.

Moreover, the disease involves the great nerve-trunks, where the leprous nodules can often be felt during life.

Histology.—Careful microscopical investigations by Virchow, Thoma, and others showed that the disease consists in infiltration of the deepest layers of the cutis with granulation tissue. Leprosy was therefore classed by Virchow in proximity to lupus, from which, however, its clinical course, geographical distribution, and entire natural history widely separate it.

A bacillus was discovered by Hansen, of Bergen, in 1874, which he described and figured in the 'Quart. Journ. of Micro. Sci.' for 1880 (vol. xx, p. 92).* These microphyta appear constantly in leprous nodules, and in

* They have since been found by Neisser, Cornil and Babes, Köbner, Dr Hillis ('Path.

extraordinary numbers. The *bacillus leprœ* is 5 μ long, very slender, and immobile. It stains like the bacillus of lupus.

Attempts at inoculation have hitherto failed: see Dr Beaven Rake's report of his experiments at Trinidad ('Brit. Med. Journ.,' Feb. 5th, 1887).

Course.—The progress of leprosy is extremely slow, and it resembles syphilis and lupus in producing but little pain. Patches of anæsthesia are sometimes found and may be followed by ulceration before tubercles appear. It is said that in rare instances hyperæsthesia precedes or takes the place of loss of sensibility. The anæsthetic spots usually show some amount of atrophy, and the hairs are small and deficient in colour.

While this terrible disease goes on its course, interrupted from time to time by temporary improvement and healing of the ulcers, but never more than checked, the general condition of the patient is wonderfully little affected. Even perspiration takes place very much as usual. The hair, however, is gradually lost, not only that of the scalp, but also the beard, eyebrows and eyelashes. There is no fever, the temperature is usually subnormal, and the patient suffers much from cold. The pulse is slow, the appetite and other organic functions, including the quality of the urine, are very little altered. There appears to be no foundation whatever for the assertion of the ancient physicians that the sexual instinct is increased: perhaps the name *satyriasis*, first applied to the distorted and hideous features of the sufferer, was afterwards misinterpreted.

Death seldom occurs directly from leprosy, for there is neither excessive pain nor hæmorrhage nor invasion of vital organs to cause it; but when once fallen into a condition of anæmia and marasmus, the miserable leper is cut off by some intercurrent affection—pleurisy, pneumonia, dysentery, or Bright's disease, all of which have been recorded by the Norwegian pathologists, Boeck and Danielssen, but none with sufficient frequency to show more than an accidental connection with leprosy.

Ætiology.—The essential cause of leprosy is entirely unknown. It has probably existed from the earliest times, and has only disappeared from civilized Europe within the last 400 years. We may hope that it is in slow but steady process of extinction in other regions. Notwithstanding the presence of the *bacillus leprœ*, the disease is, under its usual conditions, non-contagious; it is not transmissible by living in the same house, by contact, or even by sexual intercourse. It is, however, possible that contact of actually ulcerating leprous nodules with a fissured skin or mucous membrane might produce the disease, and there is reason to believe that a contagious quality is more marked when the disease is newly introduced, as into the Sandwich Islands, and also, according to Dr Liveing, into Australia by the Chinese immigrants. Dr Gairdner has lately published a case which seems to show that leprosy may be inoculated by vaccination ('Brit. Med. Journ.,' June 11th, 1887).

Whether or not it is under any circumstances contagious, leprosy is undoubtedly *hereditary*, and its occurrence in persons of pure European parentage is excessively rare. Patients in England are usually either half-castes or persons who were born and lived in India, and one of whose parents was perhaps of mixed blood.

It is doubtful whether leprosy has any predilection for castes or races

Trans.,' 1883, pl. xxii), Dr Thin ('Med.-Chir. Trans.,' vol. lxvi, pls. xii, xiii), Dr L. J. Steven, of Glasgow ('Brit. Med. Journ.,' July 18th, 1885), and by Dr Rake ('Path. Trans.,' 1887). Köbner tried inoculation unsuccessfully.

as such, although at the present day it is, as above stated, almost confined to certain of the dark races of mankind, and where prevalent is rare among the well-fed and well-cared-for classes.

Mr Hutchinson has suggested that leprosy depends upon eating fish, probably fish in a state of decomposition. This view certainly agrees with its presence, not only on the sea-coast, but also in the neighbourhood of great rivers and inland lakes ; and it also accords with the large consumption of salt fish in the Middle Ages, when it formed the principal animal food throughout the winter. There is no proof, however, of the connection of leprosy with eating fish. The disease does not appear in many parts where fish, both fresh and putrid, is eaten, and it is prevalent in certain districts where fish do not form an article of diet.

Leprosy appears to be somewhat more common in men than in women—in Bombay, according to Dr Carter, very much so. It usually begins about the time of puberty or in young adults. No congenital case appears to be on record.

Treatment.—This is unfortunately almost hopeless, and we must rather look to the gradual rooting out of the disease by improved conditions of life than to therapeutics. Various drugs have been vaunted from time to time as specifics, but have all in turn been discredited. Cod-liver oil is the only internal remedy which can be said to do more than alleviate symptoms. Externally Gurjun and Chaulmoogra oils have been supposed to be valuable. The writer has tried the former in three cases with no benefit. Dr Liveing's much larger experience makes it probable that the latter is sometimes of service.

Mr Hutchinson has recorded a case of gradual spontaneous recovery ('Med.-Chir. Trans.,' lxii, p. 331).

Leprosy is the only exotic disease of the skin of practical importance to practitioners in England.

Frambœsia or Yaws, apparently a contagious malady, and by some authors believed to be nothing but Syphilodermia, was known to Bateman, and is described at length by Kaposi in Hebra's 'Handbook.' It is endemic on the west coast of Africa, but appears to be identical with what is known as *Pian* in Java and as *Verrugas* in Peru. Less clear is its relation to *Parangi*, a cutaneous disease, endemic in Ceylon. *Radesyge* in Norway is, according to Hebra, lupus. *Sibbens*, the old Scottish name of a disease of the skin, was syphilis.

Aleppo evil, the Oriental sore, known also as *bouton d'Alep* or *de Biskra*, and the *Delhi boil*, has been ascribed to syphilis, but without proof.

Pellagra, an epidemic erythema, was first observed in Lombardy, and connected with eating diseased maize ; it is probably identical with *Acrodynia*, described by Alibert as epidemic in Paris during 1828 and 1829. Winternitz ("Eine klinische Studie ü. das Pellagra," 'Vierteljahresschrift f. Derm. u. Syph.,' 1876) doubts the very existence of the former. Acrodynia appears to be endemic in the Levant (See Behrend, 'Hautkrankheiten,' pp. 154 and 156).

Ringworm appears in peculiar forms in certain foreign countries. Burmese ringworm has been already referred to (p. 970) ; and Dr Anderson has published an interesting account, with figures, of *Tinea imbricata* from Tokelau, in the South Seas ('Edin. Med. Journ.,' Sept., 1880).

TUMOURS OF THE SKIN

MULTIPLE FIBROMA.—*Distinction from molluscum sebaceum—Anatomy and distribution of the tumours—Their course and treatment—Neuroma—Myoma.*
CHELOID.—*Terminology and history—Appearance, course, and symptoms—Histology—Relation to scars—Distribution—Prognosis and treatment.*
Angioma or vascular nævus—Elephantiasis teleangiectodes—Lymphangioma.
Mycosis fungoides—Kaposi's xerodermia maligna—Carcinoma, sarcoma, and rodent ulcer of the skin.

PASSING from the deep intractable ulcerations, combined with hypertrophic or neoplastic processes, of lupus and leprosy, we come to the new growths or tumours of the skin in a more restricted sense.

The relation between deep and chronic inflammation of the skin, hypertrophy, and new growth is so close, that lupus, tertiary syphilis, and leprosy might be classed either as deep destructive forms of dermatitis or as cutaneous granulomata; while warts and condylomata, gutta rosea, xanthelasma, and elephantiasis are as much new growths as inflammations. But we have now to treat of neoplasms which are neither hypertrophies nor inflammations.

As in other parts of the body, the tumours of the skin are clinically "innocent," "malignant," or "semi-malignant;" while anatomically they are "homologous" or "heterologous" (cf. vol. i, p. 97).

FIBROMA.*—This affection, named *molluscum* by Willan, differs altogether from molluscum contagiosum treated of above (p. 950), except in the fact that they both consist of multiple pedunculated tumours. Those of fibroma are not cystic growths, they are not glandular, and they have none of the histological characters of contagious molluscum. They are soft and painless, the skin over them is unaffected, they are more or less pedunculated, they vary in size from a pea to a marble or a fist, and when cut into they show œdematous, inelastic connective-tissue. They are not unlike, both in appearance and structure, the firmer kinds of polypi of the nasal fossæ, the colon, rectum, uterus, and other parts of the mucous membrane. They might in fact be well termed "multiple cutaneous fibroma" or "multiple fibrous polypi of the skin."

The number of these tumours is sometimes almost innumerable, as is seen in the well-known case of Virchow which forms the frontispiece to his work on morbid growths ('Kr. Geschw.,' Bd. i, S. 325). The size varies from a pin's head to a foot or more in diameter.

The celebrated case of Tilesius, of Leipzig, published in 1793, was named molluscum by Willan from the soft fleshy character of the tumours (*corpus tectum est verrucis mollibus sive molluscis*). Bateman recognised that these were not glandular and were quite distinct from the molluscum contagiosum

* *Synonyms.*—Molluscum fibrosum, areolo-fibrosum, non-contagiosum, simplex, pendulum—Fibroma molluscum (Virchow).

described by himself. The skin of Rheinhard, the Mühlberg peasant, who came under the notice of Tilesius, is still preserved in the museum of Leipzig.

Dr. Fagge believed that these tumours begin in the outer sheath of the hair-follicles and sebaceous glands; and in one case he found an enlarged sacculated gland occupying the interior of one of the growths. This has, however, not been again observed, and the occurrence of similar tumours in the palm and sole seems to prove that the coincidence was accidental.

There appears to be little local predilection for these fibrous polypi. We sometimes see a single one on the face or elsewhere, or they may cover the face, the trunk, and the limbs. They also sometimes appear on the prolabium and the palate, as in a remarkable case described by Dr Fagge in the 'Medico-Chirurgical Transactions' for 1870, vol. liii, which was figured in Plate 18 of the Sydenham Society's 'Atlas,' and modelled for the Guy's Hospital museum (No. 497).

Cutaneous fibromata occasion no pain, and single ones may be met with in perfectly healthy persons, to whom they cause no inconvenience. Some of these are congenital, but the typical multiple fibromata are certainly not so. They usually appear in childhood. Multiple fibromata are certainly rare, and probably few cases have failed of being recorded; but one or two polypi are not infrequently seen if looked for.

When they have attained their full growth they undergo no further change, and neither degenerate nor become absorbed. But, as Mr Hutchinson has pointed out, they sometimes lose their firm, fleshy feel, and become flaccid so as to feel almost like empty cysts. (See the 16th of his Clinical Lectures "On Rare Diseases of the Skin.")

Virchow has recorded a case in which the father, grandfather, and brother of a patient were all affected with multiple fibromata of the skin.

According to Hebra, when they are numerous the patient is usually ill developed in mind and body. But this is certainly not always the case.

The only treatment is removal by scissors or the knife. The polypi show no tendency to return.

Neuromata are multiple, painful fibrous tumours of the nerve-trunks, scattered over both trunk and limbs, have been long known and have lately formed the subject of a monograph by von Recklinghausen ('Ueber die multiplen Fibrome der Haut und ihre Beziehung zu den multiplen Neuromen,' 1882). Except in the pain which accompanies them, these tumours are indistinguishable from ordinary fibromata. Dr Duhring has described some severe cases marked by paroxysms of neuralgia.

Lipomata, or true fatty tumours, never affect the skin itself, but are always subcutaneous.

Myoma—Tumours of unstriped muscular fibre (*liomyomata*) have been described by Virchow ('Archiv,' vols. iii and vi), Klebs, Axel-Key; Rindfleisch, and Besnier ('Annales de Dermatologie,' 1880). They probably take their origin in the muscular bands connected with the hair-sacs. They are of no clinical significance.

Cheloid.—In the 'Arbre des Dermatoses' of Alibert appears, among many other fantastic names, a new term for what was an undescribed disease —*Kéloide*. The etymology of the word was long a puzzle. It was supposed by some to be derived from κήλις, a mark, by others from κήλη, a tumour.

Being taken by Addison in the former sense—*quasi ustione facta macula*—as meaning a scar from a burn, it was transferred to the curious affection still known as "Addison's keloid," but better named morphœa or circumscribed sclerodermia, described above (p. 989). It is now certain from the researches of Dr Fagge that Alibert meant by the word "kéloide" to denote the claw-like offshoots which characterise the disease in question, and intended to derive it from χηλή, a crab's claw. The right spelling is now generally used, and Alibert's is recognised as the only "true" cheloid.

Alibert described it as "cancroide," and Bazin and other French dermatologists have hence called it malignant and regarded it as closely allied to epithelioma of the skin; but it is not improbable that by "cancroide" Alibert did not mean "cancer-like" but "crab-like;" at all events it is not a cancroid tumour in the modern sense of the word.

Biett and Lebert afterwards published cases; Addison gave an excellent account of the disease in the 'Med.-Chir. Trans.' for 1854 (reprinted in his 'Collected Works'), and a paper by Dieburgs appeared in the 'Deutsche Klinik' for 1852, No. 33.

The affection is a rare one. It begins as a pink, smooth, slightly raised, flat nodule which increases in extent without becoming relatively more prominent. It is remarkably firm in feel. The centre becomes paler and is sometimes depressed, and the raised edges are surrounded by a slight erythematous border; the epidermis is completely adherent; it is in and not under the skin. Sometimes, however, especially in the later stages, it spreads to the subcutaneous tissue and forms adhesions to the deeper parts, but it never invades more than the integument.

The most characteristic part of the disease is the presence of radiating bands, which appear after a time, run across the original nodule, and afterwards project from its edge. These undergo contraction in the same way as the cicatrices of a wound, and the whole tumour is sometimes puckered and deformed by this process. In the earlier period the nodule might pass for a hypertrophied scar; in the later stages it still more closely resembles a large indurated and contracted cicatrix, as from a deep burn or a syphilitic ulcer or a carbuncle.

The tumour is usually single but two or more may exist on the same patient, as in a young man at present in Philip Ward. The disease is of very slow growth. It occurs most often in young adults of either sex. From the very commencement it is usually attended with pricking and itching with a sense of constriction, and sometimes there are severe stabbing pains. It is almost always tender to the touch.

Histology.—Microscopic sections show that the epidermis is thin but otherwise unaffected, the papillæ are destroyed and the cutis vera and subcutaneous tissue occupied by bands of dense fibrous tissue which are quite indistinguishable from those of a true scar. As in all cicatrices, the sweat-glands, hair-sacs, and sebaceous sacs are destroyed in the process. Dr Warren, of Boston, published a valuable histological account of cheloid in the Transactions of the 'k. k. Acad. d. Wissensch.' Vienna, March, 1868. See also that by Babes in 'Ziemssen's Handbuch,' xiv, p. 434.

Diagnosis.—Neither in the histology nor in the symptoms does there seem to be any obvious distinction between a cheloid tumour and a hypertrophied and painful scar. Hebra, in fact, defines cheloid as an idiopathic or primary cicatrix. Others have maintained that all cheloid tumours are hypertrophied scars, and undoubtedly they often arise from ordinary cica-

trices or from the slight marks left after leech bites or acne pustules—"acne-cheloid," quite distinct from the papillary affection sometimes so called (cf. supra, p. 956). It commonly occurs upon the shoulders, where acne-cicatrices are usually the deepest and most extensive. Dr Goodhart has published a remarkable case of cheloid growths following smallpox in the 'Clinical Transactions,' vol. xiii, p. 51.

Dr Liveing, while admitting that cheloid growths often begin in scars, finds the distinction between them and hypertrophied cicatrices in two points; first, that the bands of fibrous tissue in cheloid run in definite parallel or radiating bundles, whereas those of a cicatrix form an irregular network; secondly, that the cheloid growth invades healthy tissues, which hypertrophied scars never do, and that this is the case even when cheloid appears in a previous scar. The new growth can be distinguished as it invades the old cicatricial tissue.

Locality.—Cheloid tumours occur most frequently in the skin over the sternum. They have also been observed on the abdomen, shoulders, arms, and face. They are usually single, and very rarely more than two in number, except in the case of cicatricial, so-called false, cheloid, when the new growth may appear in as many scars as were originally present.

We had once in hospital a well-marked case of cheloid affecting the pubes in a patient of Mr Bryant, a man who probably had never had ulceration, syphilitic or other, of this part.

Nature.—Pathologically cheloid is not a mere hypertrophy nor a granuloma, but a fibro-cellular new growth, a true sarcoma, sometimes consisting chiefly of spindle-cells, sometimes more exclusively of fibres. It has two characteristic marks of sarcoma apart from its histology, namely, that it is very apt to return again and again after removal, while on the other hand it does not reappear in the neighbouring lymph-glands or in the viscera.

A number of excellent models of cheloid, Nos. 454 to 466, were made by Mr Towne for the Guy's Hospital museum.

Traumatic, or false cheloid, is better called a hypertrophied scar ("die warzige Narbengeschwulst" of Dieburg). It occurs whenever a burn, ulcer, or other injury produces a scar which hypertrophies and becomes painful.

Prognosis and treatment.—Trustworthy observers have recorded the spontaneous disappearance of cheloid tumours, but this must be extremely rare. They seldom or never ulcerate. They grow slowly and appear not to menace life, but the pain they occasion is sometimes severe.

Unfortunately, if removed by the knife, by galvano-cautery, or by caustics, the tumours almost always return. Nor have any of the milder applications which have been tried produced absorption. Mr Hutchinson, however, in an interesting paper on the subject ('Medical Times,' May 23rd, 1885), has recorded exceptional cases in which operation proved successful.

VASCULAR TUMOUR.* — Excluding moles or pigment-spots, true or vascular nævi have always essentially the same structure. But they vary in appearance, from the smooth, flat, "port-wine stains" as they are called, which sometimes cover the greater part of the face, head or even trunk, to the circumscribed pulsating tumour-like mass which can be removed by ligature, galvano-cautery, or other mechanical means.

* *Synonyms.*—Nævus flammeus—Nævi vasculares—Angiomata—Mother's marks.

Of similar structure though different pathology are the *stigmata* of gutta rosea and erythematous lupus, and the permanently injected patches which sometimes accompany the cicatrisation of lupus exedens, syphilis, or any other deep form of dermatitis.

There are, however, some rare and remarkable forms of disease of the skin which, though anatomically angioma, differ from true nævi not only in being acquired instead of congenital, but also in their course and event. Sometimes they will, as described by Hebra, while spreading in some directions, return to a normal condition in others; or, again, they may acquire a tumour-like and semi-malignant character, growing rapidly and forming large masses of erectile tissue. They are most often seen upon the extremities, though even here they are happily rare. They are sometimes complicated with fibrous growths, which have not only the pain of neuroma, but also its histological characters. Bruns recorded such cases on the lower extremities as *elephantiasis neuromatosa*.

Virchow and Kaposi described under the somewhat similar title of *elephantiasis teleangiectodes*, multiple fibro-vascular growths, which begin as separate lobulated tumours, but afterwards form diffuse, vascular thickenings of the skin.

Apparently identical with these is a case in a child under Dr West, which was examined by Dr Liveing and figured by the late Dr Tilbury Fox under the name of *fibroma fungoides* (pp. 352—354 of his work on 'Skin Diseases'). He there described other cases of fibro-vascular ulcerating growths which he considered to be of the same nature. One of these, however, may probably have been syphilitic.

Lymphangioma.—A curious affection of the skin, which has been described under this name, consists in what looks like a group of vesicles; but, on careful examination, they are found to be more deeply seated than usual, and in the event prove not to be inflammatory at all, but new formations, lasting unchanged for an indefinite period. In one case of the writer's they strikingly resembled the vesicles of zona, appearing in several groups, and arranged in a tolerably regular line. In this instance, the affection was complicated by appearing upon a large congenital port-wine stain, and the result was that many of the lymph-cysts became pink by admixture of their contents with blood, and when accidentally ruptured, thick red or black scabs were formed. This coincidence with ordinary vascular nævi (which others also have noticed) as well as histological investigation, seem to prove that the disease is rightly regarded as analogous to acquired vascular nævi. But Mr Hutchinson has described the affection under the unfortunate name of "lupus lymphaticus" ('Path. Trans.,' 1880, with fig.). Several cases have been brought before the Dermatological Society within the last few years. A careful histological description with figures by Mr Stewart will also be found in the 'Path. Trans.' for 1875, in the volume for 1879 (xxx, 474) by Drs T. and T. C. Fox, and in that for the following year (xxxi, p. 346) by Dr Sangster. Kaposi described a remarkable case of lymphangioma in a woman twenty-two years old who had several hundred violet-red pimples, round or oval in shape, some of them as small as a lentil, situated in the cutis and somewhat resembling certain forms of syphilodermia. A minute portion, being excised, showed that the cutis was filled with dilated lymph-spaces lined with endothelium. He named it *lymphangioma tuberosum multiplex*.

Mycosis Fungoides.*—Under this name Alibert described certain mulberry-masses of ulceration which he supposed to be syphilitic. Bazin in 1851 met with a case which resembled Alibert's account, and invented a *diathèse fungoïde* to explain it. In 1869 Ranvier showed that these curious soft tumours, readily ulcerating into fungous papillary masses, showed histologically a series of lymphatic spaces with cytogenic tissue between (Gillot: 'Thèse de Paris,' 1869). Auspitz has since discovered a micrococcus in these tumours which he thinks may be characteristic.

It is a very rare affection. It has been observed by Landouzy in an infant, but most cases recorded have been in adults, both men and women. The position of the tumour is usually on the trunk, less often on the face or limbs. It begins like patches of chronic eczema, with considerable itching, and after a variable period there follows deep induration and thickening of the cutis, with formation of separate raised swellings. These continue long stationary, sometimes shrivel again, but more often ulcerate and form the fungating tumours which are most characteristic of the disease. The progress is very slow, but with one or two exceptions the result is fatal.

The disease resembles both syphilis and leprosy, and in its early stages eczema and some forms of lichen. No treatment is known to be of service.

Xerodermia Maligna.†—Perhaps the most remarkable of all cutaneous diseases, one which is at once allied to nævi in its early stages and markedly malignant in its later development, is a rare affection, first described by Kaposi in 1870 under the borrowed title of *xeroderma*, a name which had been previously applied to a totally different condition by Wilson.

It begins with spots of erythematous appearance not unlike those of measles. Then they fade and form pigment-spots like freckles. The third stage may be months or years in appearing. When it arrives, the apparent ephelides become atrophic, the skin dry, thin, and wrinkled. Next it gradually contracts, so as to form a smooth, tightly-drawn surface, which may evert the eyelids or the lips or contract one of the joints. At the same time fresh brown pigment-spots and stigmata appear on the affected surface. The former undergo the same atrophic changes, the latter may increase until they resemble congenital vascular nævi.

The disease is not accompanied with itching or pain, yet after continuing in this comparatively innocent form for months or sometimes years, the vascular spots begin to become warty and to ulcerate. Fungoid growths of a most malignant character appear at last, not only in the maculæ, but also in distant places, and death ensues by hæmorrhage or exhaustion.

Hebra and Kaposi together observed only four cases of this remarkable affection. Erasmus Wilson described another under the name "general atrophy of the skin." Kaposi in 1885 ('Wiener med. Wochenscrift,' No. 44) tabulates only thirty-eight published cases; the youngest patient was five months, the oldest forty; eighteen were males and twenty females. Rüder saw seven brothers affected! A remarkable case was shown at the Clinical Society as one of lupus, and was recognised as identical with Kaposi's disease by Dr T. C. Fox. This same case, with the others in the same

* *Synonyms.*—Eczema tuberculatum (E. Wilson)—Lichen hypertrophicus (Hardy); who, however, now recognises its character as a new growth and accepts the title: Lymphadénie cutanée — Papilloma areo-elevatum (Beigel)— Granuloma fungoides — Sarcoma lymphadenoides (Auspitz)—Papillome etalé ou en plaque (Charpy).

† *Synonyms.*—Kaposi's disease. It has also been called "xeroderma pigmentosum," "angioma pigmentosum et atrophicum," and "atrophoderma pigmentosum."

family, will be found fully described by Dr Crocker in the 67th volume of the 'Medico-Chirurgical Transactions,' p. 169, with coloured lithographs, and a table of thirty-four cases. Most of these were recorded by Rüder in a monograph on the subject, the rest by R. W. Taylor, of New York, by Neisser, and by Vidal.

From this table it appears that the disease has never been yet observed above the age of puberty. One case occurred in an infant four months old, most under two years, one at nine, and one as late as sixteen.

It occurs in boys and girls indifferently and most frequently more than one case is found in a family. Twenty-six of the thirty-four cases in Dr Crocker's table belonged to nine families.

The histological characters of the spots are those of vascular dilatation, of pigmentation, and of atrophy. The final tumours appear to be always true epithelial carcinoma, not sarcoma.

Treatment has at present been unavailing.

The ordinary malignant growths of the skin are happily infrequent, nor have they many special points of interest; for their pathology is essentially the same as that of the corresponding growths upon mucous membranes; moreover, their recognition is not difficult and their treatment purely surgical, so that but little need be said of them in this place.

Carcinoma fibrosum, or scirrhous cancer, the most typical of all the forms of cancer, rarely affects the skin primarily, though it frequently infiltrates it as the result of primary carcinoma of deeper parts, as, for instance, of the mamma. The writer has seen three examples of the remarkable form of hard, indurating, and widely-spread cancer of the skin, described by Velpeau as *squirrhe en cuirasse*. One was a patient under Velpeau himself, in whom the disease had spread from a cancerous breast; another was a patient of Dr Humphry's, of Cambridge. The remarkable and widespread induration, before ulceration begins and before implication of deeper organs occurs, renders it peculiar; and causes a superficial resemblance to sclerodermia or to certain forms of lupus. It is usually secondary to mammary cancer.

Epithelioma,* or keratoid cancer (see vol. i, p. 118), is the most common form of malignant disease in the skin. Even this is rare, compared with its frequency in the œsophagus and large intestine, and at the labial, anal, and urogenital orifices. Its formerly most frequent seat, the scrotum, is happily no longer so, and "chimney-sweep's cancer" has become a rare curiosity in this country.

Rodent ulcer.—This affection, originally described by Jacob, of Dublin, is now ascertained to be histologically carcinoma. (See Mr Hulke's paper in the 'Path. Trans.,' vol. xxii.) The presence of epithelial cells in the cutis vera and of the nest-cells characteristic of the horny form of cancer leave no doubt of its real pathology. It is, however, the least malignant of cancerous growths, for it spreads slowly, there is little new growth, and it rarely affects even the neighbouring lymph-glands. It is usually seen near the eye, upon the side of the nose, on the cheek, or the temple. Like other kinds of carcinoma, it is a disease of mature life or of old age. Its early stages are those of a small, smooth, pale growth not unlike a wart. If, as

* The term *epithelioma*, applied by Hannover, of Copenhagen, to this disease, of which he was the first to describe the histology, was discarded by Virchow for "epithelial cancer." Unfortunately "epithelioma" is now used by some German writers to designate molluscum contagiosum.

is sometimes the case, it has begun in a congenital mole, it retains the pigment of that structure. It often has a pearly aspect, so as to look somewhat like a molluscum tumour or even like the cysts not unfrequently found about the eyelids. When ulceration begins, it is covered by a rather thin, dark, and adherent crust. It produces little or no pain and advances so slowly that when it first comes under the surgeon's eye it presents the appearance of a chronic, indolent, indurated ulcer with sharp, well-defined nodular edges, and no granulations. In its later stages it resembles more nearly its pathological allies, epithelial cancer of the lip, the scrotum, the glans, or the vulva.

Other cases which are clinically rodent ulcer appear to have a different histological structure (Verneuil, 'Arch. gén. de Méd.,' 1854, ii, 458, and Thin, 'Path. Trans.,' 1878, pp. 237, 241). A review of these and other papers from Thiersch downwards, by Dr Hume, of Newcastle, with histological drawings, will be found in the 'Brit. Med. Journ.,' Jan. 5th, 1884.

The diagnosis from tertiary syphilis lies in the ulcer being single, in its not invading the bones or other tissues, and in there being no other sign of syphilitic disease. From lupus it is distinguished by the scab being thin and dark, by its beginning at a much later period of life, and in the last resort, by microscopical examination of the material obtained by scraping or squeezing the edges of the ulcer. See also p. 1033.

Sarcoma.—Beside carcinoma in the proper sense of the word, the skin is occasionally liable to multiple sarcomata. These are almost always secondary to some internal growth; by their large number, small size, and hæmorrhagic or sometimes melanotic character they may resemble certain forms of purpura or pigmentation. Cases of this remarkable affection have been already described in the first volume, pp. 95, 96.

PIGMENTAL, HÆMORRHAGIC, AND NEUROTIC AFFECTIONS OF THE SKIN

Albinism—Leucodermia, congenital and acquired—Its relation to leprosy—Canities —Melanodermia, secondary to dermatitis, syphilis, adrenal disease, malaria, &c. —Ephelis—Lentigo—Chloasma—Relation of melanodermia to leucodermia. Petechiæ and vibices—Peliosis rheumatica.
Trophic neuroses—Zona—Symmetry in cutaneous diseases.

Anomalies of Pigmentation.—We are familiar with degrees of pigmentation of the skin not only in different races, but also in the wide difference between individuals belonging to the same stock, and even to the same family.

Albinism, or complete absence of pigment, not only from the skin and its appendages, but from the iris and choroid, is always a *congenital* defect in the human race, as in rabbits, mice, horses, and other animals. The so-called "white" elephants are either albinos or piebald.

Albinos occur occasionally among the dark races. The "white" negroes have a dirty pale skin, colourless hair, and pink irides with dark red pupils.

*Leucodermia.**—This also may be a congenital variety of colouration or "malformation." Piebald horses may be called "abnormal," but we should scarcely say so of cattle, dogs, swine, or guinea-pigs. This condition is, however, far more common in domesticated races than in a state of nature.

A similar congenital "piebald" state of the skin is occasionally seen in human beings. In negroes and in the Indian population it appears to be not uncommon. We sometimes see it in this country, as white locks of hair.

When acquired after birth, leucodermia has been, and still is, confounded with leprosy. In fact, "white leprosy," when it does not apply to psoriasis, seems generally to mean leucodermia occurring in patches.

Celsus, lib. iii, c. 25, distinguishes elephantiasis (*i. e.* leprosy) from *vitiligo* (calf's skin, parchment skin), which he divides into three species (lib. v. c. 16):—*V. alphos*, scattered, colourless, slightly rough patches; *V. melas*, pigment-spots, to be presently mentioned under melanodermia; and *V. leuce*, still whiter than alphos, with white hairs growing on the patches. But later writers speak of vitiligo, and more particularly of leuce, as varieties of leprosy. The same explanation appears to apply to the Arabic term "Baras," the equivalent of Leuce or Alphos in Greek and Vitiligo in Latin, which was also intended to denote a white leprosy. The confusion is due to patches of skin occurring in true leprosy, which are either deeper or paler in tint than the surrounding surface. The ambiguity appears still to exist not only among the natives of Southern India and Ceylon, but among some physicians, judging by their reports in the Blue-book referred

* *Synonyms.*—Partial albinismus—Vitiligo—White leprosy. The term vitiligo has been also applied to a circumscribed smooth white indurated spot level with or slightly sunk below the surface. This would make it identical with morphœa, *i. e.* with circumscribed scleroderma (*v. supra*, p. 990). The word may well be abandoned.

to before as published in 1867. Dr Vandyke Carter states expressly that leucodermia is commonly confounded with anæsthetic leprosy.

The skin in leucodermia is perfectly normal except for the loss of pigment. The Malpighian layer and also the hair are affected. There is no anæsthesia. The border is convex, and often a pigmented line separates it from the normal skin around. This was the case in the specimen which Gustav Simon first examined histologically. The patches are usually multiple, sometimes very numerous. They are occasionally symmetrical,* more often irregular, with no predilection for one surface of a limb or the other. They may occur anywhere, but are most frequent on the trunk, especially the abdomen and genitals, where natural pigmentation is deepest.

Leucodermia is more common in hot countries and in the south of Europe than in England, but here cases are readily overlooked, since in most cases they are inconspicuous, and give rise to no discomfort.

Removal of the white patches has been attempted by blisters and other irritants, and also by tattooing. The result is not often satisfactory.

Canities.—General blanching of the hair is a well-known senile change. But, like baldness, it often occurs in early adult life, especially when the hair is very dark. Besides suffering loss of pigment, which gives the dull, yellowish, "milk-white" appearance, the hair is apt to become dry and admit air-bubbles, which increase its refractive power, and produce the glistening steel-grey or "silvery" aspect.

Many instances are on record of rapid blanching of the hair of head or face in consequence of mental anxiety or grief. The cases of Sir Thomas More, of Henry the Fourth of France, and of Marie Antoinette, have become historical, and it seems impossible to deny the fact that this premature senile change may come on in the course of a few hours. Bichat and Alibert record cases which they actually saw, and Brown-Séquard has seen rapid blanching in his own beard ('Arch. de Phys.,' 1869, p. 442). A grey patch sometimes follows neuralgia (cf. vol. i, p. 671). The late Dr Laycock quoted an instance in which a sepoy was seen to turn grey in half an hour ('Med. Times and Gaz.,' 1862). A young man who once consulted the writer for some slight ailment had perfectly white hair. In answer to inquiry he stated that a few years before he had fallen asleep after a debauch, and on waking in a cold room in the morning found that his hair had turned white. The objection that his beard was brown was answered by the explanation that when the change of colour occurred it had not yet grown.

This sudden change is probably due to development of air-bubbles in the shaft of the hair.

Melanodermia (*Melasma cutis, nigrities*).—Increased pigmentation of the skin, like its diminution, may occur either universally or in patches.

The former condition is never congenital like albinismus. A dark skin at birth is always hereditary. It may occur as the result of exposure to the heat of the sun or to other irritants, or as the result of certain internal diseases.

As the result of hyperæmia, or slight superficial inflammation ("eczema solare"), one sees increased pigmentation produced by the wind in cold weather, or in driving, or by the cold of snowfields, which, as Alpine climbers know, will scorch the face without sunshine. Among Professor Hebra's patients there once appeared a youth who had wandered over a

* See a remarkable case of perfectly symmetrical leuco- and melanodermia figured by Dr Lesser ('Ziemssen's Handbuch,' Bd. xiv, 2te Hälfte., p. 186, fig. 11).

great part of Hungary in rags during the depth of winter. The exposed parts of the skin had become almost the colour of a mulatto, yet there had been little or no sunshine.

Although all hyperæmia produces more or less increased pigment, there is considerable difference in different inflammatory diseases. The deeper and more chronic forms of dermatitis have very little effect, as we should anticipate from their primary seat in the cutis vera. Long standing eczema and chronic traumatic inflammation produce much darkening of the skin, as seen in the brown, almost black patches which surround indurated varicose ulcers in old people. Ordinary eczema, however, has little effect, and impetigo and scabies none at all.

Of the superficial inflammations, chronic inveterate prurigo produces the greatest pigmentation, and prurigo pedicularis almost as much, aided probably by the scratching which it occasions and also by the age of the patient; for all pigmentation is slow in childhood and rapid in old age.

Certain forms of erythema are accompanied with increase of pigment, particularly pellagra (acrodynia), and urticaria pigmentosa.

Psoriasis very early and readily causes pigmentation, and the colour is sometimes quite indistinguishable from the coppery hue of a syphilitic eruption. Indeed, we may say that next to syphilis, psoriasis will produce pigmentation in the shortest time.

Besides the well-known brownish pigment which gives its characteristic colour to even early forms of specific eruption, a somewhat rare form of syphilide has been described by French authors as the "café au lait" form (*syphilide pigmentaire*). Cases have been seen, both in Paris and in London, in the form of ill-defined brownish maculæ occurring on the neck of women who were the subjects of secondary syphilis.

Pigment has already been mentioned as occurring in some cases of sclerodermia and in the malignant kind of atrophic nævi of the skin called "xerodermia" by Kaposi (p. 1018). The remarkable increase of pigment in the course of Addison's disease has been fully described in the chapter on that subject. Similar pigmentation, though far less intense, is observed as the result of malaria, and occasionally in the cachexia of cancer.

Maculæ—Ephelides—Lentigo.—It remains to mention circumscribed pigment-patches, which occur without inflammation and independently of any other morbid sign. The most familiar are the small, dark brown or yellowish spots which, when they occur on the face, are named freckles (*ephelides*). They are no doubt, as their name implies, the result of exposure to the sun. They occur most frequently on the face, but also upon the hands and arms when these are bare. They are almost confined to xanthochroic complexions, and are particularly common in persons with red hair, blue eyes, and the delicate pink and white skin which so often goes with them. These freckles, like the diffuse pigmentation of sunburn, disappear in time, though much more slowly.

Precisely similar minute dark spots appear in covered parts of the skin, and in mucous membranes, sometimes along with the melasma of Addison's disease or with pigmentation from malaria, and sometimes in conditions of health. Others are congenital and may then be described as pigmentary nævi or "mothers' marks." When combined with a congenital papillary growth, often covered with a strong growth of hair, they are called "moles."

Chloasma.—More diffused and less intense patches of pigment occur upon

the forehead of pregnant women, and have long been known under the name *chloasma uterinum*. In some cases they appear during each pregnancy and disappear after delivery. The word chloasma was at one time extended to the pigmented patches on the trunk which we now know to be due to a fungus and call tinea (or pityriasis) versicolor. But there seems no reason why at present the term should not be reapplied in its original signification.

Similar pigment-spots on the forehead and about the eyes are symptomatic of ovarian irritation, and appear in some cases of dysmenorrhœa with each menstrual period. (See eight cases reported by Dr Champneys with valuable comments in the 'St Barth. Hosp. Rep.,' vol. xv.)

Such pigmentation may also be the result of sexual excesses in male subjects, but this cannot be distinguished from the dark circles round the eyes which often accompany severe attacks of headache, especially megrim. All these cases may be grouped together by their clearly neurotic origin. They must be carefully diagnosed from not unfrequent instances in which lamp-black or other pigment has been designedly applied to the face, forehead, and eyes by hysterical or otherwise deceitful women.

Lastly, there are certain cases in which patches of pigmentation occur in various parts of the body, unconnected with local irritation and without any nternal disease.

These cases of idiopathic circumscribed melanodermia are decidedly rare and are generally associated with leucodermia; that is to say, white patches occur in the pigmented surface. The former are sharply defined and have convex borders, the dark surface is most marked close to the white (beyond the effect of contrast), and gradually shades away into the normal skin. Most cases may be called either melanodermia or leucodermia, or both at once; and apparently consist in an irregular distribution of pigment. The white patches come usually first.

Treatment.—Solutions of corrosive sublimate, such as "virgins' milk" and "Gowland's cosmetic" (p. 946), are believed to have the power of removing freckles. The mingled patches of white and dark skin just described are best left alone, but circumscribed pigmentary nævi which cause disfigurement on the face may, if small, be removed by excision or galvano-causis.

Cutaneous hæmorrhage.—The most important conditions in which ecchymoses, whether the small ones like fleabites (hence called *petechiæ*) or the larger ones named *vibices*, are seen upon the skin are those of scurvy and of purpura. In both cases hæmorrhage occurs in other parts as well as the cutaneous surface, and in scurvy the ætiology of the disease serves to define it. Both affections have been already discussed (pp. 780, 787).

It only remains to mention the principal cases—beside ecchymosis from mechanical injury—in which hæmorrhage accompanies cutaneous diseases.

The peculiarities of hæmorrhagic smallpox and scarlatina have been already described (vol. i, p. 229 and also p. 237, *ibid.*, p. 213).

In eczema, scabies, psoriasis, lichen, and prurigo, ecchymoses never occur except as the result of scratching. Slight hæmorrhage often tinges the contents of the bullæ of pemphigus, especially in the gangrenous, and cachectic forms of the disease; but it seems not to occur either in Herpes gestationis or in Pemphigus foliaceus.

All forms of erythema are liable to be complicated with hæmorrhage. It is a rare complication in urticaria (when it constitutes the *Purpura urticans* of Willan), very common in erythema nodosum, when it produces the subsequent

ruise-like pigmentation, and most frequent in the forms of erythema which occur in the course of rheumatic fever. This last condition seems to have been first observed by Schönlein, who named it *Peliosis rheumatica*. The erythematous patches appear acutely, with fever and synovitis. They are most often seen on the back of the hands and feet, the forearms and shins, but may also affect the thighs, hips, and trunk; they are not often symmetrical. Either from the beginning, or soon after their appearance, the redness is found no longer to fade on pressure; hæmorrhage has taken place. Successive crops of these papules or large patches may occur, each lasting about a week, and disappearing with only a slight macule to mark its place.

There is no need of a special name for this disorder. It is a true erythema, whether occurring in the course of rheumatic fever or in persons who have already suffered from that disease, and we have seen that both this connection and liability to hæmorrhage are characteristic of the whole group of erythemata. On this subject see "A Case of Rheumatic Purpura," with notes by Dr Wickham Legg ('St Barth. Hosp. Rep.,' vol. xix).

Neurotic affections of the skin.—The only cutaneous disease which is certainly related to nervous disturbance is the following.

ZONA.*—The names for this disease owe their origin to the fact (with which the elder Pliny was acquainted) that it passes round the trunk of the body like a girdle. The brief notice of it given by Pliny also implies the knowledge of another striking feature, namely, that it is limited to one lateral half of the cutaneous surface. He says "*enecat, si cinxerit,*"—"it kills, if it encircles," and a popular tradition to the same effect still exists in England. Nevertheless, zoster is never fatal, and does not pass over to the other side of the body from that first affected.

The most elementary acquaintance with anatomy could not fail to suggest, to anyone who had observed the distribution of the eruption in a case of shingles, that it corresponds exactly with that of the peripheral distribution of one or more of the dorsal nerves. Accordingly this has been recognised for many years; and, with it, the necessary consequence, that certain eruptions on the face and limbs, which follow the course of the nerves supplied to those parts, are identical with it. To these also the name of herpes zoster is now given, although when so applied it loses its meaning, since the affected area no longer has the form of a belt.

The *eruption* of shingles consists of vesicles. These are of flattened form, and larger than those of eczema, being often as big as split peas; they are arranged in clusters of perhaps twenty or thirty, each cluster lying on a reddened and slightly swollen patch of skin; when the vesicles are thickly set, they often run together, and form flat bullæ of irregular shapes.

Some years ago, Dr Haight, of New York, found an opportunity in Vienna of investigating their structure with the microscope. His observations showed that their roofs consist of the horny layer of the cuticle, with some of the superficial elements of the rete mucosum adherent to the under surface; their floors are formed by the bare summits of the papillæ, with the deepest elements of the rete occupying the depressions between them; their cavities are traversed by numerous bands, consisting of masses of the intermediate elements of the rete, drawn out into long spindle-cells and cells with several tapering processes. The fluid which the vesicles contain is at

* *Synonyms.*—Herpes zoster—Shingles; a corruption of Cingulum = zona and zoster, a girdle.

first transparent, but after a time the presence of floating leucocytes renders it opalescent, and ultimately it may become purulent, or acquire a purple colour from the escape of blood through the softened tissues beneath. The cutis itself seems always to take some share in the inflammation, leucocytes being scattered in the spaces between its fibrous bundles, and along the vessels and nerves. When pus is formed, if the roofs of the vesicles have been removed by the friction of the clothes, ash-coloured surfaces are exposed, looking like layers of false membrane. In other words, the histological changes in zoster are essentially identical with those which have been described at p. 231 for variola.

The number of clusters is very variable, from a single one to ten or even more. They are generally developed, not all at the same time, but in quick succession; those coming out first which lie nearest the roots of the nerve whose branches they follow. After a few days fresh ones cease to make their appearance. There is a short papular stage; and some of the latest clusters not infrequently abort, without going beyond it. In certain very mild cases, when only one or two clusters are formed, none of them pass into a vesicular condition. Even if the disease should be of considerable severity, the eruption begins to dry up from the fifth to the eighth day; the centres of the vesicles become depressed, yellowish or brownish crusts form, and in the course of the third week these fall off, leaving reddish or purple stains. But when the cutis is thickly infiltrated with pus-cells, its superficial layer undergoes destruction, and an eschar has then to be thrown off: thus the process of healing is retarded, and an indelible cicatrix results. The distribution of such cicatrices in the course of a particular nerve shows at once the nature of the disease from which they arose. When zoster attacks the face it is particularly likely to leave permanent scars behind it.

Writers have given names to numerous varieties of zoster, according to the nerves affected; but such distinctions are useless. When the disease affects the face, the nerve which it follows is the fifth, the greater part of which answers to the sensory portion of an ordinary spinal nerve. Indeed, it is remarkable how exactly the clusters of vesicles sometimes map out the points of emergence of the several twigs of the trifacial nerve from their bony canals. When the first division of the nerve is affected, the loose tissue of the upper eyelid becomes extremely œdematous and swollen, so that the affection may be mistaken for erysipelas by a careless observer. Another peculiarity of this form of zona is that it is often attended with ulceration of the cornea and iritis, by which the sight may be seriously damaged. Mr Hutchinson, who has specially investigated such cases, has remarked that the ocular affection never arises unless the eruption occupies the distribution of the nasal twig. When the two lower divisions of the trifacial nerve are involved, a few vesicles often appear on the mucous membrane of the mouth and palate. Paget has recorded an instance in which necrosis of the alveoli followed inframaxillary zona, so that some of the teeth fell out.

Cervical zoster, and that which affects the upper limb, follow exactly the distribution of the several nerves. In some instances the vesicles reach down to the fingers, but this is very exceptional; in the great majority of cases they do not extend below the elbow.

On the trunk, which is by far the most frequent seat of the disease, the area occupied by the eruption of course slants more and more downwards as it approaches the pubes. It often happens that one or two vesicles lie

slightly beyond the meridian plane, both at the linea alba and at the spine; this probably depends upon the fact that the nerves of the opposite sides overlap in their distribution, just as in the Siamese twins there was a part of the connecting band which received nervous filaments from each of them.

In the lower limb the distribution of zoster presents this peculiarity, that it is almost invariably confined to the buttock and thigh. Mr Hutchinson says that it never extends below the knee, and the only instance to the contrary is one figured by von Bärensprung in which there were a few small papules as low as the middle of the calf.

Mr Hutchinson once saw a zoster in the course of the fourth dorsal nerve on the *right* side associated with a frontal zoster on the *left* side. In one of von Bärensprung's cases an ordinary zoster, limited to the *right* half of the thorax, was accompanied by a single vesicle in the *left* axilla, the patient having been suffering from severe burning pains on both sides.

An attack of shingles is very seldom attended with any febrile disturbance or disorder of the general health. In children who are liable to it at all periods after the first year, it commonly runs its course without any unpleasant sensation, or is merely accompanied by a little numbness and tingling. Von Bärensprung tested the cutaneous sensibility with a pair of compasses, and found that in two cases it was considerably increased, while in a third it was diminished. Sir Thomas Watson relates a curious case in which zona affected the scalp, and in which the patient, who had for seven years been plagued with continual noises in the head, became free from this symptom, and remained so for eighteen months afterwards. He also mentions another case, of a man in whom the eruption came out in February, and who suddenly lost a cough which had teased him all the winter. On the other hand, von Bärensprung met with two cases of zoster affecting the distribution of the fourth cervical nerve, in each of which vomiting occurred at the commencement,—a consequence, he supposes, of "sympathetic irritation" of the vagus and phrenic nerves.

But by far the most important subjective symptom of herpes zoster is pain of a neuralgic character, and referred to the same nerves, the distribution of which is followed by the eruption. This is entirely absent in young patients, but in adults it is generally present, and in old people it is apt to be exceedingly severe. Von Bärensprung cites a case in which there was only a single patch, of the breadth of two or three fingers, but in which the tenderness was such that the patient kept the part covered with the palm of his hand all night and all day, lest his linen should come into contact with the vesicles. When the eruption gets well the pain commonly subsides; but in some instances it continues long afterwards, for months, or even for years, with scarcely any abatement of its intensity. This suffering may last ten years; and Trousseau mentions an instance in which the pain lasted for fourteen years. Sir Thomas Watson alludes to a case of this kind in which the patient, a lady, could at all times bring on the pain by drinking some cold liquid. The persons in whom shingles leaves behind it this terrible neuralgia are always advanced in age.

Sometimes the pain precedes the development of the eruption by several days. This has led certain writers, among whom was Anstie, to regard herpes zoster as a mere complication of neuralgia, analogous to several other curious "trophic" changes which are met with in that disease. But in opposition to such a view it must be urged that in the majority of cases of shingles (in two out of three, according to von Bärensprung) pain is

altogether absent, and that when it is the earliest symptom the cutaneous affection always appears within a definite period, a fortnight at latest. Some instances have, indeed, been recorded, of an eruption, regarded as zoster, which seemed to be caused by pressure upon the corresponding nerve-trunks, or which occurred in the course of ordinary neuralgia. Thus Charcot and Cotard have published a case in which one half of the neck and one shoulder were covered with the vesicles, the cervical nerves of that side being compressed by cancerous disease of the vertebræ. Charcot is said to have met with another case in which a patient, during a second attack of sciatica, presented herpetic vesicles on the lower part of the thigh. In these instances it seems not improbable that the eruption really was shingles, and that it was caused by an extension of the morbid process from the trunks of the nerves to the ganglia on the posterior nerve-roots. But there are other cases of which such an explanation is less easy. Thus v. Bärensprung reports an observation by Esmarch of abscesses about the pelvis and under the gluteus maximus, where *post mortem* the great sciatic nerve was found to be swollen and reddened. About five weeks before the patient's death an eruption of groups of vesicles appeared on the back of his leg and on the sole of the foot as far as its middle. In this instance the seat of the cutaneous affection was unlike that of zoster, for that scarcely ever goes below the knee. The same objection applies to Charcot's oft-quoted case of a man who suffered from pain in the leg and back of the foot after a gunshot wound of the thigh, and in whom a herpetic affection repeatedly developed itself upon those parts. Moreover, the fact that the eruption recurred is of itself sufficient to show that it was not really zoster, for shingles never relapses.

Mr Hutchinson goes so far as to maintain that zona protects against itself, like an exanthem. In a series of a hundred cases collected by him there was only one in which there was any history of a previous attack. But this may depend on the fact that shingles is of infrequent occurrence, and perhaps does not affect more than 1 per cent. of the population.

If further arguments are needed, one may be found in the fact that herpes zoster is associated with a series of changes in the ganglia of the posterior nerve-roots, such as are not known to occur in neuralgia proper. That these ganglia are the starting-points of the disease was suggested by von Bärensprung in 1861. His opinion was verified by Charcot and Cotard, who found (in a case already referred to) that whereas the nerve-roots were healthy, the ganglia and the nerve-trunks to a little distance outside the intervertebral notches were much reddened and slightly swollen, their stroma being also unduly rich in nuclei. Precisely similar appearances were afterwards discovered by von Bärensprung himself in a child which died soon after an attack of shingles.

With regard to the *causes* of herpes zoster almost nothing is known. It occurs equally in both sexes, and at almost every age. It has repeatedly been observed in persons who have recently been taking arsenic.

In the *treatment* of this disease there is but little to be done. The vesicles must be protected by a soft linen rag, with a pad of cotton wool or a flannel bandage. Some writers recommend that flexible collodion should be painted over them to facilitate their drying up; or a little starch powder may be dusted over the affected part as soon as any discharge appears. Children require no medicine whatever.

The treatment of the neuralgia which sometimes follows shingles is

unsatisfactory. Bazin is said to have used arsenic with success; but as a rule it utterly fails. Dr Fagge used to prescribe vinum colchici; in several instances the pain has quickly subsided while the patient has been taking this medicine. Locally, anodynes are generally applied, but without much benefit. Von Bärensprung says that he found blisters useful.

The sensory nerves of the skin lead by the medium of *pruritus* to scratching, and how important an agent this is has been shown in our accounts of eczema, prurigo, scabies, urticaria, &c. These pruriginous diseases are in striking contrast to syphilodermia and lupus.

But it has been supposed that disorders, not of sensory but of trophic nerves, produce cutaneous diseases other than zona. The existence of trophic nerves is a physiological fact, but their presence gives so easy an explanation that we must beware of admitting it without adequate proof.

Area has been referred to a neurotic cause, but there is no proof of it. Little if any anæsthesia is to be detected, and the patches do not follow the course of cutaneous nerves.

Leucodermia has been referred to the same cause. But here again there does not seem to be any reason for ascribing the disease to nerves, but the difficulty of finding a better explanation. All that can be said in favour of the neurotic origin of *morphœa* will be found in Mr Hutchinson's 'Clinical Lectures,' vol. i, p. 313.

The formation of the wheals of *urticaria* may be ascribed to vaso-motor nerves, and it sometimes follows mental emotion with great rapidity.

Certain *bullous* eruptions also appear to be connected with neuroses.

Some authorities believe that symmetry points to a nervous origin of a cutaneous disease; but others hold symmetry to be the mark of "blood diseases" and asymmetry that of neuroses. Neither belief seems to be well supported. Symmetry points neither to a constitutional nor to a blood disease. All general diseases are symmetrical because the human body is so; a one-armed man would be unsymmetrically affected by scarlatina or psoriasis. Again, psoriasis and eczema are symmetrical because they affect the skin of a certain structure and surroundings which is found on the corresponding parts of the limbs, ears, and other parts. The only disease of the skin which we know to be of nervous origin—zona—is, like neuralgia, markedly unsymmetrical.

See, however, on this subject a valuable paper by Dr Crocker, with numerous references ('Brain,' October, 1884, p. 343), and the monograph by Leloir, 'Recherches sur les Affections cutanées d'origine nerveuse,' 1882.

REMARKS ON THE PRACTICAL CLASSIFICATION AND DIAGNOSIS OF CUTANEOUS DISEASES

In the preceding chapters the diagnosis between two diseases which may be mistaken one for the other has only occasionally been stated in a formal manner. If the characteristic symptoms and course of a malady are duly described, they form the only bases for its diagnosis; and although it is a useful exercise for a student to make lists of the distinctive characters of two or more diseases, the attempt to fix them in a tabular form is of little service to others, and perhaps tends to artificial memory of words rather than to familiarity with things. Symptoms differ endlessly at the bedside, and none of them are really what is called "pathognomonic." Moreover, the diagnosis of cutaneous diseases in particular often turns upon very slight differences in the form of lesion or in the distribution, which it is impossible to put into words. Lastly, much of what is called diagnosis is not distinction between one pathological condition and another, but only between certain more or less arbitrary forms which have been fitted with still more arbitrary names.

In the present chapter it is proposed to treat briefly of this question of diagnosis on which, in its true meaning, all successful treatment must rest.

There are not a few diseases which are so rare that they rank as little more than as curiosities. Such, for instance, are Pemphigus foliaceus, Urticaria pigmentosa, Favus, and Xerodermia maligna.

Many important diseases of the skin again are exotic, as Leprosy and Lichen agrius, and only of practical importance for English readers who may practise their profession in India or the colonies.

Neglecting these, there are certain common affections which, differing by more or less important characters in appearance and in histology, nevertheless agree very closely in their general pathology, in their causes so far as they are known, and, what is most important, in the kind of treatment which is generally suitable.

From a practical point of view, looking chiefly to questions of prognosis and treatment, we may divide diseases of the skin into the following :—

I. *Factitious eruptions.*—We must never forget the possibility of the affection before us being artificial. All kinds of dermatitis, eczema, erysipelas, pemphigus, impetigo may be simulated by the application of various irritants. Pigmentation also has been often imitated with success. Such artificial diseases will generally be found upon the arms, rarely on the face, and scarcely ever beyond reach of the patient's hands. The persons who are guilty of such attempts at imposition are usually either deliberate malingerers, like prisoners in gaol, or else they are hysterical young women and neurotic girls or boys. When one's suspicions are once awakened it is seldom difficult to detect the imposture. Mustard, cantharides, and some other irritants can be distinguished by help of the microscope.

II. *Traumatic eruptions.*—In all cases of dermatitis we should seek for the irritant, and sometimes it is so directly the cause of the disease that the

eczema or impetigo in question may be considered purely traumatic, and efficient treatment immediately follows accurate diagnosis: *sublata causa tollitur effectus.*

Pediculi in the hair should be carefully looked for in all cases of impetigo in children, pediculi vestimentorum in prurigo of old people. Scabies itself is but an extremely definite and well-characterised dermatitis resulting from the presence of a living source of irritation. But beside these well-known cases of parasitic dermatitis it will be found that some supposed cases of purpura in children are nothing but fleabites, resemblance to which originated the name petechia. Moreover, many cases of infantile prurigo, urticaria, and ecthyma are due to the presence of bugs or gnats. In adults pediculi pubis may sometimes be found in the axillæ as well as in their proper region, and when they have been destroyed by mercurial ointment the patient is at once relieved from pruritus.

In many trades an irritant must be sought in the objects which the patient habitually handles. The coarser kinds of brown sugar are a frequent cause of eczema of the hands (grocers' itch). So with many of the "chemicals" used in a variety of modern handicrafts. Constant wetness of the hands in washerwomen, in scrubbers, in potmen and many others produces eczema rimosum. The heat of the sun is the cause of eczema solare and ephelides, the heat of the fire of the pigment-spots on the shins of elderly people. Sweat again is a very common irritant, producing the erythema which usually accompanies sudamina and also intertrigo of opposed surfaces. Scratching as a cause of traumatic dermatitis has been repeatedly referred to.

III. *Febrile rashes.*—We must take care never to forget the possibility of a cutaneous eruption being part of an acute exanthem. The use of a clinical thermometer is a great help in this respect, but the writer has seen a man with typhus (and the rash fully out) appear as an out-patient for a skin disease, and modified variola and varicella are not unfrequently mistaken for acne or impetigo.

IV. *Syphilodermia.*—When we have satisfied ourselves that the eruption before us is not factitious, nor directly traumatic, nor a symptomatic eruption, we may next consider whether or not it is due to syphilis. In this inquiry it is undesirable to ask questions, the answers to which are as apt to mislead as to guide aright.

(1) We should first consider the *colour* of the affected skin, remembering, however, that the pigmentation which gives the so-called coppery or raw ham tint to a syphilitic eruption is the same which is sooner or later produced by all forms of dermatitis. Psoriasis, chronic eczema, lichen planus, and prurigo may all produce shades which bear the closest resemblance to syphilodermia.

(2) The lesions of syphilis are *multiform* or polymorphic. It is rare in any but syphilitic affections to find mere hyperæmia in one part and associated pustules, papules, scales, or ulcers in others, and it is not often that a syphilitic eruption exhibits only a single elementary lesion.

A pustular eruption in an adult should always suggest the question of syphilis, when that of scabies has been answered in the negative.

(3) Syphilitic eruptions for some unknown reason *do not itch*, and the exceptions to this rule are remarkably few; they usually occur during the

stage of scabbing of pustular rashes or during the healing of tertiary ulcers. An ordinary secondary syphilide may, however, as a rare exception, be so irritable that wheals and scratch-marks are produced. On the other hand, psoriasis is often free from irritation, while the degree of itching of eczema and even of scabies and prurigo varies greatly.

(4) The local *distribution* of syphilitic diseases is a great aid in diagnosis. Specific eruptions are certainly not, as is often stated, symmetrical; the early roseolous rash is only so because it is general, and therefore, upon a symmetrical surface like the human body, more or less symmetrical. Moreover, as it chiefly affects the face, chest, and trunk generally, it is near the middle line. But we do not see symmetrical patches of syphilide in corresponding parts of both sides of the face, both sides of the trunk, or the right and left limbs. In all but the earliest syphilides the affected patches are very decidedly and constantly *unsymmetrical*, irregularly scattered over head, trunk, and limbs, and chiefly remarkable for having no well-marked seats of predilection.

The forehead, especially about the roots of the hair, is, however, very frequently the seat both of the early and middle erythematous, scaly, and pustular syphilides, and the palms of the hands and soles of the feet are frequently symmetrically affected with the later scaly eruption.

Practically, when we find a disease of the skin occupying some unusual position we should at least consider the question of syphilitic origin.

(5) These signs alone or in combination serve to distinguish early specific roseola from erythema, eczema, scarlatina, and measles, and the later eruptions from eczema, lichen, scabies, impetigo, and psoriasis.

The eruptions of *congenital syphilis* which are most liable to be mistaken are—the so-called pemphigus of infants, which is known by its affecting the palms and soles; rupia, which by the form of the crusts and the ulcerated surface beneath, may always be distinguished from impetigo; an erythematous rash of the nates and genitals of infants, which is distinguished from eczema of the same parts, also common at that age, by its coppery colour, its blotchy distribution, and more defined margin.

The *tertiary ulcers* of syphilis are distinguished by their appearing on unusual places, by their punched-out edges, circular or so-called horseshoe shape, and by their usually producing little pain or discomfort. Tertiary ulcers have no predilection for the outer side of the leg, but inasmuch as the part above the inner ankle is for anatomical causes the chosen seat of varicose ulcers, most ulcers in the first position will be syphilitic, and in the latter not. For the same reason most ulcers on the arms are found to be tertiary.

V. *Tineæ.*—The next great group of skin diseases includes those which are due to vegetable parasites—tinea versicolor of the trunk, eczema marginatum of the perinæum and thighs, tinea circinata of the neck and other parts, tinea sycosis of the chin, and tinea tonsurans of the scalp. Here the general characters detailed in the chapter on the tineæ are generally sufficient to show the nature of the affection to a practised eye, but in all doubtful cases the microscope should be employed.

Tinea of the scalp is rare in adults, and tinea circinata still more so; tinea marginata occurs only in adult males.

VI. *Primary superficial inflammations.*—To distinguish the superficial from

the deeper kinds of dermatitis we should notice whether the cutis alone is infiltrated and thickened or whether it is bound down by adhesions to the subcutaneous tissues. The presence of scars, however slight, is a proof that the process has gone deeper than the papillæ and has more or less extensively destroyed the papillary layer. Superficial inflammations, excluding those due to the acarus, to pediculi, and to other direct irritants, and excluding those which are the result of vegetable parasites and of syphilis, fall with respect to their treatment into three large groups :

(1) The first, represented by impetigo and most forms of eczema, are subacute and accompanied with burning, itching, and pain, sometimes with a slight degree of fever. They are to be treated by local remedies designed to reduce the hyperæmia, diminish the exudation and calm the irritation, aided by light diet, free diluents, laxatives, and diuretics. In short they are to be treated according to the modern antiphlogistic method.

(2) The second group of superficial inflammations of the skin is typically represented by psoriasis, but includes lichen planus, the more chronic, dry, and obstinate forms of eczema, and true prurigo. They are chronic, with little irritation, exudation, pain, or active signs. They are best treated locally by tar or allied preparations, internally by arsenic.

(3) The third group is that of Erythemata. Here the indication is to correct some internal disorder of which the eruption is the symptom.

VII. *The acne group.*—Acne, both in its pathology and ætiology, differs from other forms of dermatitis. The age of the patient and its distribution are sufficient for diagnosis. It is at once a superficial and a deep dermatitis and is often followed by scars. Its treatment consists entirely, or almost entirely, in local applications directed to the correction of the sebaceous affection. With acne may be classed Sycosis and Furunculus.

VIII. *Deep affections.*—When we have ascertained that the affection of the skin is deep, that is to say, that it goes below the papillary layer, the field for diagnosis is limited.

Excluding erysipelas, which is distinguished by its acute character and febrile symptoms ; excluding the pustular affections which affect the skin deeply and produce scars only at isolated points, such as acne, variola, and zona ; and excluding, thirdly, leprosy and other exotic diseases—we have to distinguish, in the great majority of cases which come before us in this country : first, traumatic and varicose ulcers ; secondly, gummata and syphilitic ulcers ; thirdly, lupus ; fourthly, rodent ulcer and carcinoma of the skin.

With regard to the first of these, we must not assume because a sore upon the skin is said to be the result of a blow or a kick, that it is purely traumatic, for syphilitic ulcers often arise in this way. Malignant ulcers are rare and usually obvious from the age of the patient, the pain they occasion, their tumid margins, and their blood-stained secretions. Moreover, they are, with few exceptions, confined to the neighbourhood of the orifices of the body, especially the lower lip, the urethra, the vulva, and the anus. Rodent ulcer, however, is very difficult to be sure of. Its locality, its slow and painless progress, and its belonging to the latter half of life, usually serve to distinguish it from lupus, and its being single, excessively chronic, and unaccompanied by nodes or other syphilitic lesions, are the best characters for diagnosis from a tertiary ulcer.

Between lupus and syphilis the difficulty of diagnosis is occasionally extreme. Lupus, however, is rarely more than single, syphilis is usually multiple; both are commonly free from pain and itching, but in syphilis the colour tends from red to rusty brown, in lupus from red to violet blue; the scars of syphilis are depressed and pigmented, those of lupus hypertrophic and white; the edges of a lupous ulcer are beset with nodules, those of syphilis are either thin and smooth or indurated by chronic inflammation; lupus is in the majority of cases a disease of the face, syphilitic ulcers are quite as frequently on the limbs or trunk; lupus is a disease of the skin alone, syphilis affects the subjacent tissues also.

END OF VOL. II.

INDEX OF SUBJECTS

The Roman numeral refers to the volume, the Arabic to the page.

A.

Abdomen in cerebral disease, i, 683
— in enteric fever, i, 174
Abortion in chorea, i, 744
— in relapsing fever, i, 156
— in syphilis, i, 331
— in typhus, i, 140
— in variola, i, 230
Abscess, *see* Brain, liver, spleen, kidney, mediastinum
Acarus scabiei, ii, 923
Acetonæmia in diabetes, ii, 719
Achorion, ii, 971
Achromatopsia, i, 536, 574, 825
Acne, ii, 942
— atrophica, ii, 947, *footnote*
— cheloid, ii, 956, 1016
— cornea, ii, 948
— frontalis, ii, 947, *footnote*
— varioliformis, ii, 947
— rosacea, ii, 979
Acrodynia, ii, 1012
Actinomycosis, i, 382
Acupuncture for cardiac dropsy, ii, 978
— for renal dropsy, ii, 655
Acute yellow atrophy of liver, ii, 531
Addison's disease, ii, 733
— anæmia, ii, 772
Adenoid growths in pharynx, ii, 310
Adenoma, i, 116
Adherent pericardium, i, 945
Adrenals, diseases of, ii, 733, 741
Ægophony, i, 91, 170
Ætiology, i, 6
Agraphia, i, 577
Ague, i, 338 *et seq.*, 587, 786
Alalia, i, 576
Albinism, ii, 1021
Albuminuria, ii, 595
— in cholera, i, 284
— in diphtheria, i, 273
— in enteric fever, i, 174
— in erysipelas, i, 255
— in glanders, i, 378
— in pemphigus, ii, 920
— in pneumonia, ii, 138
— in scarlatina, i, 211, 214
— in typhus, i, 138
— in variola, i, 236
— in yellow fever, i, 357
Albuminuria, physiological, ii, 599
Albuminuric retinitis, ii, 609
Alcohol in treatment of diphtheria, i, 275
— — of typhus, i, 150
— — of enteric fever, i, 202
— — of pneumonia, ii, 146
— — of phthisis, ii, 261
— in relation to cirrhosis, ii, 524
— — to gout, ii, 804
— — to paraplegia, i, 463
— — to atrophy of brain, i, 673
— — to cerebral hæmorrhage, i, 566
— — to general paralysis, i, 665, 673
— — to phthisis, ii, 237
Alcoholic coma, i, 593
— insanity, i, 862
Alcoholism, i, 883
— acute, i, 593, 885
— chronic, i, 883
— diagnosis from general paralysis, i, 670
Aleppo evil, ii, 1012
Alkalies in gout, ii, 808
— in rheumatism, ii, 822
Alkaline urine from fixed salts, ii, 565
— from ammonia, ii, 579
Allorrhythmia, i, 901
Alopecia, ii, 974
— areata, ii, 975
— febrilis, ii, 975
— leprosa, ii, 1011
— senilis, ii, 974
— syphilitica, i, 320; ii, 975
— from tinea tonsurans, ii, 963, 974
— universalis, ii, 976
— — congenita, ii, 977
Alphos, ii, 910, 1009
Amaurosis in hysteria, i, 825
— from tumor cerebri, i, 616
— from uræmia, ii, 618
Amblyopia, crossed, i, 574
Ambulatory enteric fever, i, 177
Ammoniacal fermentation in urine, ii, 579
Amnesia, i, 578
Amnesic aphasia, i, 582
"Amyloid" disease, ii, 630, *see* Lardaceous
Anæmia, ii, 747
— from Bilharzia, ii, 703
— cerebral, in epilepsy, i, 802
— Egyptian, ii, 453,
— essential, ii, 772

Anæmia, idiopathic, ii, 772
— lymphatic, ii, 761
— from sclerostomum, ii, 452
— of spinal cord, i, 453
— pernicious, ii, 772
— tropical, ii, 453
Anæmic murmurs, ii, 751
Anæsthesia, i, 396
— in chronic alcoholism, i, 883
— in cerebral lesions, i, 571, 573, 600
— in hemiparaplegia, i, 471
— in hemiplegia, i, 571
— in hysteria, i, 824
— in leprosy, i, 1011
— in paraplegia, i, 437
— in paralysis of the fifth nerve, i, 414
— in peripheral paralysis, i, 389
— in sciatica, i, 424
— in tabes, i, 532
— in writers' cramp, i, 724
Analgesia, i, 396
— in hysteria, i, 824
— in peripheral paralysis, i, 396
— in tabes, i, 532
Anarthria, i, 518, 519
— in bulbar paralysis, i, 518, 519
Anasarca, *see* Dropsy
— without albuminuria, ii, 606
— in chronic bronchitis, ii, 194
— in cirrhosis of kidneys, ii, 646
— in heart disease, i, 975
— renal, ii, 605, 627, 637
— in scarlatina, i, 215
Ancylostomum duodenale, ii, 452
Ancylosis in dengue, i, 365
— from rheumatism, ii, 812
— from gonorrhœal synovitis, ii, 838
— from scurvy, ii, 783
Aneurysm, i, 987
— abdominal, i, 995
— of aorta, ascending, i, 989
— — descending, i, 993
— — thoracic, i, 987
— of the aortic arch, i, 991
— cerebral, a result of embolism, i, 560
— of heart, i, 937, 997
— — acute, i, 953
— fusiform, i, 987
— intrathoracic, i, 987
— of pulmonary artery, ii, 217
Aneurysms of cerebral arteries, i, 564
Angina, ii, 305, 306, *see* Cynanche
— pectoris, i, 908
— — vaso-motoria, i, 912
Angioma pigmentosum atrophicum, ii, 1018
Angulus Ludovici in emphysema, ii, 113
Anidrosis, ii, 959
Ankle-clonus, i, 440
Anorexia, ii, 335
— nervosa vel hysterica, i, 835
Anosmia, i, 415, 618
— association with aphasia, i, 583
Anthracosis, ii, 241
Anthrax, i, 367
— in animals, i, 368
— diagnosis of, from erysipelas, i, 256
— gastro-intestinal, i, 372
Anthrax, myelitis in, i, 450
— pulmonary, i, 373
— of thoracic viscera, i, 373
Anticipation of ague fit, i, 341
Antipyretics, i, 203
Anuria from obstruction of ureter, ii, 670
Aorta undeveloped in anæmia, ii, 770
Aortic aneurysm, i, 990, *see* Aneurysm
Aphasia, i, 576
— amnesic, i, 582
— association with anosmia, i, 582
— — hemiplegia, i, 579
— expression by gestures in, i, 578
— — intelligence in, i, 582
— from meningitis, i, 686, 692, 694
— from migraine, i, 783
— localisation of lesion in, i, 579
— theories of, i, 581
Aphemia, i, 576
Aphonia in chronic catarrhal laryngitis, ii, 34
— in hysteria, i, 825, 840
— in paralysis of adductors, ii, 11
— in tubercular disease of larynx, ii, 39
Aphtha epizootica, i, 380
Aphthæ, ii, 300
Aphthous ulcers, ii, 299
Apnœa, i, 11
— cardiac, i, 973
Apneumatosis, ii, 101
Apoplectic attacks in insular sclerosis, i, 550
— — in Menière's disease, i, 815
— — in general paralysis, i, 667
— — from cerebral congestion, i, 596, 796
Apoplectic cicatrix, i, 563
Apoplexy, i, 553, 566, 584
— in Bright's disease, ii, 617, 645
— ingravescent, i, 589, 603
— pulmonary, i, 956
— simulation by malarial poisoning, i, 343
— meningeal, i, 562
— from organic disease of brain, i, 598
— simple, i, 597
— from syphilitic arteritis, i, 600
Arachnitis, i, 674
Area, ii, 975
Argyll-Robertson pupil, i, 536
Arrhythmia, i, 899
Arsenic in ague, i, 355
— in chorea, i, 756
— in eczema, ii, 891
— in Hodgkin's disease, ii, 767
— in idiopathic anæmia, ii, 778
— in lichen ruber, ii, 903
— in neuralgia, i, 427
— in osteoarthritis, ii, 847
— in pemphigus, ii, 922
— in prurigo, ii, 906
— in psoriasis, ii, 916
— in purpura, ii, 791
— subcutaneous injection of, in paralysis agitans, i, 734
— — in spasmodic torticollis, i, 719
Arsenical eruption, ii, 941
Arteries, cerebral, atheroma of, i, 564
— — distribution of, i, 557
— — obstruction of, i, 554

Arteries, cerebral, syphilitic disease of, i, 556
— in purpura, ii, 790
— in Bright's disease, ii, 613
Arterio-capillary fibrosis, ii, 613, 616
"Artery of cerebral hæmorrhage," i, 561
Arthritic affection in dengue, i, 363
— — in fœtid bronchitis, ii, 120
— — in locomotor ataxy, i, 536; ii, 848
— — in lesions of nerve-trunks, i, 398
— — in secondary syphilis, i, 313
— — in tuberculosis, ii, 266
Arthritis atrophica, i, 536; ii, 848
— deformans, ii, 840
— gonorrhœal, ii, 835
— in hæmophilia, ii, 794
— tabid, i, 536; ii, 848
— tubercular, i, 95; ii, 266
Arthropathie ataxique, i, 536
Ascaris lumbricoides, ii, 410, 446
— mystax, ii, 450
Ascites, ii, 480
— from atrophy of liver, ii, 529
— from cancer of biliary passages, ii, 541
— from cancer of liver, ii, 530, 540
— from cirrhosis of liver, ii, 528
— from perihepatitis, ii, 529
— from peritonitis, chronic, ii, 474
— — malignant, ii, 478
— — tubercular, ii, 476
— from pylephlebitis adhesiva, ii, 530
— from syphilitic hepatitis, ii, 529
Asphyxia, i, 11
Aspiration, ii, 521, 555
— in pleuritic effusion, ii, 183
Asteatosis, ii, 949, *see* Xerodermia
Asthenia, i, 18
Asthma, ii, 292
— from chronic bronchitis, ii, 294
— from emphysema of lungs, ii, 111, 294
— caused by hay, ii, 283, 296
Atavism in gout, ii, 803
— in phthisis, ii, 232
Ataxia of muscles, i, 537
Atelectasis, ii, 101
Atheroma of cerebral arteries, i, 564
— of pulmonary artery, ii, 105
Athetosis, i, 576
Atrophic arthritis, ii, 848
Atrophodermia pigmentosa maligna, ii, 1018
Atrophy of the brain, i, 663, 671
— of liver, ii, 529, 531
— of muscles—
— — in acute atrophic paralysis, i, 498
— — in chronic atrophic paralysis, i, 503
— — progressive, i, 507
— of one kidney, ii, 672
Atropine, subcutaneous injection of, ii, 260
Auditory affections, *see* Ears, affections of
— — in paroxysmal vertigo, i, 813
— — in simple meningitis, i, 704
— vertigo, i, 814
Aura, epileptic, i, 794
Auscultation, ii, 81
— in diagnosis of stenosis of trachea or bronchi, ii, 66
— of the breathing, ii, 83
Auscultation of the voice, ii, 89
— of stricture of œsophagus, ii, 315

B.

Bacilli in genere, i, 24
Bacilluria aseptica, ii, 582
Bacillus anthracis, i, 368, 369, 375
— of cholera, i, 291
— of diphtheria, i, 262
— of lepra, ii, 742
— of lupus, ii, 1002
— of malaria, i, 359
— of phthisis, ii, 214, 261
— subtilis, i, 368
— tuberculosis, i, 84, 86; ii, 214
Bacteria in genere, i, 23
— in bromidrosis, ii, 960
— in diphtheria, i, 262
— in erysipelas, i, 251
— in pneumonia, ii, 142
— in purpura, ii, 791
— in rhinoscleroma, ii, 1008
— in urine, ii, 580
Bacterium decalvans, ii, 976
— lactis, i, 23 *et seq.*
— termo, i, 262
Barrel-shaped chest, ii, 113
Basedow's disease, i, 1009
Baths, cold, in enteric fever, i, 204
— — in rheumatic hyperpyrexia, ii, 832
— of Droitwich, in spinal meningitis, i, 495
— of Gastein, in hemiplegia, i, 604
— — in hypochondriasis, i, 845
— — in myelitis, i, 469
— of Nauheim, in spinal meningitis, i, 495
— — in locomotor ataxy, i, 544
— of Pfeffers, in hemiplegia, i, 604
— — in myelitis, i, 471
— of Ragatz, in myelitis, i, 471
— of Rehme, in spinal meningitis, i, 495
— — in locomotor ataxy, i, 544
— of Schlangenbad, in myelitis, i, 471
— of Strathpeffer, in pityriasis rubra, ii, 909
— of Töplitz, in myelitis, i, 471
— of Vals, in gout, ii, 809
— of Vichy, in gout, ii, 809
— of Wiesbaden, in myelitis, i, 471
— warm, in Bright's disease, ii, 654
— — in croup, ii, 31
"Beading" of ribs in rickets, ii, 852
Bedsore in paraplegia, i, 444
— in status epilepticus, i, 798
— in enteric fever, i, 183
— electrolytic treatment of, i, 469
Belladonna in aortic disease, i, 978
— in epilepsy, i, 811
— in migraine, 792
— in pertussis, ii, 291
— rash, ii, 940
Bellocq's cannula, ii, 283
Bellows murmur, i, 957
Beri-beri, i, 432
Bile acids, tests for, ii, 495
Bilharzia hæmatobia, ii, 702
Biliary colic, ii, 500

Bilious disorders, ii, 487
Bilirubin, test for, ii, 494
Bird's-nest cells, i, 118
Bismuth internally in erythema, ii, 938
— in diarrhœa, ii, 384
— in gastritis, ii, 329
Black vomit in yellow fever, i, 356
Bladder, hypertrophied, in diabetes, ii, 720
— inflammation of, ii, 681
— in paraplegia, i, 441
Bladder-worm, ii, 437
Bleb, *see* Bulla
Bleeders, ii, 792
Blistering for ringworm, ii, 969
— for rheumatism, ii, 825
Blister-test of gout, ii, 801
Blood, in Bright's disease, ii, 607
— in cholera, i, 287
— estimate of hæmoglobin, ii, 749
— — corpuscles, ii, 748
— in gout, ii, 800
— in Hodgkin's disease, ii, 763
— in leuchæmia, ii, 755
— in purpura, ii, 789
Blood-letting in apoplexy, i, 603
— — in aneurysm, i, 993, 994
— — in cardiac distension, i, 978
— — in epilepsy, i, 810
— — in meningitis, i, 706
— — in pneumonia, ii, 144, 146
Blue line of gums, i, 506; ii, 372
Boils, ii, 956
— in diabetes, ii, 717
— in smallpox, i, 238
Bones in congenital syphilis, i, 322, 336
— in mollities ossium, ii, 863
— in rickets, ii, 851
— in sarcomatosis, i, 113
Boroglycerine in ringworm, ii, 968
Bothriocephalus latus, ii, 442
Bovine heart, i, 956
— tuberculosis, i, 89; ii, 201
Brain, diseases of, i, 553–605
— anæmia of, in epilepsy, i, 802
— arteries, distribution of, i, 558
— — obstruction of, i, 554, 559
— atrophy of, i, 663, 671
— cortical centres of, i, 622
— cysts of, i, 608
— diffused organic lesions of, i, 645
— glioma of, i, 609
— gumma of, i, 607
— hydatid of, ii, 557
— hypertrophy of, i, 647
— local organic affections of, i, 605
— softening of, i, 559, 609, 634
— — in meningitis, i, 675, 679
— tubercle of, i, 605
— tumours of, i, 605
— vacuolation of, i, 669, 672
Brass-founders' ague, i, 353
Breast-pang, i, 908
Breath, odour, in diabetes, ii, 716
Breathing, bronchial, ii, 84
— cavernous, ii, 85
— cogged-wheel, ii, 220
— puerile, ii, 84
Breathing, tubular, ii, 84
Bright's disease, ii, 591 *et seq.*
— — forms of, ii, 591, 652
— — consecutive, ii, 648
Bromide rash, ii, 939
Bromidrosis, ii, 960
Bromism, i, 810
Bronchial breathing, ii, 84
— polypi, ii, 124
Bronchiectasis, ii, 115, 155
— cylindrical, ii, 116
— in cirrhosis of lung, ii, 155
— in phthisis, ii, 207
— sacculated, i, 116
Bronchitic asthma, ii, 294
Bronchitis, ii, 94
— acute, ii, 99
— capillary, ii, 99
— chronic, ii, 104
— typhoid, i, 181
— fœtid, ii, 118
— plastic, ii, 124
Bronchophony, ii, 89
Broncho-pneumonia, ii, 148
— cerebral symptoms in, ii, 150
— association with pleurisy, ii, 149
Bronchorrhœa serosa, ii, 105
Brow-ague, i, 344, 787
Brown degeneration of heart, i, 941
— induration of lung, i, 956
Brucia, poisoning by, i, 763
Bruit d'airain, ii, 195
— de diable, i, 958; ii, 751
— de pot fêlé, ii, 80
— — in chronic hydrocephalus, i, 654
— — in phthisis, ii, 222
— — in pneumonia, ii, 134
— over pulmonary artery in phthisis, ii, 220
— de râpe, i, 958
— de rappel, i, 964
— de scie, i, 958
— de souffle, i, 957
— d'oboe, i, 958
Bruits, cardiac, i, 957
— functional, ii, 751
Bubo in syphilis, i, 313
— in typhus, i, 140
— in plague, i, 303
Bucnemia, ii, 991, *see* Elephantiasis
Bulbar paralysis, i, 517
Bulla, ii, 869, 917, 919, 924, 1025
Burmese ringworm, ii, 970
Butter-kidney, ii, 634

C.

Cachexia, cancerous, i, 98
— malarial, i, 345
Caffein in cardiac dieease, i, 978
Calabar bean in spastic paraplegia, i, 492
— in tetanus, i, 766
Calcareous degeneration of heart, i, 941
Calcic carbonate calculus, ii, 660
— phosphate in urine, ii, 566
— sulphate in urine, ii, 579
Calculi, biliary, ii, 500

Calculi, urinary, ii, 657
— — of triple phosphate, ii, 660
— — of calcic carbonate, ii, 660
— — of calcic oxalate, ii, 658
— — of calcic phosphate, ii, 660
— — of cystin, ii, 658
— — of indigo, ii, 659
— — fusible, ii, 660
— — of urate of soda, ii, 658
— — of uric acid, ii, 658
— — urostealith, ii, 659
— — of xanthine, ii, 659
— — treatment of, ii, 685
Calculous pyelitis, ii, 682
Callosities, ii, 982
Cancer, i, 97, *see also* Carcinoma
Cancer-bodies, i, 118
Cancer-cell, i, 97
Cancer-juice, i, 118
Cancer of the biliary passages, ii, 541
— of brain, i, 608
— of bowel, ii, 426
— of gall-bladder, ii, 539, 541
— of kidneys, ii, 695
— of liver, ii, 530, 538
— of lung, i, 1005
— of œsophagus, ii, 314
— of peritoneum, ii, 479
— of the skin, ii, 1019
— of the stomach, ii, 354
Canities, ii, 1022
Cannabis indica, rash from, ii, 939
Capillary pulse, i, 967
Carbolic acid inhalation, in pertussis, ii, 291
— oil in ringworm, ii, 967
Carbuncle, ii, 959
— in diabetes, ii, 717
— in plague, i, 303
— facial, diagnosis from anthrax, i, 371
Carcinoma, i, 97, 116, *see also* Cancer
— criteria of, i, 99
— distribution of, i, 100, *footnote*, 101
— colloid, i, 121
— columnar, i, 119
— cylinder, i, 119
— epithelial, i, 118
— keratoid, i, 118
— origin of, i, 120
— scirrhous, i, 117
Carcinomatin, i, 98
Cardiac disease, i, 897 *et seq.*
— — from rheumatism, ii, 813
— dulness, i, 923
— hypertrophy, i, 918
— — in Bright's disease, ii, 611
— symptoms of chronic m. Brightii, ii, 646
Cardialgia, i, 903
Cardio-vascular changes in morbus Brightii, ii, 611
Caries of spine, i, 478
— — in Addison's disease, ii, 739
"Carswell's grapes" in phthisis, ii, 205
Caseation of adrenals, ii, 734
— of carcinoma, i, 121
— in caries of spine, i, 480
— of inflammatory products, i, 65
— of mesenteric glands in enterica, i, 169
Caseation of lungs in phthisis, ii, 201
— of sarcomata, i, 108
— of tubercle, i, 80, 84
— a cause of tuberculosis, i, 88
Casts, bronchial, ii, 125
— gastric, ii, 320
— intestinal, ii, 405
— renal, ii, 603
— — blood, ii, 603, 628
— — epithelial, ii, 603, 629, 637
— — false, ii, 604
— — fatty, ii, 603, 629, 637
— — granular, ii, 603, 629, 637, 644
— — hyaline, ii, 603, 628, 629, 637, 644
— — lardaceous, ii, 604
Catalepsy, i, 830
— epileptic, i, 793
— hysterical, i, 830
Cataract, diabetic, ii, 717
Catarrh, nasal, ii, 271
— relation to phthisis, ii, 246
Catarrhal inflammation, i, 61
Catarrhe sec, ii, 105
Causalgia, i, 398, 401
Cauterisation in hydrophobia, i, 775
— in spasmodic torticollis, i, 720
Cavernous breathing, ii, 85
Cells, origin of, i, 52
Centric cough, ii, 74
Cephalalgia from cerebral tumour, i, 612
— in lithæmia, ii, 489
— in migraine, i, 779
— in meningitis, i, 681, 682, 691, 702
— in syphilis, i, 313
— in uræmia, ii, 618
Cerebellum, tumours of, i, 619
Cerebral abscess, i, 635
— aneurysm, i, 560
— embolism, i, 554, 560, 599
— hæmorrhage, i, 561, 600
— — miliary aneurysms in, i, 564
— — in renal disease, i, 562; ii, 645
— — seats of, i, 562
— — traumatic, i, 638
— — from tumour, i, 598
— meninges, new growths in, i, 607
— — inflammation of, i, 674 *et seq.*
— softening, i, 560, 610, 634
— thrombosis, i, 556
— symptoms in broncho-pneumonia, ii, 150
— — in cirrhosis of liver, ii, 527
— — in diabetes, ii, 719
— — in enteric fever, i, 174
— — in rheumatism, ii, 820
— — from salicylates, ii, 829
— — in typhus, i, 134
— — in yellow atrophy of liver, ii, 533
Cerebritis, *see* Encephalitis
Cerebro-spinal fever, i, 690
— — epidemic, i, 691
— — simple, i, 698
— fluid in chronic hydrocephalus, i, 650
— — in tubercular meningitis, i, 675
— — normal characters of, i, 649
Cestoidea, ii, 435
Chalicosis, ii, 243
Chancre, i, 309

Chancre, infecting and mixed, i, 310
— non-infecting, i, 309
— soft, i, 310
Chaps or fissures, ii, 869
Charbon, i, 370
Charcot's cirrhosis of liver, ii, 528
— crystals in asthma, ii, 297
— joint-disease, i, 536; ii, 848
Chaulmoogra oil in leprosy, ii, 1012
Cheloid, ii, 1014
— relation to scars, ii, 1015
— traumatic, ii, 1016
Cheyne-Stokes respiration in apoplexy, i, 586
— — in cardiac disease, i, 974
— — in meningitis, i, 684
Chilblain, ii, 930
Chiro-pompholyx, ii, 921
Chloral hydrate, in delirium tremens, i, 888
— — in acute delirious mania, i, 850
— — in mania, i, 853
— — in chorea, i, 757
— — in general paralysis, i, 671
— — in tetanus, i, 766
Chloasma, ii, 972, 1022, 1023
Chlorosis, ii, 769
— in boys, ii, 770
Chloroform in angina pectoris, i, 916
— in biliary colic, ii, 511
Choked optic disc, i, 590, 614
Cholesteatoma, i, 608; ii, 950
Cholestearæmia, ii, 528
Cholera, i, 280
— nostras, i, 298
Cholera-typhoid, i, 285
Choleraic diarrhœa, i, 286
Cholerine, i, 286
Chorea, i, 714, 740
— gravis, i, 743
— post-hemiplegic, i, 576
— of dogs, i, 747
— relation to rheumatism, i, 752
Choroid, tubercle of, i, 687; ii, 267
Chromidrosis, ii, 960
Chromogens in urine, ii, 575
Chrysophanic acid in psoriasis, ii, 915
— in ringworm, ii, 967
Chyluria, ii, 699, 992
Cicatrices and cheloid, ii, 1015
Circulation of the blood in cirrhosis, ii, 526
Cirrhosis of kidneys, ii, 594, 639
— of liver, ii, 522
— — in ague, i, 346
— — atrophic form, ii, 524
— — hypertrophic form, ii, 528
— — a cause of jaundice, ii, 499
— — syphilitic, ii, 525, 529
— of lung, ii, 152
— of stomach, ii, 366
Classification of diseases, i, 126; ii, 385
— — of the nervous system, i, 388, 433, 713
— — of the kidneys, ii, 592
— — of the skin, ii, 865, 1030
Clavus, ii, 982
— hystericus, i, 780
Clergyman's sore-throat, ii, 35
Climatic influence in ague, i, 348
— — in cholera, i, 298
Climatic influence in enterica, i, 199
Clinical thermometer, i, 37
Clonus, i, 440
"Clownisme," i, 895, *footnote*
Clubbing of fingers, ii, 155
Coagula in urine, ii, 664
Codeia in treatment of diabetes, ii, 732
Cod-liver oil in phthisis, ii, 260
Coffee-ground vomit, ii, 359
Cogged-wheel breathing, ii, 220
Colchicum in gout, ii, 807
— in zona, ii, 1029
Cold baths in treatment of fever, i, 204
— — of gout, ii, 808
— — of heat-stroke, i, 894
— — of meningitis, i, 705
— — of pneumonia, ii, 144
— — of rheumatism, ii, 832
Colic, biliary, ii, 500
— intestinal, ii, 368
— plumbic, ii, 370
— renal, ii, 667
Colitis, acute catarrhal, ii, 404
— dysenteric, ii, 388
— membranous, ii, 405
— ulcerative, ii, 405
Collapse, i, 16
— in biliary colic, ii, 501, 502
— in cholera, i, 281
— in hysteria, i, 835
— in acute obstruction of intestine, ii, 429
— in peritonitis, ii, 464
— in pneumonia, ii, 139
— of pulmonary tissue, ii, 101
Colloid cancer of stomach, ii, 358
— transformation of cancer, i, 121
Colour blindness in hemiplegia, i, 574
— — in hysteria, i, 825
— — in locomotor ataxy, i, 536
Coma, its analysis, i, 9
— in ague, i, 343
— in alcoholic poisoning, i, 593, 887
— in apoplexy, 585, 589
— from cerebral abscess, i, 642
— — tumour, i, 613
— diabetic, ii, 719
— in enteric fever, 1, 178
— epileptic, i, 597
— in general paralysis of the insane, i, 667
— from heat-stroke, i, 892
— in meningitis, i, 684, 702
— from narcotic poisoning, i, 593
— pyæmic, i, 595
— traumatic, i, 591
— uræmic, i, 595; ii, 617
— — in delirium tremens, i, 890
— in variola, i, 236
Coma-vigil, in typhus, i, 139
— in cerebral disease, i, 613
— in idiopathic anæmia, ii, 778
Comedo, ii, 868, 943, 948.
Compound granule-masses in acute spinal sclerosis, i, 501
— in cerebral softening, i, 559
— in chronic spinal sclerosis, i, 504
— in disseminated sclerosis, i, 548
— in myelitis, i, 446

Compressed air in bronchitis, ii, 123
— — in phthisis, ii, 261
Compression-paraplegia, i, 478
Concretions, gastric, ii, 367
— intestinal, ii, 416
— pulmonary, ii, 219
Concussion of spinal cord, i, 459
Condyloma, ii, 985
— flat, i, 317
Congenital syphilis, i, 329–337
— disease of heart, i, 979
Conjugate deviation of eyes, i, 570
Conjunctiva in scurvy, ii, 782
Conjunctival diphtheria, i, 266
Consecutive Bright's disease, ii, 648
Consonance in respiratory sounds, ii, 88
Constipation, ii, 375
— from acute intestinal obstruction, ii, 429
— from chronic obstruction, ii, 423
— from peritonitis, ii, 464
— from anæmia, ii, 770
Contagia, destruction of, i, 34
— persistence of, i, 33
— criteria of, i, 22, *footnote*
Contagion, i, 20 *et seq.*
— of ague, i, 351
— of anthrax, i, 369
— of cholera, i, 291
— of dengue, i, 365
— of diphtheria, i, 264
— of enteric fever, i, 188
— of epidemic meningitis, i, 696
— of erysipelas, i, 250
— of glanders, i, 376, 379
— of hydrophobia, i, 770
— of influenza, ii, 276
— of measles, i, 217
— of pertussis, ii, 289
— of phthisis, ii, 229
— of plague, i, 304
— of relapsing fever, i, 159
— of rötheln, i, 224
— of scarlatina, i, 209
— of tubercle, i, 87
— of typhus, i, 141
— of varicella, i, 247
— of yellow fever, i, 358, 360
Contagious diseases, i, 21, 22
— pus, i, 26
Contagium vivum, i, 23 *et seq.*
Contraction of small intestine, ii, 417
— of large intestine, ii, 418
Contracture of muscles in hysteria, i, 826,
— — after facial paralysis, i, 406
Convallaria in cardiac disease, i, 983
Convulsions in acute alcoholism, i, 887
— in chronic alcoholism, i, 883
— in biliary colic, ii, 501
— from cerebral abscess, i, 641
— from cerebral embolism, i, 600
— from cerebral lesions generally, i, 624
— from disseminated sclerosis, i, 552
— epileptic, i, 795
— epileptiform, i, 792, 806
— in general paralysis, i, 667
— in hysteria, i, 829, 834
— from hydrocephalus, i, 656
Convulsions, infantile, i, 806
— from meningeal hæmorrhage, i, 590
— from meningitis, i, 682, 685, 702
— puerperal, i, 806
— in renal colic, ii, 667
— in scarlatina, i, 213
— in uræmia, ii, 616
Copper test for sugar in urine, ii, 708
Cord, *see* Spinal cord
Corneal ulcers in epidemic meningitis, i, 693
— — in smallpox, i, 238
— — from supra-orbital zona, ii, 1062
Corns, ii, 982
Cornu cutaneum, ii, 988
Corpora amylacea in myelitis, i, 448
— — in disseminated sclerosis, i, 548
Corpuscles of Gluge, i, 446, 501, 548, 559
Corpuscular richness of the blood, ii, 750
Corymbose smallpox, i, 237
Coryza, ii, 271
— in measles, i, 218
— in rötheln, i, 225
Cough, ii, 71
— centric, ii, 74
— from ear, ii, 72
— in bronchitis, ii, 94
— in capillary bronchitis, ii, 99
— from hepatic abscess, ii, 518
— after paracentesis thoracis, ii, 184
— in pertussis, ii, 286
— in pleurisy, ii, 172
— reflex, ii, 72
— from stomach, ii, 73
— from tooth, ii, 73
— from throat, ii, 72
Couperose, ii, 979, *see* Gutta rosea
Cracked-pot sound, i, 654; ii, 80, 134, 222
Craniotabes, ii, 854, 859
— in congenital syphilis, i, 336
— in rickets, ii, 854
— its relation to syphilis and rickets, ii, 859
Crepitation, ii, 88
— pneumonic, ii, 133
Cretinism, endemic, i, 876
— sporadic, i, 878
Cretinoid condition in adults, i, 881
Crisis in intermittent form of ague, i, 341
— — remittent form, i, 345
— in enteric fever, i, 175
— in erysipelas, i, 255
— in measles, i, 218
— in acute pneumonia, ii, 140
— in pyrexia generally, i, 46
— in relapsing fever, i, 156
— in scarlatina, i, 211
— in typhus, i, 138
Criteria of pathogenetic organisms, i, 22
Crossed amblyopia, i, 574
Croup, ii, 18
— relation to diphtheria, i, 260; ii, 18, 24
— spasmodic or spurious, ii, 22
— tracheotomy in, ii, 31
Crust or scab, ii, 869
Cry, the hydrocephalic, i, 682
Cubebs-rash, ii, 938

Cultivation of microzymes, i, 25, 28
Cuniculus, ii, 924
Curare in hydrophobia, i, 775
— in tetanus, i, 766
Cutis anserina in ague, i, 340
Cyanosis from bronchitis, ii, 95
— from cardiac disease, i, 976
— from emphysema, ii, 112
Cyclic albuminuria, ii, 600
Cylindroma, i, 119
Cynanche, ii, 306
— contagiosa, i, 259
— parotidea, ii, 302
— tonsillaris, ii, 306
— trachealis, ii, 18
Cyrtometer, ii, 93
Cystic disease of kidneys, ii, 650
Cysticercus cellulosæ, ii, 439
— of brain, i, 609
— of subcutaneous tissue, ii, 440
Cystine in urine, ii, 578
— calculus, ii, 658
Cystitis in paraplegia, i, 444
— with ammoniacal urine, ii, 582
Cysts of brain, i, 608
— in granular kidney, ii, 643, 650
— in pancreas, ii, 743

D.

Dandy fever or dengue, i, 363
Deaf-mutism from meningitis, i, 693
Deafness in paroxysmal vertigo, i, 813
— in typhus, i, 135
— from salicylates, ii, 829
Death by asphyxia, i, 11
— by asthenia, i, 18
— by collapse, i, 16
— by coma, i, 9
— diagnosis of, i, 19
— by hæmorrhage, i, 16
— modes of, i, 8
— by paralysis of respiratory centre, i, 10
— by syncope, i, 14
Decubitus acutus in hemiplegia, i, 575
— in paraplegia, i, 444
Deep inflammation of the skin, ii, 978
Deformities from atrophic paralysis, i, 499
— from gout, ii, 799
— from osteo-arthritis, ii, 843
— from mollities ossium, ii, 862
— from rickets, ii, 851 *et seq.*
Délire des grandeurs with disseminated sclerosis, i, 550
— in general paralysis of the insane, i, 665
Delirium in epilepsy, i, 798
— in enteric fever, i, 174, 175
— in meningitis, i, 692, 702
— in pneumonia, ii, 138
— in rheumatism, ii, 812, 820
— in smallpox, i, 239
— in typhus, i, 133, 134
— tremens, i, 885
Delusional insanity, i, 857
Delusions in acute delirious mania, i, 850
— in chronic mania, i, 851
Delusions in general paralysis, i, 665
— in melancholia, i, 855
Dementia, acute, i, 856
— from chronic alcoholism, i, 883
— in epilepsy, i, 798
— in general paralysis, i, 667
— paralytica, i, 663
Demodex folliculorum, ii, 943
Dengue, i, 363
Dentition in rickets, ii, 853
Depth of cutaneous diseases, ii, 870
Dermatitis, superficial, ii, 872
— deep, ii, 978
— papillomatosa capilitii, ii, 957
Dermatology, historical sketch, ii, 866
Dermoid cyst of mediastinum, i, 1003
Desquamation in dengue, i, 284
— in cholera, i, 365
— in eczema, ii, 875
— in erysipelas, i, 255
— in measles, i, 219
— in psoriasis, ii, 910
— in pityriasis rubra, ii, 908
— in rubeola, i, 225
— in scarlatina, i, 212
Diabetes insipidus, ii, 561
— mellitus, ii, 708
— — phthisis in, ii, 238, 718
— — theories of, ii, 723
Diabetic coma, ii, 719
Diapedesis, i, 53
Diaphoretics in Bright's disease, ii, 655
Diaphragm, its degeneration in bronchitis, ii, 105
Diarrhœa, ii, 378
— choleraica, i, 286
— in enteric fever, i, 173, 206
— from lardaceous disease of intestine, ii, 382
— in phthisis, ii, 213
— in smallpox, i, 236
— a cause of tetany, i, 738
— in uræmia, ii, 619
Diathesis, ii, 866, 880, 913
— gouty or arthritic, ii, 802, 880
— lithæmic, ii, 488
— rheumatic, ii, 817
— scrofulous, ii, 232, 233
— tubercular, ii, 232
Dicrotism, i, 971
Diet in diabetes, ii, 728
— in convalescence from enteric fever, i, 206
— — from rheumatism, ii, 834
— in dyspepsia, ii, 330
— during infancy, ii, 859
— in scurvy, ii, 786
Digitalis, in acute alcoholism, i, 890
— in Bright's disease, ii, 656
— in valvular disease of the heart, i, 976
Digitorum nodi, ii, 842
Dilatation of heart, i, 918
— of stomach, ii, 323, 362
Diphtheria, i, 259
— of air-passages, i, 264, 271, 277
— sine diphtheriâ, i, 261
— a cause of locomotor ataxy, i, 542
— in course of measles, i, 221

Diphtheria, of nasal fossæ, i, 270, 277
— laryngeal, i, 270, 277
— and croup, i, 264, 266; ii, 18
— and diphtheritic inflammation, i, 260
— traumatic, i, 266; ii, 19
Diphtheritic dysentery, ii, 390
— inflammation, i, 260, 262
— — of skin in smallpox, i, 232
— membrane of pharynx in glanders, i, 378
— — of larynx in measles, i, 221
— — of pharynx in scarlatina, i, 214
— — of pharynx in smallpox, i, 237
Diplopia in locomotor ataxy, i, 535
Direction of cardiac murmurs, i, 962
Disease, meaning of the term, i, 1
— contagious, i, 21
— epizootic, i, 367
— functional, i, 2
— infective, i, 22
— miasmatic, i, 21
— miasmatic-contagious, i, 21
— organic, i, 2
— specific, i, 20
— zymotic, i, 22
Diseases, i, 1 *et seq.*
— of the adrenals, ii, 733–742
— of the alimentary system, ii, 299–462
— of the blood, ii, 746–796
— of the blood-vessels, i, 987–1014
— of the bones, ii, 850–864
— of the brain, i, 553–712
— of the heart, i, 897–986
— — congenital, i, 979
— — in children, i, 982
— of the intestines, ii, 368–462
— of the joints, ii, 797–849
— of the kidneys, ii, 559–707
— of the larynx and trachea, ii, 1–68
— of the liver, ii, 487–558
— of the lungs, ii, 69–298
— of the mouth and throat, ii, 299–317
— of the nerve-trunks, i, 389–432
— of the nervous system, i, 385–896
— of the œsophagus, ii, 312–317
— of the peritoneum, ii, 463–486
— of the respiratory system, ii, 1–298
— of the skin, ii, 865–1034
— of the spinal cord, i, 433–552
— of the spleen, ii, 743–745
— of the stomach, ii, 318–367
Disinfectants, comparative value of, i, 35
Dislocations in epilepsy, i, 798
Disseminated sclerosis, i, 547
Distension of stomach, ii, 362
Distoma hæmatobium or bilharzia, ii, 702
Dochmius duodenalis, ii, 452
Double quartan ague, i, 341
— tertian ague, i, 341
Drink in diabetes insipidus, ii, 562
— in diabetes mellitus, ii, 730
Dropsy in Bright's disease, ii, 605
— from chronic bronchitis, ii, 104
— from cirrhosis of kidneys, ii, 646
— in heart disease, i, 975
— from lardaceous nephritis, ii, 637
— from parenchymatous nephritis, ii, 627
— in scarlatina, i, 215
Dumb-bell crystals of uric acid, ii, 570
— — of oxalate of lime, ii, 576
Dura mater, hæmatoma of, i, 668, 709
— inflammation, i, 674
— thrombosis of sinuses, i, 699, 708
Dust-inhalation, producing asthma, ii, 295
— — producing phthisis, ii, 239
Dysæsthesia in hysteria, i, 824
— in paraplegia, i, 485
Dyscrasia, ii, 866
Dysentery, ii, 388
— catarrhal form, ii, 388
— diphtheritic form, ii, 390
— endemic form, ii, 392
— sporadic form, ii, 392
Dysidrosis, ii, 961
Dyspepsia, acute, ii, 318
— bilious, ii, 487
— chronic, ii, 325
Dysphagia from aneurysm of aorta, i, 992
— from bulbar paralysis, i, 518
— in hydrophobia, i, 770
— in hysteria, i, 825
— from laryngeal carcinoma, ii, 57
— from œdematous laryngitis, ii, 48
— from tubercular laryngitis, ii, 39
— lusoria, ii, 313
— from stricture of œsophagus, ii, 315
Dyspnœa, ii, 69
— from anæmia, ii, 752
— in bronchitis, ii, 95, 99
— cardiac, i, 973
— in diabetic coma, ii, 719
— in miliary tuberculosis, ii, 264
— in phthisis, ii, 225
— in pleurisy, ii, 171
— in pneumonia, ii, 136
— laryngeal, ii, 65
— nervous, ii, 70
— from paralysis of diaphragm, ii, 70
— from tracheo-bronchial stenosis, ii, 66
— from uræmia, ii, 619

E.

Ear-cough, ii, 72
Ears, affections of, *see* Otitis and Otorrhœa
— — in gout, ii, 800
— — in measles, i, 221
— — in epidemic meningitis, i, 693
— — in meningitis, i, 704
— — in paroxysmal vertigo, i, 813
— — in scarlatina, i, 214
Eburnation of cartilages, ii, 842
Ecchondroses, ii, 842
Echinococcus, ii, 437, 546, *see* also Hydatid
Echinorhynchus, ii, 462
Eclampsia, i, 792
— hysterical, i, 829
— infantile, i, 806
— puerperal, i, 806
— uræmic, ii, 617
Ecstasy, i, 831
Ecthyma, ii, 957
Eczema, ii, 872
— diagnosis of, ii, 881

Eczema flavum, ii, 898
— and gout, ii, 880
— histology of, ii, 875
— local varieties of, ii, 876
— marginatum, ii, 970, *see* Tinea marginata
— of mucous membranes, ii, 879
— prognosis of, ii, 882
— treatment of, dietetic, ii, 887
— — internal, ii, 888
— — local, ii, 884
— — preventive, ii, 882
Electricity (Faradic, Galvanic, and static) as used in the treatment—
— of bedsore, i, 469
— of bulbar paralysis, i, 523
— of exophthalmic goitre, i, 1014
— of hemiplegia, i, 604
— of hysteria, i, 840
— of laryngismus stridulus, ii, 17
— of locomotor ataxy, i, 544
— of mercurialism, i, 896
— of spinal meningitis, i, 495
— of neuralgia, i, 427
— of osteo-arthritis, ii, 847
— of paralysis agitans, i, 734
— of acute atrophic paralysis, i, 502
— of chronic atrophic paralysis, i, 505
— of facial paralysis, i, 406
— of general paralysis of the insane, i, 671
— of laryngeal paralysis, ii, 10, 12
— of peripheral paralysis, i, 400
— of pseudo-hypertrophic paralysis, i, 529
— of progressive muscular atrophy, i, 516
— of spasmodic torticollis, i, 719
— of spastic paraplegia, i, 490
— of writers' cramp, i, 727
Electrical reactions, i, 393
— in catalepsy, i, 830
— in chorea, i, 741
— in hemiplegia, i, 573
— in lesions of nerve-trunks, i, 395, 397
— in locomotor ataxy, i, 534
— in paralysis—
— — acute atrophic, i, 498
— — agitans, i, 730
— — bulbar, i, 521
— — chronic diffused atrophic, i, 503
— — diaphragmatic, ii, 71
— — diphtheritic, i, 279
— — facial, i, 402
— — peripheral, i, 395
— — plumbic, i, 506
— — pseudo-hypertrophic, i, 527
— in progressive muscular atrophy, i, 511
— in paraplegia, i, 441
— — hysterical, i, 459
— — spastic, i, 489
— in sciatica, i, 424
— in tetany, i, 737
— in writers' cramp, i, 726
Electrolysis in hydatid disease, ii, 555
— — for bedsore, i, 469
Elementary lesions of the skin, ii, 867
Elephantiasis, ii, 991, 1009
— Græcorum, ii, 1009, *see* Leprosy
— neuromatosa, ii, 1017
Elephantiasis, telangiectodes, ii, 1017
Elephas, ii, 991
Emaciation in chorea gravis, i, 743
— in fever, i, 47
— in phthisis, i, 954
— from vomiting, ii, 338
Embolism, i, 1006
— cerebral, i, 554, 560, 599
— — in chorea, i, 754
— — in enteric fever, i, 181
— of adrenals, ii, 741
— pulmonary, i, 1007, 1109
— from endocarditis, i, 953, 1010
— of splenic arteries in enteric fever, i, 181
— of spleen, i, 957; ii, 744
Emetics in croup, ii, 30
— in plastic bronchitis, ii, 128
Emphysema of lungs—
— interstitial, ii, 106
— vesicular, ii, 105
— — friction-sound in, ii, 115, 176
— senile, ii, 110
Emphysema, subcutaneous—
— in pertussis, ii, 288
— — in phthisis, ii, 223
Emphysema, subpleural—
— — in diphtheria, i, 271
— — in pertussis, ii, 288
— — in asthma, ii, 294
Emprosthotonos in tetanus, i, 759
— in hydrophobia, i, 769
Empyema, ii, 165, 172
— diagnosis from serous effusion, ii, 172
— in course of pneumothorax, ii, 192
Encephalitis, i, 635
— chronic diffused, i, 646
Enchondroma, i, 104
Endemic hæmaturia, ii, 702
Endocarditis, acute, i, 949
— chronic, i, 951
— in chorea, i, 745, 753
— in diphtheria, i, 273
— in rheumatism, ii, 814
— in scarlatina, i, 215
— ulcerative, i, 952
English cholera, i, 298
Enteric fever, i, 164
— diagnosis of, i, 185
— — from ague, i, 186
— — from colitis, i, 188
— — from genito-urinary diseases, i, 188
— — from hepatic abscess, i, 188
— — from perinephric abscess, i, 188
— — from pneumonia, i, 187
— — from pyæmia, i, 187
— — from trichiniasis, i, 187
— — from tubercular meningitis, i, 687
— — from tuberculosis, i, 186, 187
— — from typhilitis, i, 187
— — from ulcerative endocarditis, i, 187
— differentiation from typhus, i, 164
— hæmorrhage, i, 178
— myelitis a sequela, i, 449
— perforation of bowel, i, 180
— tuberculosis a sequela, ii, 270
Enteritis, ii, 384
Ephelides, ii, 1023

Ephemeral fever, i, 207
Epilation in favus, ii, 972
— in ringworm, ii, 969
— in sycosis, ii, 995
Epilepsia major, i, 794
— minor, i, 793
Epilepsy, i, 792
— abortive, i, 796
— apoplectiform, i, 596
— diagnosis from hysteria, i, 807
— masked, i, 796
— relation to paroxysmal insanity, i, 818
— simulation of, i, 807
Epileptic centre, i, 799
— coma, i, 596
— dementia, i, 798
— hemiplegia, i, 798
— mania, i, 798, 818
— vertigo, i, 792
Epileptiform convulsions, *see* Eclampsia
Epistaxis, ii, 281
— in Bright's disease, ii, 616
— in enteric fever, i, 172
— in hæmophilia, ii, 793
— in migraine, i, 782
— in miliary tuberculosis, ii, 265
— in purpura, ii, 788
— in smallpox, i, 237
Epithelial reproduction, i, 66
Epithelioma, i, 116; ii, 56, 1019
— of skin, ii, 1019
Epulis, i, 108
Equinia, i, 376
Ergot in myelitis, i, 470
— in hæmoptysis, ii, 266
Eruptions or exanthems
— in cholera, i, 284
— in dengue, i, 364
— from drugs, ii, 938
— in enteric fever, i, 172
— in foot-and-mouth disease, i, 380
— factitious, i, 823; ii, 960, 1030
— in glanders, i, 377, 378
— in measles, i, 219
— in epidemic meningitis, i, 693
— in migraine, i, 784
— in relapsing fever, i, 155
— in rubeola, i, 225
— in scarlatina, i, 211
— in smallpox, i, 228, 230
— in syphilis, i, 314, 333
— in typhus, i, 133
— in varicella, i, 245
Erysipelas, i, 249
— faucial, i, 254
— idiopathic, i, 252
— surgical, i, 249, 251
Erythema, ii, 927
— annulatum, ii, 931
— definition of, ii, 928
— bullosum, ii, 931
— exudativum, ii, 931
— fugax, ii, 931
— gyratum, ii, 931
— iris, ii, 933
— leve, ii, 931
— marginatum, ii, 931
Erythema multiforme, ii, 813, 931
— nodosum, ii, 934
— papulatum, ii, 900, 931
— relation to rheumatism, ii, 813, 930
— vesiculæ, ii, 931
Essera Vogelii, i, 226
État pointillé of intestine, i, 168
État ponctué of eczema, ii, 875
Exaltation of ideas—
— in disseminated sclerosis, i, 550
— in general paralysis of the insane, i, 665
Excision of renal calculi, ii, 686
— larynx, ii, 57
— pylorus, ii, 366
— gallstones, ii, 511
Exfoliative dermatitis, ii, 908
Exophthalmic goitre, i, 1009
Exotic diseases of the skin, ii, 1012
Exudation, catarrhal, i, 61
— croupous, i, 62
— diphtheritic, i, 62
— plastic, i, 60
— purulent, i, 61

F.

Facial paralysis, i, 401
— spasm, i, 701
Factitious lesions of the skin, i, 823; ii, 1030
— urticaria, ii, 935
— chromidrosis, ii, 960
Famine-fever, i, 161
Faradisation, *see* Electricity
Farcy, i, 376
Fat-embolism in diabetes, ii, 721
Fatty degeneration in anæmia, ii, 590
— — of heart, i, 940
— — of kidneys, ii, 626
— — of liver, ii, 542
Fauces, affections of, ii, 305
Favus, ii, 971
Febricula, i, 207
Febrile jaundice, ii, 497
Febris variolosa, i, 230
Feeding by nasal tube, i, 850, 855
— by œsophageal tube, i, 743
— by stomach pump, i, 850, 856
— of lunatics by force, i, 850
Fermentation, alcoholic, i, 23
— lactic, i, 23
— as a test for sugar in urine, ii, 710, 712
Ferments in disease, i, 22
Fever, i, 37
Fibrinous bronchitis, ii, 124
Fibroid disease of heart, i, 933
— phthisis, ii, 152, 209
Fibroma, i, 104, 607; ii, 53, 1013
— molluscum, ii, 1013
— recurrent, i, 106
Fibro-sarcoma, i, 107
— papillare, ii, 53
Fibrous rheumatism, ii, 811, *footnote*
Filaria sanguinis hominis, ii, 459, 992
Fistula in ano, ii, 227
Flatulence in hysteria, i, 828
— in dyspepsia, ii, 325

Flexibilitas cerea, i, 830
Floating kidney, ii, 705
Fluctuation in ascites, ii, 481
" Fluid vein," of anæmic murmurs, ii, 751
— — of cardiac murmurs, i, 957
— — of respiratory murmur, ii, 85
Follicles, solitary, in enteric fever, i, 166
Food, *see* Diet
Foot-and-mouth disease in cattle, i, 380
— in man, i, 381
Fragilitas crinium, ii, 977
— ossium, ii, 862
Frambœsia, ii, 1012
Freckles, ii, 1023
Frémissement cataire, i, 963
Fremitus, hydatid, ii, 549
— vocal, ii, 92
Friction-sound in emphysema, ii, 114, 176
— in pericarditis, i, 944, 945
— in pleurisy, ii, 163
— in peritonitis, ii, 465
Friedreich's disease, i, 545
Function-spasm, i, 720
Fungus hæmatodes, i, 107
Fungi, i, 24
— in pertussis, ii, 290
— in phthisical vomicæ, ii, 208
— of the skin and hair, ii, 962
— in thrush, ii, 301
Ferrocyanide test for albuminuria, ii, 596
Furor epilepticus, i, 818
Furunculus, ii, 956
Fusible calculus, ii, 660

G.

Gait in paralysis agitans, i, 731
— in paraplegia, i, 437
— in tabes dorsalis, i, 531
Gall-stones, ii, 500, 510
Galvanism, *see* Electricity
Gangrene, i, 63
— in cholera, i, 286
— in diabetes, ii, 717
— from embolus, i, 1007
— in enteric fever, i, 180
— in smallpox, i, 238
— in typhus, i, 140
— of genitalia in measles, i, 221
— of lung, ii, 159
— — in typhus, i, 140
— of spleen in ague, i, 345
Gas, its composition in pneumothorax, ii, 192
Gastralgia, ii, 332
Gastric catarrh, acute, ii, 319
— chronic, ii, 326
— carcinoma, ii, 354
— casts, ii, 320
— concretions, ii, 367
— crises, i, 535
— dilatation, acute, ii, 323
— — chronic, ii, 362
— disorder in chronic alcoholism, i, 883
— induration, ii, 366
— ulcer ii, 344
— sarcoma, ii, 356
Gastritis, acute, ii, 319
— chronic ii, 326
— phlegmonous, ii, 324
Gastro-colic fistula, ii, 363
Gastro-cutaneous fistula from cancer ii, 356
— from simple ulcer of stomach, ii, 346
Gastrodynia, ii, 332
Gastro-enteritis, i, 208
Gastro-intestinal anthrax, i, 372
Gastrostomy ii, 317
Geographical distribution—
— of ague, i, 347
— of anthrax, i, 367
— of bronchitis, ii, 121
— of cholera, i, 289
— of dengue, i, 363
— of epidemic dysentery, ii, 393
— of eczema, ii, 881
— of elephantiasis, ii, 992
— of favus, ii, 972
— of gout, ii, 804
— of influenza, ii, 276
— of leprosy, ii, 1009
— of lichen tropicus, ii, 899
— of measles, i, 217
— of epidemic meningitis, i, 690, 696
— of phthisis, ii, 255
— of relapsing fever, i, 154
— of typhus, i, 131, 145
— of yellow fever, i, 355, 361
Giant-cells—
— in lupus, ii, 996
— in rhinoscleroma, ii, 1008
— in sarcomata, i, 107
— in simple inflammation, i, 66
— in tubercle, i, 81
Gingivitis, ii, 300
Girdle-feeling in paraplegia, i, 486
— in chronic spinal meningitis, i, 495
— in locomotor ataxy, i, 534
— in myelitis, i, 451
Glanders, i, 375
— acute, i, 377
— chronic i, 378
Globus hystericus, i, 828
Glomerulo-nephritis, ii, 625
Glossitis in chorea, i, 745
Glosso-labio-pharyngeal paralysis, i, 517
Glossy skin, i, 398
Glottis, spasm of, in tetanus, i, 763
— — in laryngismus stridulus, ii, 15
— — in spurious croup, ii, 22
— œdema of, ii, 46
Glycogenesis, increased, ii, 725
Glycose in urine, tests for, ii, 708
Glycosuria, theory of, ii, 721
Goa powder, ii, 968
Goitre compressing trachea, ii, 60
— endemic, i, 877
— exophthalmic, i, 1009
— substernal, ii, 60
Gonorrhœal synovitis, ii, 835
Gout ii, 797
— and insanity, i, 864
Gowland's cosmetic, ii, 946
Granular degeneration of heart, i, 941; ii, 105

Granular kidney, ii, 640
— pharyngitis, ii, 308
Granulation-tumours, i, 114
Graves's disease, i, 1009
Greenstick fractures in rickets, ii, 851
Green-sickness, ii, 769
Gurjun oil in leprosy, ii, 1012
Guaiacum test for blood in urine, ii, 663
Guarana in migraine, i, 791
Gummata, i, 321
— in congenital syphilis, i, 331, 335
— of brain, i, 607
— of heart, i, 935
— of larynx, ii, 44
— of liver, ii, 530
— of lung, ii, 239
— of placenta, i, 331
— of spinal meninges, i, 482
Gums in scurvy, ii, 781
Gurgling in disease of the lungs, ii, 88
— in enteric fever, i, 174
Gutta rosea, ii, 944, 979
Gyrate eruptions, ii, 911, 931

H.

Hæmatemesis, ii, 339
— from cancer of stomach, ii, 342, 359
— from cirrhosis of liver, ii, 342, 526
— from gastric ulcer, ii, 342, 350
— treatment of, ii, 343
— vicarious, ii. 342
Hæmatidrosis, ii, 961
Hæmatinuria, intermittent, ii, 586
Hæmatoma of dura mater, i, 668, 709
Hæmatomyelia, i, 465
Hæmaturia, ii, 663
— from acute nephritis, ii, 628
— endemic, ii, 702
— parasitic, ii, 702
— from renal calculus, ii, 665
— from renal cancer, ii, 665, 697
— from renal tuberculosis, ii, 692
— treatment of, ii, 666
Hæmochrometer, ii, 749
Hæmocytometer, ii, 748
Hæmoglobin, estimate of, in blood, i, 749
Hæmoglobinometer, ii, 749
Hæmoglobinuria, ii, 583
— infantile, ii, 585
— paroxysmal, ii, 586
Hæmophilia, ii, 792
Hæmoptysis—
— in cirrhosis of lung, ii, 155
— in emphysema, ii, 112
— in syphilitic laryngitis, ii, 46
— from mediastinal tumour, i, 1003
— in miliary tuberculosis of lungs, ii, 264
— in phthisis, ii, 215, 227
— in plastic bronchitis, ii, 126
— in pneumonia, ii, 137
— treatment of, ii, 256
— vicarious, ii, 219
Hæmorrhage—
— cerebral, i, 561, 588, 591, 600; ii, 620
Hæmorrhage in Bright's disease, i, 563; ii, 609, 616
— from cirrhosis of liver, ii, 527
— in enteric fever, i, 178
— from invagination of intestine, ii, 411
— in leuchæmia, ii, 756
— meningeal, i, 590
— in remittent fever, i, 345
— in scurvy, ii, 780
— spinal meningeal, i, 493
— of spinal cord, i, 465
— in yellow fever, i, 356, 357
Hæmorrhagic affections of the skin, ii, 1024
— arthritis, ii, 794
— diathesis, ii, 792
— erosions of stomach, ii, 328
Hæmothorax, i, 165, 184; ii, 178
Hair, affections of, ii, 962
Hallucinations in hysteria, i, 829
Handwriting in general paralysis, i, 666
Haut mal, i, 792
Hay-asthma, ii, 296
Hay-fever, ii, 283
Headache from cerebral tumour, i, 612
— in lithæmia, ii, 489
— from meningitis, i, 681, 691, 702
— sick, i, 779
— in syphilis, i, 313
— in uræmia, ii, 618
Health-resorts in phthisis, ii, 257
Heart, diseases of, i, 897–986
— aneurysm of, i, 937, 997
— calcareous infiltration of, i, 941
— congenital disease of, i, 979
— dilatation, i, 918
— — in asthma, ii, 294
— — in chronic bronchitis, ii, 874
— — in cirrhosis of lung, ii, 156
— displacement of—
— — in pleuritic effusion, ii, 168
— — in pneumothorax, ii, 196
— fatty, i, 940
— fibroid, i, 933
— granular degeneration of, i, 941; ii, 105
— hydatid of, ii, 557
— hypertrophy of, i, 918
— — in Bright's disease, ii, 611
— inhibition of, i, 15
— palpitation of, i, 902
— paralysis of, in tetanus, i, 764
— in pyrexia, i, 49
— rupture of, i, 941
— valvular diseases of, i, 949
— — their relation to phthisis, ii, 254
Heat-regulation in fever, i, 42
Heat-stroke, i, 45, 891
Hemeralopia, ii, 782
Hemianæsthesia in hemiplegia, i, 574
— in hysteria, i, 824
Hemicrania, i, 779
Hemichorea, i, 741
Hemilaryngoplegia, ii, 4
Hemiopia from lesions of chiasma, i, 618
— lateral, i, 573
— in meningitis, i, 682
— in migraine, i, 780
Hemiparaplegia, i, 471

Hemiplegia, i, 553, 567
— association with aphasia, i, 580
— alternate, i, 571
— from lesion of basal ganglia, i, 572
— in chorea, i, 746
— from cortical lesions, i, 572
— crossed, i, 571, 574
— in disseminated sclerosis, i, 550
— in epilepsy, i, 798
— in course of general paralysis, i, 667
— with hemianæsthesia, i, 574
— from hydrocephalus, i, 655
— in hysteria, i, 825, 837
— with lateral hemiopia, i, 573
— from meningitis, i, 685, 692
— with early rigidity, i, 570
— with late rigidity, i, 574
— spinal, i, 471
— with visual affections, i, 573
Hemispasm, i, 622
Hemitritæus, i, 165, 341
Hepatic abscess, ii, 514
— diagnosis from enteric fever, i, 188
— — pleuritic effusion, ii, 177
Heredity in asthma, ii, 294
— in cancer, i, 122
— in chorea, i, 751
— in diabetes, ii, 726
— in disseminated sclerosis, i, 551
— in eclampsia, i, 806
— in epilepsy, i, 803
— in general paralysis, i, 664
— in gout, ii, 803
— in hæmophilia, ii, 792
— in insanity, i, 848
— in leprosy, ii, 1011
— in migraine, i, 786
— in molluscum fibrosum, ii, 1014
— in neuroses generally, i, 778
— in phthisis, ii, 231
— in progressive muscular atrophy, i, 510
— in pseudo-hypertrophic paralysis, i, 528
— in rheumatism, ii, 816
— in tabes, i, 545
Hernia, internal, ii, 420
Herpes, ii, 932
— circinatus, ii, 932, 970
— facialis, ii, 932
— — in epidemic meningitis, i, 693
— — in pneumonia, ii, 138
— gestationis, ii, 934
— iris, ii, 933
— labialis, ii, 932
— præputialis, ii, 932
— symptomaticus, ii, 138
— zoster, ii, 932, 1025
Heubner's disease of cerebral arteries, i, 556
Hiccough, ii, 339
— in uræmia, ii, 619
Histrionic spasm, i, 715
Hobnail liver, ii, 522
Hodgkin's disease, ii, 761
Horseshoe kidney, ii, 705
Humoral pathology, ii, 866
Hyaline casts in urine, ii, 603
Hydatids, ii, 545
— of brain, i, 609; ii, 557
Hydatids of heart, ii, 557
— of kidney, ii, 678, 698
— of liver, ii, 545
— of lung, ii, 553, 557
— of pelvis, ii, 558
— of spine, i, 481
— of spleen, ii, 557
Hydrargyria, ii, 941
Hydroa, ii, 921, 934
Hydrocephalic cry, i, 682
Hydrocephalus, acute, i, 675
— of adults, i, 659
— of childhood, i, 649
— externus, i, 652, 710
— spurious, i, 687
— from tumour of brain, i, 610
Hydromyelus, i, 514
Hydronephrosis, ii, 674
Hydropericardium, i, 947
Hydrophobia, i, 714, 767
— spurious, i, 774
Hydropneumopericardium, i, 943
Hydropneumothorax, ii, 192
Hydrothorax, ii, 178
Hyperæsthesia in epidemic meningitis, i, 692
— in hysteria, i, 824
— in leprosy, ii, 1011
— in neuralgia, i, 430
— in rickets, ii, 856
Hyperæsthetic zone in hemiparaplegia, i, 473
Hyperalgesia in hysteria, i, 824
Hyperidrosis, ii, 959
Hyperpyrexia, i, 40
— from heat-stroke, i, 892
— in chorea, i, 745
— in erysipelas, i, 253
— in hysteria, i, 835
— paradoxical, i, 41
— in rheumatism, ii, 832
— in scarlatina, i, 213
— in smallpox, i, 236
— in tetanus, i, 759
Hyper-resonance, ii, 78, 113, 293
Hypertrophic affections of the skin, ii, 981
Hypertrophy of brain, i, 647
— of heart, i, 918
— of liver, ii, 542
Hypochondriasis, i, 843
— distinction from hysteria, i, 843
— — from insanity, i, 844
Hypodermic syringe in diagnosis, ii, 178
Hypoplasia of aorta in anæmia, ii, 591
— of blood-vessels in hæmophilia, ii, 796
Hysteria, i, 821
— diagnosis from cerebral tumour, i, 628
— — epilepsy, i, 807
— — meningitis, i, 689
— epileptiform, i, 834
— in males, i, 837
— mental condition in, i, 822
— relation to neuroses, i, 838
— simulated by tetanus, i, 762
Hysterical contracture, i, 826
— aphonia, ii, 12
— fit, i, 829
— ischuria, ii, 669
Hystero-epilepsy, i, 834

I.

Ichthyosis, ii, 985
— congenita, ii, 988
— hystrix, ii, 987
— linguæ, ii, 913, 983, *see* Leucoplacia
Icterus, ii, 492, *see* Jaundice
— gravis, ii, 531
— simplex, ii, 495
Idiocy, i, 872
— congenital, i, 873
— from deprivation of senses, i, 875
— epileptic, i, 874
— hydrocephalic, i, 874
— inflammatory, i, 874
— traumatic, i, 874
Idiopathic anæmia of Addison, ii, 772
Idiosyncrasy, i, 7
Illusions, in chronic dementia, i, 747
— in mania, i, 851
Imbecility, i, 872
Imitation, a cause of chorea, i, 752
— a cause of hysteria, i, 838
Impaction of bowel, ii, 415
Impetigo, ii, 894
— contagiosa, ii, 896
— herpetiformis, ii, 934
In-coordination, in tabes, i, 538
Incubation, i, 27
— of ague, i, 339
— of cholera, i, 280
— of diphtheria, i, 267
— of dysentery, ii, 396
— of enteric fever, i, 170
— of erysipelas, i, 252
— of foot-and-mouth disease, i, 381
— of hydrophobia, i, 967
— of influenza, ii, 274
— of mumps, ii, 304
— of pertussis, ii, 286
— of plague, i, 303
— of relapsing fever, i, 155
— of rubeola, i, 224
— of smallpox, i, 227
— of syphilis, i, 308
— of tubercle, i, 89
— of typhus, i, 132
— of vaccinia, i, 241
— of varicella, i, 244
— of whooping-cough, ii, 286
— of yellow fever, i, 356
Indican in urine, ii, 498, 568
Indigo calculus, ii, 659
Infantile convulsions, i, 806
Infarctus, i, 1008
— a cause of fibroid disease of heart, i, 936
— of lung in leuchæmia, ii, 757
Infecting chancre, i, 309
Infection, i, 22
— prevention of, i, 34
— protection from, by former attacks, i, 29
Infective diseases, i, 22, 130
Inflammation, ætiology of, i, 67
— cardinal symptoms of, i, 52, 57
— catarrhal, i, 61
— chronic, i, 68
— contagious, i, 26
Inflammation, membranous, i, 61, 182, 214, 221, 231, 236, 378
— — from traumatic causes, i, 266; ii, 19
— diphtheritic, i, 62, 260
— erysipelatous, i, 249, 252
— gangrenous, i, 63
— infective, i, 26
— microscopical appearances in frogs, i, 53
— — in warm-blooded animals, i, 57
— a physiological process, i, 55
— putrid, i, 63
Inflation of bowel for intussusception, ii, 413
Influenza, ii, 274
Ingravescent apoplexy, i, 589, 603
Inhalation in acute laryngitis, ii, 34
Inoculation, i, 240
Inosite in urine, ii, 561
Insanity, i, 846
— alcoholic, i, 862
— certificates of, i, 868, 869
— delusional, i, 857
— forms and varieties of, i, 846
— medico-legal relations of, i, 866
— moral, i, 863
— paralytic, i, 865
— paroxysmal, i, 817
— puerperal, i, 860
— relation to hysteria, i, 833
— — to hypochondriasis, i, 844
— statistics of, i, 847
Insolatio, i, 891
Insomnia in acute alcoholism, i, 886
— in acute mania, i, 852
— in cardiac disease, ii, 974, 978
— in delirious mania, i, 850
— in pneumonia, i, 145
— in typhus fever, i, 151
Inspection of thorax, ii, 92
Intermittent fever, i, 338
— pulse, i, 899
Interstitial keratitis, i, 336
Intertrigo, ii, 930
Interval in ague, i, 341
Intestinal casts, ii, 405
— obstruction, ii, 407, 415
—ulceration in dysentery, ii, 389
— — in enteric fever, i, 166
— — in erysipelas, i, 257
— — in phthisis, ii, 381
Intestine, diseases of, ii, 368
Intracardiac thrombi, i, 935
Intrathoracic tumours, i, 998
Intussusception of intestine, ii, 407
Invagination of intestine, ii, 407
Invasion—
— of acute tuberculosis, ii, 265
— of ague, i, 339
— of cholera, i, 280
— of dengue, i, 363
— of diphtheria, i, 268
— of enteric fever, i, 170
— of erysipelas, i, 253
— of influenza, ii, 274
— of measles, i, 218
— of plague, i, 303
— of pneumonia, i, 135
— of relapsing fever, i, 155

Invasion—
— of rubeola, i, 225
— of scarlatina, i, 210
— of syphilis, i, 313
— of typhus, i, 132
— of varicella, i, 244
— of variola, i, 227
— of yellow fever, i, 356
Iodide of potash a cause of purpura, ii, 788
— in syphilis, i, 329, 335
— effect on urine, ii, 569
Iodide rash, ii, 939
Iodine in osteo-arthritis, ii, 848
— as a test for lardacein, ii, 630
Ipecacuanha in asthma, ii, 298
— in bronchitis, ii, 122, 123
— in dysentery, ii, 402
Iris or herpes iris, ii, 933
Iritis, i, 319, 337
Iron in anæmia, ii, 778
— in Bright's disease, ii, 656
— in cardiac disease, i, 977
— in chlorosis, ii, 771
— in chorea, i, 756
— in diphtheria, i, 275, 276, 279
— in eczema, ii, 890
— in erysipelas, i, 257
— in erythema nodosum, ii, 938
— in phthisis, ii, 261
— in convalescence from rheumatism, ii, 834
— presence in the liver in anæmia, ii, 751
Irregular pulse, i, 899
Irritable heart, i, 904

J.

Jaundice, ii, 492, 495
— from acute yellow atrophy of liver, ii, 531
— from cardiac disease, i, 975
— from cancer of biliary duct, ii, 541
— — of liver, ii, 540
— from cirrhosis of liver, ii, 499, 527
— from congenital occlusion of duct, ii, 499
— epidemic, ii, 497
— febrile, ii, 497
— from gall-stones, ii, 500
— in miliary tuberculosis of liver, ii, 266
— in pneumonia, ii, 138, 498
— in pyæmia, ii, 498
— from pylephlebitis, ii, 498
— in relapsing fever, i, 155
— from stricture of bile-duct, ii, 500
— in typhus, i, 140
— in yellow fever, i, 356, 358
Jungle fever, i, 344

K.

Kakké, i, 432
Keloid of Addison, ii, 989, *see* Sclerodermia
— of Alibert, ii, 1014, *see* Cheloid
Keratitis, i, 336
Keratolysis, ii, 908
Keratosis, ii, 899, 903
— diffusa intra-uterina, ii, 988
— linguæ, ii, 984
— pilaris, ii, 899
Kerion, ii, 968
Kidney—
— absent, ii, 672
— cancer of, ii, 695
— cirrhosis of, ii, 639
— cysts of, ii, 394
— floating, ii, 705
— hydatid of, ii, 678, 698
— hypertrophy of, ii, 673
— lardaceous, ii, 634
— large red, ii, 624
— large white, ii, 625
— malformations of, ii, 705
— moveable, ii, 705
— multicystic disease of, ii, 594, 650
— sarcoma of, ii, 694
— small red, ii, 639
— small white, ii, 627
— tubercular, ii, 689
— waxy, ii, 634
Knee-jerk, i, 440
— in locomotor ataxy, i, 537
— in spastic paraplegia, i, 489
Kousso in tapeworm, ii, 445

L.

Labyrinthine vertigo, i, 813
Lactic acid and acute rheumatism, ii, 817
Lactic fermentation, i, 23
Lac virginum, ii, 946
Lancing the gums for laryngismus, ii, 17
Laparotomy, ii, 431
Lardaceous disease, ii, 630
— of adrenals, ii, 714
— from congenital syphilis, ii, 632
— of intestine, ii, 382
— of kidneys, ii, 594, 629
— of liver, ii, 545
— in phthisis, ii, 227
— of spleen, ii, 638
— from suppuration, ii, 632
— from syphilis, ii, 632
Laryngeal insufflation, ii, 42
— paralysis, ii, 4
— phthisis, ii, 37
Laryngismus stridulus, ii, 14
— — relation to rickets, i, 738
Laryngitis, acute catarrhal, ii, 33
— chronic catarrhal, ii, 34
— œdematous, ii, 46
— phlegmonous, ii, 46
— stridulous, ii, 18, 21
— syphilitic, ii, 44
Laryngoplegia, ii, 4
Laryngoscope, ii, 1 *et seq.*
Laryngoscopic appearances—
— in carcinoma of larynx, ii, 57
— in chronic laryngitis, ii, 35
— in laryngeal malformations, ii, 58
— in paralysis of abductor muscles, ii, 8
— — of adductor muscles, ii, 10
— — of recurrent laryngeal nerve, ii, 5
— in tracheal obstruction, ii, 65
— in tubercular disease of larynx, ii, 40
Larynx, angioma of, ii, 54

Larynx, carcinoma of, ii, 56
— diphtheria of, i, 270, 277
— enteric ulceration of, i, 181
— extirpation of, ii, 57
— fibroma of, ii, 53
— foreign bodies within, ii, 59
— lipoma of, ii, 54
— lupus of, ii, 43
— malformations of, ii, 58
— mucous cyst of, ii, 54
— myxoma of, ii, 54
— œdema of, in Bright's disease, ii, 47
— — in erysipelas, i, 257
— — in glanders, i, 378
— operations on, ii, 55
— papilloma of, ii, 53
— perichondritis of, ii, 49
— syphilitic ulceration of, ii, 44
— sarcoma of, ii, 56
— scarification of, in œdema, ii, 49
— spasm of, i, 738; ii, 18,
— tubercular disease of, ii, 37
— in glanders, i, 378
Lateral curvature of spine, i, 478
Lead-colic, ii, 370
Lead-palsy, i, 505, *see* also Plumbism
Lemon-juice in rheumatism, ii, 825
Lentigo, ii, 1023
Leontiasis, ii, 1010
Lepra vera, ii, 910, 1009
Leprosy, ii, 1009
Leptomeningitis infantum, i, 679
Leuchæmia, i, 114; ii, 753
— lymphatica, ii, 761
— splenica, ii, 753
Leuchæmic enlargement of liver, ii, 542
Leucin in urine, ii, 534
Leucodermia, ii, 1021
Leucoplacia, ii, 913, 983
Lichen, ii, 897
— agrius, ii, 899
— circumscriptus, ii, 897
— pilaris, ii, 899
— planus, ii, 901
— ruber, ii, 901
— scrofulosus, ii, 900
— tropicus, ii, 899
— urticatus, ii, 900
Lientery, ii, 363
Lightning-pains, i, 535
Lime-juice in scurvy, ii, 784
Linear atrophy of the skin, ii, 990
Lipæmia in diabetes, ii, 721
Lipoma, i, 104, 1014
Lipothymia, i, 16
Lithates, *see* Urates
Lithæmia, ii, 489
Lithiasis, ii, 573
Lithic acid, ii, 570, *see* Uric acid
Liver, abscess of, ii, 514
— acute yellow atrophy of, ii, 531, 1140
— cancer of, ii, 530, 538
— capsulitis of, ii, 529
— cirrhosis of, ii, 499, 522
— in diabetes, ii, 721
— fatty, ii, 542
— hydatid tumours of, ii, 545
Liver, hypertrophy of, ii, 542
— in leuchæmia, ii, 542, 758
— nutmeg, i, 957
— — in chronic bronchitis, ii, 105
— pulsation of, i, 965
— in rickets, ii, 852
— syphilitic disease of, ii, 529
— tuberculosis of, ii, 266, 499
— in yellow fever, i, 358
Local distribution of skin diseases, ii, 870
Localisation of cerebral lesions, i, 617
Lockjaw, i, 757
Locomotor ataxia, i, 530
— diagnosis from general paralysis, i, 671
— hereditary form of, i, 545
— in disseminated sclerosis, i, 541
— relation to paraplegia, i, 533
Lotura carnea, ii, 397
Lumbago, ii, 662
Lung—
— atelectasis of, ii, 101
— brown induration of, i, 956
— carnification of, ii, 102, 167
— cirrhosis of, ii, 152
— collapse of, ii, 101, 127, 166
— emphysema of, interstitial, ii, 106
— — vesicular, ii, 105
— gangrene of, i, 140; ii, 159, 160
— infarctus of, i, 956; ii, 757
— inflammation of, ii, 129, 148
— iron-grey induration of, ii, 206
— local compression of, i, 166
— senile atrophy of, ii, 110
— syphilitic disease of, i, 156
— tuberculosis of, i, 83; ii, 200, 260
— tumours of, i, 1005
Lupus, ii, 995 *et seq.*
— erythematosus v. sebaceous, ii, 1006
— — disseminatus, ii, 1008
— lymphaticus, ii, 1017, *see* Lymphangioma
— of larynx, ii, 43
Lymphangioma, ii, 1017
Lymph-glands (lympharia) in dengue, i, 364
— in enteric fever, i, 167, 169
— in erysipelas, i, 254, 255
— in Hodgkin's disease, ii, 761, 765
— in hydrophobia, i, 768
— in leuchæmia, ii, 757
— in scarlatina, i, 211
— tuberculosis of, ii, 767
Lymph, organisation of, i, 66
— plastic, i, 60
Lymph-scrotum, ii, 701, 992
Lymphoma, i, 81, 115, 144
Lympho-sarcoma, i, 115
Lysis, i, 40
Lyssa, i, 767

M.

Macula, ii, 870, 1023
Maculæ hepaticæ, ii, 972
Main en griffe, in hemiplegia, i, 574
— in paralysis of ulnar nerve, i, 392
— in progressive muscular atrophy, i, 508

Malacosteon, ii, 861
Malaria i, 338, 347
— relation to phthisis, ii, 253
— — to yellow fever, i, 359
Malarial cachexia, i, 345, 355
Male fern, liquid extract of, ii, 445
Malformation of kidney, ii, 705
— of heart, i, 979
— of larynx, i, 58
Malignancy, Virchow's characters of, i, 99.
Malignant endocarditis, i, 952
— disease, *see* Cancer
— pustule, i, 370
Malingering in hysteria, i, 822, 830
Malleus, i, 376
Mania, acute delirious, i, 849
— in chorea, i, 743, 746
— in chronic alcoholism, i, 883
— in epilepsy, i, 798, 817
— in general paralysis of the insane, i, 665
— in hydrophobia, i, 769
— in meningitis, i, 688
— ordinary acute, i, 851
— paroxysmal, i, 817
Marasmus in diabetes, ii, 716
— following enteric fever, i, 183
— in hysteria, i, 835
— in phthisis, ii, 211
— in pyrexia, i, 47
— primary, i, 516, *footnote*
Marriage, relation of syphilis to, i, 323
Masked ague, i, 344
Massage in peripheral paralysis, i, 400
— in pseudo-hypertrophic paralysis, i, 529
— in primary marasmus, i, 516, *footnote*
Measles, i, 217
— benign, i, 220
— hæmorrhagic, i, 220
— broncho-pneumonia in, ii, 148
— malignant, i, 220
Measly meat, ii, 439
Mediastinal abscess, ii, 62
— tumours, i, 999–1005
— — obstructing trachea, ii, 62
Medicinal eruptions, ii, 938 *et seq.*
Medico-legal relations of insanity, i, 866
Megalocytes in anæmia, ii, 750
Megrim, i, 780
Melæna, ii, 340
Melanæmia, i, 346
— relation to hæmaturia, i, 347
— nature of pigment, i, 346
Melancholia, i, 853
— after chorea, i, 746
— in disseminated sclerosis, i, 550
— distinction from hypochondriasis, i, 844
— with stupor, i, 856
Melanodermia, ii, 734, 1022
Melanoma of pia mater, i, 608
Melanotic sarcoma, i, 108
Melasma suprarenale, ii, 734
— cutis, ii, 1022
Meliceris, ii, 950
Memory, loss of, in chronic dementia, i, 866
— — in general paralysis, i, 665
Ménière's disease, i, 813
Meningeal hæmorrhage, i, 562, 590
Meninges, cerebral—
— — hæmorrhage into, i, 562, 590
— — tumours of, i, 607
— spinal—
— — hæmorrhage in, i, 493
— — gumma of, i, 482
Meningitis, cerebral, acute simple, i, 698
— of base, i, 679
— in chorea, i, 745
— epidemic cerebro-spinal, i, 690
— a cause of hydrocephalus, i, 651
— diagnosis from hysteria, i, 689
— idiopathic, i, 701
— associated with pneumonia, ii, 133
— metastatic, i, 700
— tubercular, of adults, i, 684
— — of children, i, 675
— — diagnosis from enteric fever, i, 187
— — in phthisis, ii, 227
— spinal, acute, i, 493
— — chronic, i, 494
Meningo-ependymitis, i, 659
Mentagra, ii, 953, *see* Sycosis
Mercurial eruptions, ii, 941
— tremor, i, 894
Mercury—
— externally for eczema, ii, 885
— — impetigo capitis, ii, 896
— — ringworm, ii, 966
— internally in hepatic dyspepsia, ii, 490
— — in hydrocephalus of adults, i, 663
— — mitral disease, i, 977
— — in syphilis, i, 326, 335, 603
Metallic echo, ii, 195
— tinkling in pneumothorax, ii, 194
Miasmatic-contagious diseases, i, 21
Micrococci, i, 24
— in pneumonia, ii, 143
— in diphtheria, i, 262, 268, *footnote*
— in erysipelas, i, 251
— in puerperal fever, i, 262
— in purpura, ii, 791
— of ammoniacal urine, 580
Microcytes in anæmia, ii, 750
Microphytes, i, 24, *see* also Bacteria, Bacilli, Micrococci, Spirilla, Sarcina
— pleomorphism of, i, 25, *footnote*
Microsporon furfurans, ii, 973
— mentagraphytes, ii, 955
Microzymes, i, 24
— of contagia, i, 33
— of miasmata, 32
— physiological action of, i, 28
Micturition in paraplegia, i, 441
Migraine, i, 419, 779
— in ague, i, 786, 787
Miliaria, ii, 813, 961
— in measles, i, 219
— papulosa, ii, 899
— in rheumatism, ii, 813
Miliary aneurysms, i, 564, 665
— fever, ii, 813, *footnote*
— sclerosis, i, 669
— tubercles, i, 81; ii, 262
Milium, ii, 948
Milk-diet in acute gastric catarrh, ii, 322

Milk in Bright's disease, ii, 654, 656
— in diabetes, ii, 729
— in enteric fever, i, 202
— in gastric irritation, ii, 338
— in gastric ulcer, ii, 354
— in rheumatism, ii, 822, 834
— of perlsucht, a source of tubercle, i, 90
— causing spread of enteric fever, i, 196
— of foot-and-mouth disease, i, 381
Mineral waters in chronic bronchitis, ii, 124
— in chronic gastric catarrh, ii, 330
— in diabetes, ii, 732
— in eczema, ii, 888
— in gout, ii, 809
— in hepatic dyspepsia, ii, 491
— in jaundice, ii, 509
— in lithæmia, ii, 491
— in pityriasis rubra, ii, 909
Mitral stenosis, i, 962
— regurgitation, i, 965
Mixed chancre, i, 311
Molimina, i, 588
Mollities ossium, ii, 861
Molluscum, meaning of word, ii, 951, *note*
— contagiosum, i, 116; ii, 950
— corpuscles, ii, 951
— fibrosum, ii, 1013
— sebaceum, i, 950
Monomanie des grandeurs, i, 665
Moral insanity, i, 863
Morbilli, i, 217
— sine catarrho, i, 220
— sine morbillis, i, 220
Morbus maculosus, *see* Purpura
Morphæa, ii, 989
Morphia, insufflations in laryngitis, ii, 42
— in heart disease, i, 978
— eruption from, ii, 940
— injection in neuralgia, ii, 429
— — in spasmodic torticollis, i, 719
Moveable kidney, ii, 705
Movement-spasms, i, 720
Mucous patches, i, 317; ii, 44
Multilocular hydatid, ii, 548
Mumps, ii, 302
Murmurs over fontanelle, i, 654
— metamorphosing, ii, 222
— vesicular or pulmonary, ii, 84
— of anæmia, i, 957; ii, 751
— of cardiac disease, i, 957, *cf.* Bruits
— cardiac, in chorea, i, 742
— — in cirrhosis of lung, ii, 156
— — in pleuritic effusion, ii, 168
Murrain, i, 380
Muscle of heart, diseases of, i, 918
Muscles in acute atrophic paralysis, i, 498
— in cervical pachymeningitis, i, 514
— in chronic atrophic paralysis, i, 503
— in disseminated sclerosis, i, 550
— in lateral sclerosis, i, 512, 575
— in locomotor ataxia, i, 515, 534
— in sciatica, i, 424
— in paraplegia, i, 440
— in plumbism, i, 505
— in pseudo-hypertrophic paralysis, i, 526
— in progressive muscular atrophy, i, 507,
— in general paralysis of the insane, i, 666
Muscular contracture in hysteria, i, 826
— — in spasmodic torticollis, i, 716
— in-coordination, from alcoholism, i, 883
— — in locomotor ataxy, i, 538
— rigidity from insular sclerosis, i, 550
— — from lateral sclerosis, i, 488
— — from cerebral hæmorrhage, i, 571, 574
— spasm, after facial paralysis, i, 406
— tremors, in disseminated sclerosis, i, 548
— — from lead, i, 506
— — from mercury, i, 894
— — in paralysis agitans, i, 735
— — in suppression of urine, ii, 670
— — in typhus, i, 136
— — in uræmia, ii, 618
Mycosis fungoides, ii, 1018
Mydriasis, i, 412, 683, 742
Myelitis, acute, i, 445
— — in chorea, i, 745
— — in specific fevers, i, 449
— chronic, i, 447, *see* Sclerosis
Myeloid cells, i, 107
Myocarditis, i, 939
Myoma, i, 104, *footnote*
— cutis, ii, 1014
Myopathic atrophy, i, 529
Myosis in locomotor ataxy, i, 537
— in typhus, i, 135
Myxœdema, i, 881
Myxoma, i, 105, 608

N

Nævus of the skin, ii, 1016
Nails, eczema of, ii, 878
— psoriasis of, ii, 912
— pemphigus affecting, ii, 921
— tinea and favus of, ii, 971
Narcotic poisoning and apoplexy, i, 593
Nasal diphtheria, i, 270, 277
Necrosis after herpes facialis, ii, 1026
— after enteric fever, i, 182
— of laryngeal cartilages, ii, 49
Nematoid worms, ii, 446
Nephralgia, ii, 667
Nephrectomy, ii, 687
Nephritis—
— interstitial, ii, 593, 594
— lardaceous, ii, 629
— parenchymatous, ii, 594, 624
— scarlatinal, i, 214
— suppurative, ii, 181, 444, 683
Nephro-lithiasis, ii, 662
Nephro-lithotomy, ii, 687
Nephro-phthisis, ii, 689
Nephrotomy, ii, 687
"Nerve-storms," i, 612, 778
Nervous system, diseases of the, i, 385–896
Nettle-rash, *see* Urticaria
Neuralgia, i, 417
— cervico-brachial, i, 423
— cervico-occipital, i, 422
— epileptiform, i, 419
— of rachialgia, i, 473

Neuralgia, reflex, i, 417
— sciatic, i, 423
— trifacial, i, 419
— from chronic alcoholism, i, 884
— from malarial poisoning, i, 344
— from syphilis, i, 313
— after zona, ii, 1027
Neurasthenia spinalis, i, 452
Neuritis, peripheral, i, 429
Neuroma cutis, ii, 1014
Neuroses, i, 713–845
— paroxysmal, i, 778
— relation of neuralgia to, i, 419
— relations to one another, i, 778, 789, 802
— spasmodic, i, 713
Neuro-retinitis, *see* Optic neuritis
Neurotic affections of the skin, ii, 1025
Night-blindness in scurvy, ii, 782
— terrors of children, i, 820
Nitrate of silver in epilepsy, i, 811
— in tabes, i, 544
Nitric acid test for albumen in urine, ii, 596
Nitrite of amyl in angina pectoris, i, 916
— in epilepsy, i, 810
Nitroglycerine in Bright's disease, ii, 656
Node of skin, ii, 870
— of inherited syphilis, i, 335
— of tertiary syphilis, i, 321
Nodi digitorum, ii, 842
Nodule, i, 96; ii, 870
— subcutaneous, in rheumatism, ii, 815
Noma after measles, i, 221
Nomenclature, i, 386
— of cutaneous diseases, ii, 865
Non-infecting chancre, i, 309
Nosology, i, 4, 125
Nummular sputa of phthisis, ii, 213
Nutmeg liver, i, 957; ii, 105
Nyctalopia, ii, 782
Nystagmus in disseminated sclerosis, i, 549
— in hydrocephalus of childhood, i, 655
— in hereditary locomotor ataxy, i, 546

O.

Obesity, ii, 544
Obstruction of the bowels, ii, 407
— acute, ii, 429
— chronic, ii, 424
Ocular lesions of insular sclerosis, i, 549
— in locomotor ataxy, i, 535
— in migraine, i, 780
Odour of breath in diabetes, ii, 716
— of favus, ii, 972
— of chronic mania, i, 736
— of last stage in meningitis, i, 684
— of smallpox, i, 228
— of viscera in diabetes, ii, 720
Œsophagus, diseases of, ii, 312–317
— carcinoma of, ii, 62, 314
— dilatation of, ii, 313
— diphtheria of, i, 270
— stricture of spasmodic, ii, 313
— — organic, ii, 314
Oidium albicans, ii, 301
Oil of male fern for tapeworm, ii, 445
— for anchylostomum, ii, 453
Oinomania (Œnomania), i, 884
Oleate of mercury in ringworm, ii, 966
Oliguria, ii, 560
Onychomycosis, ii, 971
Ophthalmia in gonorrhœal arthritis, ii, 837
— in measles, i, 221
Opisthotonos in tetanus, i, 758
— in tetany, i, 738
Opium in acute alcoholism, i, 885
— in cardiac disease, i, 978
— in chronic bronchitis, ii, 123
— in chronic intestinal obstruction, ii, 427
— in diabetes, ii, 731
— in meningitis, i, 706
— in neuralgia, i, 428
— in peritonitis, ii, 472
— in rheumatism, ii, 823, *footnote*
— in typhlitis, ii, 387
— poisoning by, i, 593
Optic chiasma, course of fibres in, i, 574
Optic disc—
— atrophy of, from cerebral tumour, i, 616
— — in disseminated sclerosis, i, 550
— — in hydrocephalus, i, 655
— — in locomotor ataxy, i, 536
— condition in epilepsy, i, 802
— choked from cerebral tumour, i, 614
— — in meningitis, i, 683, 686
— inflammation of, *see* Optic neuritis
Optic neuritis from Bright's disease, ii, 609
— from cerebral tumour, i, 615
— from meningitis, i, 683, 686, 693
Optic tract, course of fibres in, i, 573
Orchitis from mumps, ii, 303
Organic and functional disease, i, 2
Orthopnœa in cardiac disease, i, 931; ii, 95
Orthotonus in epidemic meningitis, i, 692
Osmidrosis, ii, 960
Osseous changes in osteo-arthritis, ii, 842
— in Charcot's disease of the joints, ii, 848
— in mollities ossium, ii, 863
— in rickets, ii, 851
— in syphilis, i, 322
— changes in congenital syphilis, i, 335
Osteitis deformans, relation to cancer, i, 113
Osteoid disease, malignant, i, 105
Osteo-arthritis, ii, 840
Osteoma, i, 104
Osteomalacia, ii, 861
Otitis in measles, i, 221
— in epidemic meningitis, i, 693
— in scarlatina, i, 214
Otorrhœa and cerebral abscess, i, 636
Ovarian affections, ii, 745
— in hysteria, i, 836
— cyst, diagnosis from ascites, ii, 452
— — — from hydronephrosis, ii, 678
Ovary, compression of, in hysteria, i, 836
Overcrowding and phthisis, ii, 235
Over-exertion and heart disease, i, 927
Oxalate of lime calculus, ii, 658
— in urine, ii, 575
Oxyakoia (oxbacoa), i, 404
Oxyuris vermicularis, ii, 450

Ozæna, ii, 280
— from glanders, i, 378

P.

Pachydermia, ii, 991
Pachymeningitis, cerebral, i, 668
— cervical, i, 513
— hæmorrhagica, i, 709
— in relapsing fever, i, 158
Palate, paralysis of, in diphtheria, i, 274
— — in tonsillitis, ii, 307
— single ulcer of, ii, 299
Palpation in ascites, ii, 480
— in diseases of the chest, ii, 91
— in gastric cancer, ii, 360
Palpitation in cardiac disease, i, 902
— in gastric disorder, ii, 360
Pancreas, diseases of, ii, 742
Papilloma, i, 116; ii, 53
Papule, ii, 868, 904
Paracentesis for ascites, ii, 485
— for hepatic abscess, ii, 521
— for hydatid cyst, ii, 555
— for hydronephrosis, ii, 679
— for pericardial effusion, i, 948
— for pleuritic effusion, ii, 182
— for pneumothorax, ii, 199
Paræsthesia, i, 397, 436
Paralysis, acute ascending, i, 461
— acute atrophic spinal, of adults, i, 502
— — of infants, i, 497
— agitans, i, 729
— alcoholic, i, 463
— Bell's, i, 401
— bulbar, acute, i, 523
— — chronic progressive, i, 510, 517, 550
— of bladder in hemiparaplegia, i, 472
— — in paraplegia, i, 441
— of brachial plexus, i, 389
— after chorea, i, 746
— chronic diffused atrophic, i, 503, 505
— of circumflex nerve, i, 391
— from lesions of crus cerebri, i, 570
— "crutch," i, 389
— Cruveilhier's, i, 507
— of diaphragm, ii, 70
— diphtheritic, i, 274, 279
— in disseminated sclerosis, i, 550
— Duchenne's i, 525
— following epilepsy, i, 794
— of facial nerve, i, 401, 521, 571, 760
— — double, i, 404
— of fifth nerve, i, 414
— of fourth nerve, i, 410
— general, of the insane, i, 663
— general spinal, i, 503
— hemiplegic, i, 568
— hydrocephalic, i, 655
— of hypoglossal nerve, i, 569
— in hemiplegia, i, 569
— hysterical, i, 459, 825
— infantile, i, 497
— labio-glosso-pharyngeal, i, 517
— Landry's, i, 461
— laryngeal, ii, 4 *et seq.*
Paralysis in lateral sclerosis, i, 513
— from lead, i, 505
— in locomotor ataxy, i, 530, 533, 535
— in meningitis, i, 683, 685
— of median nerve, i, 391
— in migraine, i, 783
— of musculo-cutaneous nerve, i, 391
— of musculo-spiral nerve, i, 391
— of oculo-motor nerves, i, 407, 520, 535
— of olfactory nerve, i, 415
— peripheral, i, 389
— of posterior thoracic nerve, i, 390
— in progressive muscular atrophy, i, 511
— pseudo-hypertrophic, i, 525
— of rectum in hemiparaplegia, i, 472
— — in paraplegia, i, 443
— of recurrent laryngeal nerve, ii, 4
— reflex, i, 390
— of sixth nerve, i, 408
— of third nerve, i, 411
— of ulnar nerve, i, 391
Parangi, ii, 1012
Paraplegia, i, 433
— from alcohol, i, 463
— from anæmia of spinal cord, i, 453
— from compression of cord, i, 478; ii, 697
— from concussion of cord, i, 459
— in disseminated sclerosis, i, 550
— from hæmorrhage into cord, i, 465
— from hyperæmia of cord, i, 455
— hysterical, i, 459
— from chronic spinal meningitis, i, 452
— from myelitis, i, 445
— from neurasthenia spinalis, i, 452
— painful, i, 483
— partial, i, 437
— reflex, i, 456
— spastic, i, 488
— syphilitic, i, 413, 464
— from tumour within the cord, i, 466
Parasites, animal—
— Acarus scabiei, ii, 923
— Ancylostomum duodenale, ii, 452
— Ascaris lumbricoides, ii, 446
— Bilharzia hæmatobia, ii, 702
— Bothriocephalus latus, ii, 442
— Oxyuris vermicularis, ii, 450
— Tænia mediocanellata, ii, 441
— — solium, ii, 439
— Trichina spiralis, ii, 454
Parasites, vegetable, i, 24, *see* Microphytes
— Achorion Schœnleinii, ii, 971
— Microsporon mentagraphytes, ii, 955
— — furfurans, ii, 973
— Trichophyton tonsurans, ii, 955, 962, 974
Parasiticides, vegetable, ii, 968
— animal, ii, 445, 449
Parasitic hæmaturia, ii, 702
— sycosis, ii, 955
Parenchymatous nephritis, ii, 594, 624
Paresis in paralysis agitans, i, 730
Parkinson's disease, i, 729
Parotitis, ii, 302, 304
— in relapsing fever, i, 159
— after typhus, i, 140
— after yellow fever, i, 259
— after influenza, i, 357

Paroxysmal insanity, i, 817
— vertigo, i, 812
Pébrine, i, 92
Pectoriloquy, ii, 89
Pediculi capitis, ii, 895
— corporis v. vestimentorum, ii, 904
Pelade, ii, 975, *see* Area
Peliosis rheumatica, ii, 813, 1025
Pellagra, ii, 1012
Pelvis in osteomalacia, ii, 862
— in rickets, ii, 853
Pemphigus, ii, 917
— an acute form, ii, 918
— foliaceus, ii, 920
— gangrænosus, ii, 920
— gestationis sive hystericus, ii, 934
— neonatorum, i, 333; ii, 919
— pruriginosus, ii, 934
— serpiginosus, ii, 920
Penicillium glaucum in urine, ii, 713
Peptones in urine, ii, 597
Percussion of chest, ii, 75
— in ascites, ii, 481
— in examination of heart, i, 923
— — of liver, ii, 533
— — of spleen, i, 325
Perforation of intestine in enteric fever, i, 180
— of stomach by gastric ulcer, ii, 346
Pericardial adhesions, i, 945
Pericarditis, i, 942
— in Bright's disease, ii, 611
— in acute pneumonia, ii, 133
— in rheumatism, i, 814
— in scarlatina, i, 215
Perichondritis, laryngeal, ii, 49
Perihepatitis, ii, 529
Perinephric abscess, ii, 683
— diagnosis from enteric fever, i, 188
Periosteal cachexia, ii, 855, *footnote*
Peripheral neuritis, i, 429
Peritoneal friction-sound, ii, 164, 465
Peritoneum, diseases of, ii, 463–486
Peritonitis, acute, ii, 463
— in enteric fever, i, 180
— from erysipelas of abdominal wall, i, 257
— from renal calculus, ii, 668
— cancerous, ii, 478
— circumscribed, ii, 468
— from perforation of gastric ulcer, ii, 465
— — of typhoid ulcer, i, 180; ii, 465
— — of appendix cæci, ii, 385, 465
— puerperal, ii, 465
— chronic, ii, 474
— tubercular, ii, 476
Perityphlitis, ii, 385
Perlsucht, i, 89; ii, 201
Pernicious anæmia, ii, 772
— malarial fever, i, 343
Pernio, ii, 933
Pertussis, ii, 286
— broncho-pneumonia in, ii, 148
Petechiæ, ii, 1024
— in epilepsy, i, 796
— in erythematous rashes, ii, 929
— in measles, i, 220
— in purpura, ii, 788
— in rheumatism ii ,813
Petechiæ, in scurvy, ii, 782
— in typhus, i, 134
Petit mal, i, 792
Peyer's patches in enteric fever, i, 166
Pharyngitis, granular, ii, 308
— membranous, in glanders, i, 378
— — in measles, i, 218
— — in scarlatina, i, 214
Pharynx, adenoid growths of, ii, 310
— anæsthesia of, in hysteria, i, 825
— diphtheria of, i, 270
— paralysis of, in bulbar paralysis, i, 518
— — in diphtheria, i, 274
Phlegmonous erysipelas, i, 249
— gastritis, ii, 324
Phosphates, diminution in alcoholism, i, 886
Phosphatic calculus, ii, 660
— urine, ii, 566, 864
Phosphorus in Hodgkin's disease, ii, 767
— in neuralgia, i, 427
— in pernicious anæmia, ii, 779
Phrenic nerves, galvanisation of, ii, 71
Phthinoid chest, ii, 233
Phthiriasis, ii, 904
Phthisis, ii, 200
— acute, ii, 212, 225, 229
— following enteric fever, i, 183
— diabetic, ii, 238, 718
— "florida," ii, 212
— ab hæmoptoë, ii, 215
— relation to heart disease, ii, 254
— from inhalation of dust, ii, 243
— — carbonaceous particles, ii, 241
— — cotton or flax dust, ii, 244
— — common dust, ii, 239
— — oxide of iron dust, ii, 243
— — siliceous particles, ii, 243
— pleurisy in, ii, 180
— pneumothorax in, ii, 227
— malaria, relation to, ii, 253
— syphilitic, ii, 238
Phthisis, traumatic, ii, 253
— relation to tubercle, i, 80, 83; ii, 203
— tubercular, ii, 203
— relation to inflammation, i, 83; ii, 153
— intestinal ulceration in, i, 94; ii, 382
Physical examination of chest, ii, 75
Physiognomy of congenital syphilis, i, 335
— of hydrocephalus, i, 653
Physiological albuminuria, ii, 599
Pian, ii, 1012
Picric acid test for albumen in urine, ii, 596
— — for sugar in urine, ii, 711
Piedra, ii, 977
Pigeon-chest, ii, 852
Pigment, biliary, ii, 494
— in melanæmia, i, 346
— urinary, ii, 567
Pigmental anomalies of the skin, ii, 1021
Pityriasis capitis, ii, 907
— nigra, ii, 907
— pilaris, ii, 899
— rosea, ii, 973
— rubra, ii, 908 *et seq.*
— tabescentium, ii, 949
— versicolor, ii, 972, *see* Tinea versicolor
Placenta, syphilis of, i, 331

Plague, i, 302 *et seq.*
Plaques dures, of enteric fever, i, 166
— molles, of enteric fever, i, 166
— opalines in syphilis, i, 319, 321
— à surface reticulée, of enteric fever, i, 168
Plastic bronchitis, ii, 124 *et seq.*
Plessimeter, ii, 76
Pleurisy, ii, 163 *et seq.*
— with broncho-pneumonia, ii, 149
— in Bright's disease, ii, 179, 611
— with cirrhosis of lung, ii, 154
— dry, ii, 163
— with pneumonia, ii, 133
— with phthisis, ii, 180
— in rheumatism, ii, 179, 814
— in scarlatina, i, 215
Pleuritic effusion, ii, 165
— diagnosis from aneurysm, ii, 177
— — from cancerous growth, ii, 177
— — from hydatid of liver, ii, 177
— — from hypophrenic abscess, ii, 177
— — from pericardial effusion, ii, 177
— serous, diagnosis from empyema, ii, 178
— friction-sound, ii, 149, 163, 174, 176
Pleurodynia, ii, 74
Pleuropneumonia, ii, 133
Pleurosthotonus, i, 760
Plica polonica, ii, 963
Plumbism, ii, 370
— causing "blue line," i, 506; ii, 372
— — atrophy of brain, i, 673
— — colic, ii, 370
— — paralysis, i, 505
— — epilepsy, i, 864
— — gout, ii, 804
— — Bright's disease, ii, 641
Pneumonia, acute, lobar, fibrinous, ii, 129
— in Bright's disease, ii, 146, 629, 645
— in croup, ii, 29
— in diabetes, ii, 718
— in disease of heart, ii, 146
— hypostatic, i, 140, 181, 586; ii, 147
— catarrhal, ii, 148 *et seq.*
— chronic, ii, 147
— — interstitial, ii, 152
— lobular, ii, 148
— — pyæmic, ii, 151
Pneumonoconiosis, ii, 239
Pneumothorax, ii, 189 *et seq.*
— composition of air, ii, 192
— from emphysema, ii, 188
— from empyema, ii, 190
— from gangrene of lung, ii, 189
— from muscular effort, ii, 189
— in pertussis, ii, 189, 288
— in phthisis, ii, 190, 227
— traumatic, ii, 189
Pock of smallpox, i, 231
Point de côté of pleurisy, ii, 171
— of acute pneumonia, ii, 136
Polyarthritis, ii, 810
Polypoid form of tumour, i, 96
— growths in chronic laryngitis, ii, 35
Polypus, a cause of invagination, ii, 410
— nasal, a cause of asthma, ii, 296
Polyuria, ii, 561, 712
Pompholyx, ii, 917
Pompholyx diutina, ii, 918
Pomphos, *see* Wheal
Porrigo, ii, 896
— decalvans, ii, 975, *see* Area
— lupinosa, ii, 971, *see* Favus
— scutuluta, ii, 962, *see* Ringworm
Post-hemiplegic chorea, i, 576
Post-peritoneal hernia, ii, 420
Post-pharyngeal abscess, ii, 29
— in scarlatina, i, 214
Postponement of ague-fit, i, 341
Potash salts in scurvy, ii, 786
— test for sugar in urine, ii, 711
Potassio-mercuric iodide test for albumen in urine, ii, 597
Potassium bromide in epilepsy, i, 810
— — in migraine, i, 791
— — in ordinary acute mania, i, 853
— — in paroxysmal vertigo, i, 815, 817
— — in pertussis, ii, 291
— — in writers' cramp, i, 727
— ferrocyanide test for albumen, ii, 596
— iodide in chronic mercurialism, i, 806
— — in general paralysis, i, 671
— — in hemiplegia, i, 604
— — in migraine, i, 791
— — in plumbism, i, 507; ii, 374
— — in syphilis, i, 329, 335
— — in syphilitic cerebral tumour, i, 633
Pott's disease of spine, i, 478
Præsystolic murmur, i, 962, 967
Pregnancy and acute atrophy of liver, ii, 535
— during chorea, i, 744, 752
Premonitory stage—
— of ague, i, 339
— of cholera, i, 280
— of enteric fever, i, 170
— of erysipelas, i, 252
— of measles, i, 218
— of meningitis, i, 681, 684
— — epidemic cerebro-spinal, i, 691
— of relapsing fever, i, 155
— of rubeola, i, 225
— of syphilis, i, 312
— of typhus, i, 132
— of varicella, 244
— of yellow fever, i, 356
Priapism in paraplegia, i, 443
Prickly heat, ii, 899
Prodroma—
— of acute ascending paralysis, i, 461
— of apoplexy, i, 588
— of epilepsy, i, 794
— of hydrophobia, i, 768
— of locomotor ataxy, i, 535
— of meningitis—
— — epidemic, i, 691
— — tubercular, i, 681, 684
Proglottis, ii, 436
Prognosis, i, 8
Progressive muscular atrophy—
— of adults, i, 507
— of childhood, i, 524
— hereditary, i, 525
— in bulbar paralysis, i, 510
— primary form, i, 529

Progressive muscular atrophy—
— secondary forms, i, 513
— — in disseminated sclerosis, i, 515
— — in lateral sclerosis, i, 513
— — in locomotor ataxy, i, 515
— — with pachymeningitis cervicalis, i, 513
— — with syringomyelus, i, 514
Prophylaxis against hydatid disease, ii, 554
— against cholera, i, 299
— against scurvy, ii, 784
— against tapeworms, ii, 445
— against trichina, ii, 458
Prosopalgia, i, 419
Prurigo, ii, 903, 905
— of Hebra, ii, 905
— infantilis, ii, 935
— senilis, ii, 904
— after varicella, i, 246; ii, 90
Pruritus, ii, 904
— from jaundice, ii, 506
— from uræmia, ii, 619
— from diabetes, ii, 717
Prussic acid, poisoning by, i, 593
Psammoma, i, 608
Psellismus mercurialis, i, 895
Pseudo-angina pectoris, i, 917
Pseudo-hypertrophic paralysis, i, 525
Psoriasis, ii, 910
— and gout, ii, 913
— guttata, ii, 914
— linguæ, ii, 913, 984, *see* Leucoplacia
— palmar and plantar, i, 317
— and syphilis, ii, 914
Psychoses, i, 846–875
Ptosis, i, 412
Ptyalism, *see* Salivation
Puerile breathing, ii, 84
Puerperal peritonitis, ii, 465
Pulmonary apoplexy, i, 956
— catarrh, ii, 151
— embolism, i, 1007, 1009
Pulse, i, 897, 905
— in anæmia, ii, 753
— in Bright's disease, ii, 613
— capillary, in aortic disease, i, 967
— frequent, i, 898
— infrequent, i, 899
— in pyrexia, i, 48
— ratio to respiration in pneumonia, ii, 136
— slow and infrequent, i, 897; ii, 496
— alternating, i, 901
Pupils, state described by Argyll Robertson, i, 536
— in chorea, i, 742
— in general paralysis of the insane, i, 666
— in locomotor ataxy, i, 536
— in meningitis, i, 682
— in migraine, i, 783
— in typhus, i, 135
Purgatives in Bright's disease, ii, 655
Purpura, ii, 787
— in epidemic meningitis, i, 693
— in measles, i, 220
— in plague, i, 303
Purpura in relapsing fever, i, 155
— in sarcomatosis, i, 111
— in scarlatina, i, 212
Purpura in smallpox, i, 229
— in typhus, i, 134
— urticans, ii, 929
— variolosa, i, 229
Purpurine, ii, 575
Pus-cells, i, 52
Pustule, ii, 868
— malignant, i, 370
Putrid urine, ii, 579
Pyæmia, i, 70
— and cerebral abscess, i, 637
— and enteric fever, i, 187
— and rheumatism, ii, 821
— idiopathic, i, 74
Pyelitis, ii, 680
Pylephlebitis, ii, 498
— adhesiva, ii, 530
Pyo-pneumothorax, ii, 175
Pyrexia, i, 28, 37, *see* Temperature
— asthenic, i, 49
— classification of, i, 40
— degrees of, i, 39
— defervescence, i, 47
— fastigium, i, 46
— hectic variety, i, 44, *footnote*
— idiopathic, i, 44
— initial stage, i, 45
— pulse in, i, 48
— respiration in, i, 49
— stages of, i, 39
— sthenic, i, 49
— symptomatic, i, 44
— theories of causation, i, 42
— wasting in, i, 47
Pyrogallic acid locally in psoriasis, ii, 915
— in tinea marginata, ii, 971
— in tinea tonsurans, ii, 968
Pyrosis, ii, 336
Pythogenic fever, i, 190
Pyuria, ii, 680
— in cystitis, ii, 681
— in suppurative nephritis, ii, 684
— in tubercular disease of kidney, ii, 693

Q.

Quarantine, i, 27
— against plague, i, 305
— against yellow fever, i, 361
Quartan ague, i, 341
Quinine in ague, i, 354
— in eczema, ii, 891
— in hæmoglobinuria, ii, 590
— in pertussis, ii, 291
— in prurigo, ii, 906
Quinsy, ii, 306
Quotidian ague, i, 341

R.

Rabies in animals, i, 770
— in man, i, 767
Rachialgia, i, 473
Rachitic chest, ii, 852

Rachitis, ii, 850, *see* Rickets
Radesyge, i, 306; ii, 1012
Râles, ii, 87
— consonating, ii, 88
Raynaud's disease, ii, 590
Reaction of degeneration, i, 392
— in acute atrophic paralysis, i, 498
— in chronic atrophic paralysis, i, 503
— in disseminated sclerosis, i, 550
— in paraplegia, i, 441
— in progressive muscular atrophy, i, 511
Reaction stage of cholera, i, 283
Recurrence of ague-fit, i, 341
— of cholera, i, 299
— of eczema, ii, 876
— of enteric fever, i, 185
— of erysipelas, i, 255
— of measles, i, 222
— of mumps, ii, 303
— of plastic bronchitis, ii, 127
— of relapsing fever, i, 156
— of rubeola, i, 226
— of scarlatina, i, 216
— of syphilis, i, 325
— of typhus, i, 146
— of varicella, i, 248
— of variola, i, 238
— of whooping-cough, ii, 289
Recurrent fibroid tumour, i, 102
Reduplication of cardiac sounds, i, 964
Reflex paraplegia, i, 456
— action in chorea, i, 747
— — in paraplegia, i, 437
— — coordination of, i, 438
— — abolished by shock, i, 438
— — in hemiparaplegia, i, 472
— — loss of, in Landry's paralysis, i, 462
— — in acute atrophic paralysis, i, 498
— — in chronic atrophic paralysis, i, 503
— — in hysteria, i, 825
Reflexes, i, 440
Regurgitation through aortic valves, i, 965
— through mitral valve, i, 965
— gastric, ii, 335
— œsophageal, ii, 313
— pulmonary valves, i, 967
— through tricuspid valve, i, 965
Relapse in cholera, i, 284
— in epidemic meningitis, i, 695
— of enteric fever, i, 183
— of measles, i, 219
— of relapsing fever, i, 157
— after rheumatism, ii, 826
— of typhus, i, 138
— of varicella, i, 246
Relapsing fever, i, 153
Remittent fever, i, 344
Renal calculi, ii, 658
— colic, ii, 667
— cyst, ii, 643, 676
— disease and cerebral hæmorrhage, i, 563
— dropsy, ii, 606
— inadequacy, ii, 564
— pulse, ii, 613
Resolution, i, 65
Resonance, amphoric, ii, 80
— pulmonary, ii, 77
Resonance, osteal, ii, 80
— subtympanitic, ii, 80
— tubular, ii, 80
— tympanitic, ii, 79
Respiration, artificial, i, 12
Retinal hæmorrhage in anæmia, ii, 776
— in Bright's disease, ii, 609
Retinitis from cerebral tumour, i, 615
— from lardaceous kidneys, ii, 638
— from leuchæmia, ii, 757
— from meningitis, i, 687
— from parenchymatous nephritis, ii, 609
— from syphilis, i, 337
Retraction of head in meningitis, i, 683, 692
Retroperitoneal hernia, ii, 420
Retropharyngeal abscess, ii, 29
Rhagades, ii, 869
"Rheumatic gout," ii, 840
Rheumatic nodules, ii, 815
Rheumatism, ii, 810
— and erythema, ii, 930, 935
— after scarlatina, i, 214
— and gout, ii, 805
— and chorea, i, 752
Rheumatoid arthritis, ii, 810
Rhinoderma, ii, 899
Rhinoscleroma, ii, 1008
Rhonchus, ii, 87, 96
Rhythm of cardiac murmurs, i, 958
Rib, excision in empyema, ii, 188
Rice-water stools of cholera, i, 281, 287
Rickets, ii, 850
— acute, ii, 855
— and hypertrophy of brain, i, 647
— and laryngismus stridulus, i, 738
— and tetany, i, 737
Rigidity, early, of hemiplegia, i, 570
— late, of hemiplegia, i, 574
— in hysteria, i, 826
— from insular sclerosis, i, 550
— from lateral sclerosis, i, 489
— from meningeal hæmorrhage, i, 590
— of neck in meningitis, i, 686, 692
— in paralysis agitans, i, 730
— in spastic paraplegia, i, 488
Rigor, i, 45; ii, 932
— in ague, i, 340
Ringworm of the body, ii, 970
— of the scalp, ii, 962
Risus sardonicus, i, 758
Rodent ulcer, i, 119; ii, 998, 1019
Roseola, ii, 927
— annulata, ii, 931
— choleraica, i, 284
— epidemica i, 223
— syphilitica, i, 315
— variolosa, i, 228
"Rotation" in hysteria, i, 832
Rötheln, i, 223
Round-worms, ii, 446
Rub in broncho-pneumonia, ii, 149
— in emphysema of lungs, ii, 115, 176
— pericardial, ii, 41
— pleuritic, ii, 163
— peritoneal, ii, 164, 465
Rubeola, i, 223

Rumination, ii, 313, 335
Rupture of heart, i, 941

S.

Saccharine in diabetes, ii, 729
Sago-grain vesicles, ii, 961
Sago-spleen, ii, 638, 745
St Vitus's dance, i, 740
Salicin in rheumatism, ii, 825
Salicylate of soda in rheumatism, ii, 826
Salicylic rash, ii, 941
Salivation, ii, 301
— in bulbar paralysis, i, 519
— in hydrophobia, i, 770
— from mercury, i, 895; ii, 301
— from potassium iodide, ii, 302
— in smallpox, i, 238
Saltatorial spasms, i, 720
Santonin, as a vermifuge, ii, 449, 453
Sarcina ventriculi, i, 24; ii, 363
— in urine, ii, 583
Sarcoma, i, 106
— alveolar, i, 107
— of brain, i, 608
— of cerebral meninges, i, 608
— of kidney, ii, 694
— of larynx, ii, 56
— lymphatic, i, 115
— of mediastinum, i, 999
— melanotic, i, 108
— multiple, i, 110; ii, 1020
— round-celled, i, 106
— of spinal cord, i, 467
— spindle-celled, i. 106
— of stomach, ii, 356
Sarcomatosis, i, 110
Sarcoptes hominis, ii, 923
Saturnine gout, ii, 804
Satyriasis, ii, 1010
Sayre's jacket in spinal caries, i, 487
Scab, ii, 869
Scabies, ii, 923
Scale, ii, 869, 875, 907
Scarification for gutta rosea, ii, 981
— for lupus, ii, 1005, 1007
Scarlatina, i, 209 *et seq.*
— anginosa, i, 214
— hæmorrhagica, i, 213
— maligna, i, 213
Scars, ii, 978
— in relation to cheloid, ii, 1015
Scelotyrbe festinans, i, 731
Scherlievo, i, 306
Sciatica, i, 423
Scirrhus, i, 117
Sclerema, ii, 990
— neonatorum, ii, 990
Scleriasis, ii, 988, 989
Sclerodermia, ii, 988
Sclerosis, i, 447
— disseminated, i, 547
— insular, i, 547
— lateral, i, 496, 512
— — ascending, i, 485
— — descending, i, 575
— — in general paralysis, i, 669
Sclerosis, miliary, i, 669
— posterior in general paralysis, i, 669
— — in locomotor ataxy, i, 537
Sclerostomum duodenale, ii, 452
Sclerotitis in gonorrhœal arthritis, ii, 837
Scoliosis, i, 478
Scorbutus, ii, 780
Scraping in treatment of lupus, ii, 1005
Scrofula, ii, 1001
Scrofulides, ii, 995, 1000
Scrofulodermia papulosa, ii, 901
Scurvy, ii, 780 *et seq.*
Sea-voyage in phthisis, ii, 257
Sebaceous cyst, ii, 950
Seborrhœa, ii, 949
— congestiva, ii, 1006
— sicca, ii, 949
Secondary fever of smallpox, i, 235
Semi-tertian ague, i, 341
Sensation in disseminated sclerosis, i, 549
— in locomotor ataxy, i, 532
— in migraine, i, 783
— in paralysis agitans, i, 731
— in paraplegia, i, 435, 483
— in writers' cramp, i, 724
— in hysteria, i, 824
Serous inflammations in m. Brightii, ii, 611
Shaking palsy, i, 729
Shingles, ii, 1025, *see* Zona
Sibbens, i, 306; ii, 1012
Sibilus, ii, 96
Sick-headache, i, 779
Siderosis, ii, 243
Silver salts in locomotor ataxy, i, 544
Simulation of epilepsy, i, 805
Sinuses of skull, thrombi of, i, 699, 708
Skin, diseases of, ii, 865–1034
Skoda's resonance in pleural effusion, ii, 170
Skull of congenital syphilis, i, 335
— of cretins, i, 876
— of hydrocephalus, i, 653
— of rickets, ii, 853
Smallpox, i, 227–239
Soft chancre, i, 309
Softening of the brain, 559, 634, 675
— — relation to hæmorrhage, i, 561
— — red, i, 560, 634
— — white, i, 559, 675
— — yellow, i, 559, 609
— of spinal cord, i, 445, 500
— — relation to hæmorrhage, i, 446, 465
Soil in causation of phthisis, ii, 247
Soil-water theory of cholera, i, 296
— — of enteric fever, i, 199
Solitary follicles in dysentery, ii, 389, 391
— in enteric fever, i, 168
— in Hodgkin's disease, ii, 766
Solvent treatment of renal calculi, ii, 685
Somnambulism in hysteria, i, 831
— in paroxysmal insanity, i, 819
Sore-throat, ii, 305
Spasm of glottis in laryngismus, ii, 14
— — in spurious croup, ii, 22
— — in true croup, ii, 26
— in hysteria, i, 826
— of muscles after facial paralysis, i, 406
— of throat in hydrophobia, i, 769

Spasmodic diseases, i, 714
— tic, i, 715
— croup, ii, 22
— torticollis, i, 716
Spasmus glottidis, ii, 14
Specific diseases, i, 20
— gravity of urine, ii, 560
Spedalskhed, ii, 1009
Speech in bulbar paralysis, i, 518
— in disseminated sclerosis, i, 549
— in general paralysis, i, 666, 667
— in idiocy, i, 873
— in locomotor ataxy, i, 545
— in migraine, i, 783
— in paralysis agitans, i, 731
— in chronic mercurialism, i, 895
Spermatorrhœa, i, 844
Sphygmograph, i, 971
Spina bifida, and hydrocephalus, i, 652
Spinal column—
— cancer of, i, 480
— caries of, i, 478; ii, 479
— hydatid of, i, 481
Spinal cord—
— anæmia of, i, 453
— concussion of, i, 459
— glioma of, i, 467
— hæmorrhage into, i, 466
— hyperæmia of, i, 455
— in acute atrophic paralysis, i, 504
— in chronic atrophic paralysis, i, 504
— in general paralysis, i, 670
— in locomotor ataxy, i, 534
— in progressive muscular atrophy, i, 513
— sarcoma of, i, 467
— sclerosis of, i, 447, *see* Sclerosis
— segment of, i, 434, *footnote*
— softening of, i, 445
— syphilis of, i, 464
— sensory and motar tracts of, i, 436
— tubercle of, i, 466
— tumour of, i, 466
— irritation of, i, 473
— inflammation of, i, 494, 674
Spirilla, i, 24
— of relapsing fever, i, 159
Spirochæta Obermeieri, i, 159
Spirometer, ii, 93
Spleen, diseases of, ii, 743
— abscess of, ii, 743
— in ague, i, 345
— in cardiac disease, i, 957
— in cirrhosis of liver, ii, 526
— in enteric fever, i, 172
— hydatid of, ii, 557
— in Hodgkin's disease, ii, 765
— lardaceous, ii, 633
— in leuchæmia, ii, 753
— physical diagnosis of, ii, 754
— in relapsing fever, i, 155
— in rickets, ii, 855
— in acute tuberculosis, ii, 265
Splenisation of lung, i, 956, 974
Spontaneous origin of contagia, i, 26
Sporadic cretinism, i, 878
Spurious croup, ii, 22
— hydrocephalus, i, 687
Sputa in asthma, ii, 297
— in bronchiectasis, i, 118
— in bronchitis, acute, ii, 99
— — chronic, ii, 104, 105
— — fœtid, ii, 118
— in gangrene of lung, ii, 160
— in miners' phthisis, ii, 241
— in phthisis, ii, 213
— in pneumonia, ii, 137
Statistics, chiefly from Guy's Hospital—
— of aneurysm, i, 998 (Parkin)
— cardiac lesions, i, 984, 985 (Shaw)
— cardiac lesions in rheumatism, ii, 814
— cerebral tumours, i, 610 (White)
— chorea, i, 742, 743, 745 (Manser)
— diabetes (anatomy), ii, 721 (Campbell)
— — and phthisis, ii, 718, *footnote* (Kelbe)
— hereditary diabetes, ii, 727 (Nevins)
— empyema, ii, 173
— gastric ulcer, ii, 348 (Keiffenheim)
— gonorrhœal synovitis, ii, 835 (Ricketts)
— gout, ii, 803
— intestinal obstruction, ii, 434 (Shaw)
— osteo-arthritis, ii, 845
— rheumatism, ii, 814, 816, 817, 819 (Manser and Shadwell)
— — and seasons, ii, 815, *footnote* (Capes)
— — natural course, ii, 824
— — treatment, ii, 825, 827, 831
— paralysis agitans, i, 732 (Shaw)
Status epilepticus, i, 797
Steatoma, ii, 950
Stenosis of aortic orifice, i, 965
— of mitral orifice, i, 962
— of pulmonary orifice, i, 966, 981
— of tricuspid orifice, i, 965
Stertor in apoplexy, i, 586
Stethometer, ii, 93
Stethoscope, ii, 82
Stomacace, ii, 300
Stomach, carcinoma of, ii, 354
— acute catarrh of, ii, 319
— chronic, ii, 326
— cirrhosis of, ii, 366
— concretions in, ii, 367
— congestion of, ii, 342
— — in cardiac disease, i, 957
— — in chronic bronchitis, ii, 105
— — in acute catarrh, ii, 320
— — in chronic catarrh, ii, 328
— — in cirrhosis of liver, ii, 526
— colloid cancer of, ii, 358
— cough from disorder of, ii, 73
— dilatation of, acute, ii, 323
— — chronic, ii, 358, 362
— post-mortem digestion of, ii, 320
— -pump, feeding by, i, 850, 856
— sarcoma of, ii, 356
— simple ulcer of, ii, 344
— washing out, ii, 365
Stomatitis, ii, 300
— in congenital syphilis, i, 334
— gangrenous, ii, 300
Stools of acute yellow atrophy, ii, 534
— of cholera, i, 281
— of dysentery, ii, 396
— of enteric fever, i, 174

Stools of hæmatemesis, ii, 340
— of obstructive jaundice, ii, 505
Stramonium in asthma, ii, 298
Strangulation of bowel, ii, 419
Striæ atrophicæ of skin, ii, 991
Stricture of bile-duct, cancerous, ii, 499
— — congenital, ii, 500
— — simple, ii, 500
— of colon, cancerous, ii, 419
— — simple, ii, 419
— of œsophagus, cancerous, ii, 314
— — functional, ii, 313
— — simple, ii, 314
— of pylorus, ii, 355, 361
— of gut after intussusception, ii, 409, 416
— — after tubercular ulcer, ii, 382, 417
— of large intestine, ii, 419
— of small intestine, ii, 416
Strobila, ii, 436
Strongylus gigas, ii, 454
Strophulus, ii, 900
Struma, ii, 1001
Strychnia in myelitis, i, 471
— in paralysis of larynx, ii, 10
— poisoning by, i, 763
Stupor, i, 856
Styptics in hæmatemesis, ii, 343
— in hæmaturia, ii, 666
— in hæmophilia, ii, 796
— in hæmoptysis, ii, 256
— in purpura, ii, 791
— in scurvy, ii, 786
Subintrant ague, i, 342
Subsultus tendinum in fever, i, 136
Succussion-splash, ii, 195
Sudamina, ii, 813, 961
Sugar in the urine, ii, 708
Suicidal tendency in melancholia, i, 855
Sulphur in treatment of scabies, ii, 926
Sunstroke, i, 891
Suppression of urine, ii, 669
— — in cholera, i, 288
Suppuration and lardaceous disease, ii, 632
Suppurative nephritis, ii, 683
— — in paraplegia, i, 444
Supra-clavicular swelling in cretins, i, 878
Sweat as an irritant, ii, 961
Sweat-glands, affections of, ii, 959 *et seq.*
Sweating in ague, i, 341
— in enteric fever, i, 171
— in paralysis agitans, i, 731
— in phthisis, ii, 212
— in rheumatism, ii, 813
— in rickets, ii, 856
— in tetanus, i, 759
Sycosis, ii, 953
— capillitii, ii, 956
— framboesiformis, ii, 956
— parasitica, ii, 955
Symmetrical gangrene, ii, 930
Symmetry in skin diseases, ii, 876, 1029
— of eczema, ii, 876
— of erythema, ii, 929
— of lupus erythematosus, ii, 1006
— of psoriasis, ii, 912
— of scabies, ii, 925
— of syphilodermia, ii, 1032
Sympathetic ganglia in Graves's disease, i, 1012
— in Addison's disease, ii, 740
— in general paralysis of the insane, i, 669
Symptoms, objective and subjective, i, 6
Syncopal ague, i, 343
Syncope, i, 14, 906
— fatal, in acute alcoholism, i, 887, 890
— — in diphtheria, i, 269
— — in phthisis, ii, 227
— — in pleuritic effusion, ii, 173
Synovial rheumatism, ii, 811
Synovitis, gonorrhœal, ii, 835
— in scarlatina, i, 214
Syphilis, i, 306
— of cerebral arteries, i, 556, 600
— — meninges and brain, i, 607
— congenital, i, 329, 336
— — of bones, i, 332, 335
— — of cornea, i, 336
— of heart, i, 935
— of larynx, ii, 44
— of liver, ii, 529
— of lungs, ii, 156
— a cause of lardaceous disease, ii, 632
— a cause of locomotor ataxy, i, 542
— a cause of meningitis, i, 701
— a cause of myelitis, i, 450
— of placenta, i, 331
— primary, i, 308, 312
— secondary, i, 315
— of spinal arteries, i, 465
— of spinal meninges, i, 464, 482
— tertiary, i, 320
Syphilitic fever, i, 313
— insanity, i, 864
— phthisis, ii, 238
— stenosis of trachea and bronchi, ii, 63
— ulcer, diagnosis from lupus, ii, 998
Syphilodermia—
— follicular, i, 316
— macular, i, 315
— papular, i, 316
— pigmentary, i, 318
— pustular, i, 318
— squamous, i, 318
— vesicular, i, 318
— secondary, i, 315
— tertiary, i, 315, 320
— of congenital syphilis, i, 333
Syringomyelus, i, 467, 515

T.

"Tabby" degeneration of heart, i, 940
Tabes dorsalis, i, 530
Tabid arthritis, ii, 848
Tache cérébrale, i, 683
Tænia mediocanellata, ii, 441
— solium, ii, 439
Tapeworms, ii, 435
Tar acne, ii, 947
— locally in eczema, ii, 885
— in lichen ruber, ii, 903
— in psoriasis, ii, 915
Tarantulism, i, 831
Teeth of inherited syphilis, i, 336

Teeth in rickets, ii, 853
Tichopsia, i, 780
Teigne pelade, ii, 975, *see* Area
Temperature—
— in acute atrophic paralysis, i, 498
— in acute yellow atrophy of liver, ii, 534
— in ague, i, 340
— in anæmia generally, ii, 753
— — grave idiopathic, ii, 776
— in broncho-pneumonia, ii, 150
— in cirrhosis of liver, ii, 527
— in cholera, i, 282, 283, 284
— in chronic atrophic paralysis, i, 504
— in dengue, i, 364
— in diabetes, ii, 716
— in diphtheria, i, 268
— in enteric fever, i, 171, 175, 203
— in erysipelas, i, 253
— in glanders, i, 378
— in jaundice, ii, 501
— in leuchæmia, ii, 758
— in measles, i, 218, 220
— in meningitis, i, 683, 684
— — epidemic cerebro-spinal, i, 692
— in myelitis, i, 451
— in peritonitis, ii, 464
— in phthisis, ii, 211
— in plague, i, 313
— in pleurisy, ii, 172
— in pneumonia, ii, 137
— in relapsing fever, i, 155, 156
— in rheumatism, ii, 812
— in rubeola, i, 225
— in scarlatina, i, 210
— in smallpox, i, 227, 236
— in syphilis, i, 313
— in tetanus, i, 759
— in acute miliary tuberculosis, ii, 215
— in renal tuberculosis, ii, 693
— in typhus, i, 133, 136
— in varicella, i, 247
— in yellow fever, i, 356
— local, in inflammation, i, 59
— — in muscular atrophy, i, 509
— paradoxical, i, 41
— post-mortem rise in tetanus, i, 759
— subnormal, i, 50
Tender points in neuralgia, i, 418
— in rachialgia, i, 473
Tendon-reflexes in paraplegia, i, 440
— in lateral sclerosis, i, 489
— absence in locomotor ataxy, i, 534
Tertian ague, i, 341
Tertiana soporosa, i, 343
Tetanilla, i, 736
Tetanus, i, 714, 757
— acute, i, 764
— chronic, i, 764
— hydrophobicus, i, 774
— idiopathic, i, 761
— traumatic, i, 761
Tetany, i, 714, 736
Thermometer for clinical use, i, 37
Thirst in diabetes insipidus, ii, 562
— — mellitus, ii, 714
Thomsen's disease, i, 734
Thoracocentesis, ii, 182
Thorax—
— deformity from cirrhosis of lung, ii, 155
— — from empyema, ii, 176
— — in phthisis, ii, 220, 233
— examination of, ii, 75, 92
— enlargement from effusion, ii, 168
— of emphysema, ii, 113
— of phthisis, ii, 233
— of rickets, ii, 852
Thread-worms, ii, 450
Thrill, cardiac, i, 958, 963
— hydatid, ii, 549
Throat-cough, ii, 72
Thrombosis, i, 1006
— cardiac, i, 935
— of cerebral arteries, i, 556, 600
— of cerebral sinuses, i, 708
— of femoral vein in enteric fever, i, 183
— of portal vein, ii, 540
Thrush ii, 300
Thymic asthma, ii, 14
Thymus, suppuration in congenital syphilis, i, 333
Thyroid body, absence of, in cretins, i, 879
— in exophthalmic goitre, i, 1010
— removal of, a cause of cretinism, i, 882
Thyrotomy, ii, 55
Tic douloureux, i, 419
— spasmodic, i, 714, 715
Tinea, ii, 962 *et seq.*
— circinata, ii, 970
— decalvans, ii, 975
— favosa, ii, 971
— imbricata, ii, 1012
— marginata, ii, 970
— rosea, ii, 973, *see* Pityriasis rosea
— scutulata, ii, 962
— sycosis, ii, 955
— tonsurans, ii, 962
— versicolor, ii, 979
Tongue in fever, i, 173
— in chronic dysentery, ii, 400
— in diabetes, ii, 714
— in pneumonia, ii, 137
— in gastritis, ii, 319, 325
— in rheumatism, ii, 812
— in scarlatina, i, 211
— in tertiary syphilis i, 321
— ichthyosis of, ii, 983
— paralysis of, i, 517, 518, 569
— ulcer of, in pertussis, ii, 287
Tonic spasm, i, 757
Tonsillitis, acute, ii, 306
— chronic, ii, 307
— in rheumatism, ii, 815
— in scarlatina, i, 211
Tooth-cough, ii, 73
Tophi, ii, 799
Torticollis, i, 716
Torula cerevisiæ, i, 23
— in diabetic urine, ii, 713
— in gastric dilatation, ii, 362
Trachea and bronchi—
— obstruction by foreign body, ii, 64
— stenosis, ii, 60
— — from abscess of mediastinum, ii, 62
— — from aneurysm, ii, 61

Trachea and bronchi—
— — from carcinoma of œsophagus, ii, 62
— — from dilated left auricle, ii, 62
— — from goitre, ii, 60
— — from primary carcinoma, ii, 63
— — from scrofulous glands, ii, 62
— — from syphilitic ulceration, ii, 63
— — from tumour of mediastinum, ii, 62
— — from ulceration in glanders, ii, 63
— tubercular disease of, ii, 37
Tracheotomy for croup, ii, 31
— for diphtheria, i, 278
— for foreign body in air-passages, ii, 68
— for chronic laryngitis, ii, 35
— for paralysis of abductor muscles, ii, 10
— for perichondritis of larynx, ii, 52
— as a cause of pneumothorax, ii, 190
Transfusion of blood in anæmia, ii, 779
— of saline solution in cholera, i, 301
Traube's plugs in fœtid bronchitis, ii, 118
Traumatic diphtheria, i, 267; ii, 19
Tremors, fibrillary, in general paralysis, i, 666
— — in muscular atrophy, i, 509
— in disseminated sclerosis, i, 548
— mercurial, i, 894
— in paralysis agitans, i, 735
— in plumbism, i, 506
— in suppression of urine, ii, 670
— in typhus, i, 136
— in uræmia, ii, 618
Trephining in epilepsy, i, 806
Trichina spiralis, ii, 454
Trichiniasis, ii, 456
— diagnosis from enteric fever, i, 187
Trichocephalus dispar, ii, 452
Trichoclasia, ii, 977
Trichophyton tonsurans, ii, 955, 963
Trichoptilose, ii, 977
Trichorrhexis nodosa, ii, 977
Tricuspid regurgitation, i, 955, 965
— stenosis, i, 965
Triethylamine in rheumatism, ii, 823
Trismus, i, 757
— neonatorum, i, 738
— in uræmia, ii, 618
Tube-casts, ii, 603
Tubercle, its general pathology, i, 79
— of adrenals, i, 93; ii, 733, 741
— artificial production of, i, 87
— articular, i, 95; ii, 266
— of brain, i, 605
— of cerebral meninges, i, 675
— of choroid, i, 687; ii, 267
— of genito-urinary tract, i, 94
— infiltrated, i, 84
— its relation to inflammation, i, 82
— of intestine, i, 94
— of kidney, i, 94; ii, 689
— of larynx, its relation to pulmonary, ii, 37
— — and trachea, ii, 37
— of lymph-glands, i, 95
— of lung, i, 81; ii, 200
— of liver, ii, 266, 499
— microscopic structure of, i, 81, 677
— obsolescence of, ii, 269
— of pericardium, i, 94, 942
Tubercle of peritoneum, i, 94; ii, 476
— of pleura, i, 94; ii, 189
— of serous membranes, i, 94
— of spinal cord, i, 466
— its relation to phthisis, i, 80, 85; ii, 200
Tubercular infiltration, i, 84
Tuberculosis, bovine, i, 90, *see* Perlsucht
— its diagnosis from enteric fever, i, 186
— a sequela of enteric fever, i, 183; ii, 270
— forms of, i, 94
— miliary, ii, 262
Tubular breathing, ii, 84
Tumours, i, 96–125
— adenoma, i, 116
— angioma, i, 103; ii, 55
— cerebral, i, 605
— carcinoma, i, 116
— cheloid, ii, 1014
— cholesteatoma, i, 608
— classification of, i, 103
— cylindroma, i, 119
— enchondroma, i, 104
— epithelioma, i, 116
— fibroma, i, 104, 607; ii, 53
— fibroma molluscum, ii, 1013
— glioma of brain, i, 609
— — of spinal cord, i, 467
— granuloma, i, 114, 379
— gumma of brain, i, 607
— of larynx, ii, 52
— of lung, i, 1005
— of liver, ii, 529
— lipoma, i, 104; ii, 54
— lymphadenoma, i, 114
— lymphangioma, ii, 1017
— lymphoma, i, 114
— lymphosarcoma, i, 115
— malignant osteoid, i, 105
— of mediastinum, i, 999
— melanoma of meninges, i, 608
— of spinal meninges, i, 481
— myxoma, i, 105, 608; ii, 54
— myoma, i, 104; ii, 1014
— neuroma, i, 103; ii, 1014
— osteoma, i, 104
— papilloma, i, 116; ii, 53
— psammoma, i, 608
— sarcoma, i, 106
— of the skin, ii, 1013
— of spinal cord, i, 466
— tuberculous, of brain, i, 605
— — of spinal cord, i, 460
— vascular, ii, 1016
— villous, of bladder, i, 116; ii, 665, 675
— xanthoma, ii, 993, *footnote*
Turpentine, causing strangury, ii, 669
— in purpura, ii, 791
Tyloma, ii, 982
Tylosis linguæ, ii, 984
Tympanites in hysteria, i, 828
— in peritonitis, ii, 464
Typhlitis, ii, 385 *et seq.*
— diagnosis from enteric fever, i, 188
Typhoid state in scarlatina, i, 213
— in typhus, i, 134
Typhus, i, 131 *et seq.*
— siderans, i, 139

Typus inversus, ii, 265
Tyrosin in urine of acute yellow atrophy of liver, ii, 534

U.

Ulcer, rodent, i, 119; ii, 998, 1019
— of intestine in dysentery, ii, 389
— — in enteric fever, i, 166
— — in erysipelas, i, 257
— of mouth, ii, 299
— of palate, ii, 299
— of tongue in pertussis, ii, 287
— — in syphilis, i, 321
Ulcerative endocarditis, i, 952
Unconsciousness in apoplexy, i, 585
— in hysteria, i, 829
Uræmia in Bright's disease, ii, 616
— in cholera, i, 285
— from lardaceous kidneys, ii, 638
— diagnosis from meningitis, i, 688
— theory of, ii, 619
Urate or lithate of soda in gout, ii, 799
Urates or lithates, amorphous, ii, 571
— crystalline, ii, 571
Urea in blood of Bright's disease, i, 285
— — of relapsing fever, i, 158
— in the urine of acute yellow atrophy of liver, ii, 534
— cutaneous excretion of, i, 285; ii, 622
— in urine of cirrhosis of kidneys, ii, 644
— of consecutive Bright's disease, ii, 649
— of diabetes insipidus, ii, 561
— — mellitus, ii, 715
— of parenchymatous nephritis, ii, 628
— of suppurative nephritis, ii, 684
Ureter, obstruction of, ii, 674
— — by calculus, ii, 676
— — by malformation, ii, 677
— — by peritoneal band, ii, 676
— — by rachitic pelvis, ii, 675
— — by traumatic stricture, ii, 676
Uric-acid calculus, ii, 658
— in diabetes, ii, 715
— estimation of, ii, 570
— theory of its formation, ii, 573
— in urine, ii, 570
Uridrosis, ii, 961
Urinary calculi, ii, 657
— pigments, ii, 567
— organs, diseases of, ii, 559–732
Urine—
— in acute alcoholism, i, 886
— in acute yellow atrophy of liver, ii, 533
— in ague, i, 340, 341, 343
— albuminous, in cirrhosis of kidney, ii, 644
— — in consecutive Bright's disease, ii, 649
— — in hæmoglobinuria, ii, 584
— — in lardaceous nephritis, ii, 636
— — in parenchymatous nephritis, ii, 628
— of ammoniacal fermentation, ii, 579
— in grave anæmia, ii, 776
— bilious, ii, 494
— bloody, ii, 663, *see* Hæmaturia
— casts, ii, 603
Urine—
— — in cirrhosis of kidney, ii, 643
— — in hæmoglobinuria, ii, 584
— — in lardaceous disease of kidney, ii, 637
— — in parenchymatous nephritis, ii, 628,
— in cholera, i, 283, 284
— in chorea, i, 742
— in croup, ii, 26
— density of, ii, 560
— — in cirrhosis of kidneys, ii, 647
— — in chorea, i, 742
— — in consecutive Bright's disease, ii, 649
— — in diabetes insipidus, ii, 561
— — in diabetes mellitus, ii, 712
— — in lardaceous nephritis, ii, 636
— — in obstructive suppression, ii, 670
— — in parenchymatous nephritis, ii, 628
— in diabetes insipidus, ii, 561
— in diabetes mellitus, ii, 708
— in diphtheria, i, 273
— in enteric fever, i, 174
— in epidemic meningitis, i, 693
— in erysipelas, i, 255
— in glanders, i, 378
— in hysteria, i, 742, 825
— leucin in, ii, 534
— in melanotic sarcoma, i, 109
— in paraplegia, i, 443 *et seq.*
— in pneumonia, ii, 138
— quantity of, ii, 560
— — in cirrhosis of kidneys, ii, 643
— — in diabetes insipidus, ii, 561
— — in diabetes mellitus, ii, 712
— — in intestinal obstruction, ii, 429
— — in lardaceous nephritis, ii, 636
— — in parenchymatous nephritis, ii, 627
— reaction of, ii, 564
— retention of—
— — in hemiparaplegia, i, 472
— — in hysteria, i, 825
— — in paraplegia, i, 441, 470
— in rheumatism, ii, 812
— sarcinæ in, ii, 583
— in smallpox, i, 229
— in suppurative nephritis, ii, 684
— in tubercular disease of kidney, ii, 692
— in variola, i, 229
— in yellow fever, i, 357
— suppression of, in cholera, i, 283, 284
— — in acute nephritis, ii, 627
— — in hysteria, ii, 669
— — in acute intestinal obstruction, ii, 429
— — non-obstructive, ii, 669
— — obstructive, ii, 670
— — in parenchymatous nephritis, ii, 627
— tests for alcohol in, i, 594
— — albumen in, ii, 595
— — biliary acids in, ii, 495
— — biliary pigment in, ii, 494
— — blood in, ii, 663
— — glycose in, ii, 708
— — tyrosin in, ii, 534
Urobilin, ii, 567
Urostealith, ii, 659
Urticaria, ii, 935
Urticaria pigmentosa, ii, 936
Uterine affections in hysteria, i, 836

V.

Vaccination, i, 240
Vaccinia, i, 239 *et seq.*
Vacuolation of brain, i, 669, 672
Valvular disease of the heart, i, 953
Varicella, i, 243 *et seq.*
Varicella-prurigo, i, 246; ii, 904
Variola, i, 227 *et seq.*
— hæmorrhagica pustulosa, i, 237
— nigra, i, 229
— sine variolis, i, 230
Variolois, i, 234
Varus, ii, 942
Venesection, *see* Bloodletting
Verruca, i, 984
Verrugas, ii, 1012
Vertebræ, *see* Spinal column.
Vertigo a stomacho læso, i, 815
— auditory, i, 813
— essential, i, 816
— labyrinthine, i, 813
— paroxysmal i, 812
— persistent, i, 816
Vesicles of the skin, ii, 868, 875
Vesicular murmur, ii, 83, 114
Vibex, ii, 1024
Villous tumour of bladder, i, 116; ii, 675
— of renal pelvis and ureters, ii, 665
Vitiligo, ii, 989, 1021
Vitiligoidea, ii, 993
Vocal fremitus, ii, 92
Voice, functional disorders of, ii, 11
Volvulus, ii, 421
Vomicæ in phthisis, ii, 206
Vomit, black, i, 356
— "coffee ground," ii, 359
Vomiting, ii, 337
— in Addison's disease, ii, 736
— in apoplexy, i, 589
— from gall-stone, ii, 501
— in Bright's disease, ii, 619, 645
— from cerebral abscess, i, 641
— from cerebral tumour, i, 613
— in cholera, i, 281
— from gastric cancer, ii, 359
— — catarrh, ii, 319, 326
— — dilatation, ii, 324
— — ulcer, ii, 349, 354
— in hydrocephalus, i, 655
— from invagination of intestine, ii, 411
— in meningitis, i, 682, 683, 689, 691, 702
— in migraine, i, 782
— from obstruction of intestine, ii, 423
— in peritonitis, ii, 464
Vomiting in phthisis, ii, 212
— in renal colic, ii, 667
— from salicylates, ii, 829
— in scarlatina, i, 210
— in smallpox i, 228, 236
— in uræmia, ii, 619
— in whooping-cough,
— in yellow fever, i, 356
— stercoraceous, ii, 424
Vox choleraica, i, 282

W.

Warts, ii, 984
Wasting palsy, i, 507
Water in spread of ague, i, 350
— — of cholera, i, 293
— — enteric fever, i, 193
Waxy liver, ii, 545
Weakmindedness, i, 858
Weber's nasal douche, i, 277
Wet packing, i, 204
Wheal, ii, 869, 928, 935
Whooping-cough, ii, 286
Woolsorters' disease, i, 370
Wrist-drop, i, 505
Writers' cramp, i, 714, 723
Wryneck, i, 716

X.

Xanthelasma, ii, 506, 993
— in migraine, i, 784
Xanthelasmoidea, ii, 936
Xanthine calculus, ii, 659
Xerodermia (of Wilson), ii, 949, 987
— pigmentosa maligna (of Kaposi), ii, 1018

Y.

Yaws, ii, 1012
Yeast plant, i, 23
Yellow fever, i, 356 *et seq.*
— simulating bilious remittent fever, i, 345,

Z.

Zinc sulphate in chorea, i, 756
Zona, ii, 932, 1025
— with facial paralysis, i, 402
Zooglœa, i, 24
Zymotic diseases, i, 22

INDEX OF AUTHORS

*** *The entries under each name are in the order of diseases followed in the text. Where no reference appears in the index it will be found in the text.*

A.

ABERCROMBIE, of Edinburgh (Diseases of the Brain, 3rd ed., 1834; Diseases of the Stomach, &c., 1828–37)
- Progressive muscular Atrophy, i, 507
- Apoplexy, i, 587 *et seq.*
- Cerebral abscess, i, 637
- Spurious hydrocephalus, i, 687
- Meningitis, i, 706 *et seq*
- Hysterical ecstacy, i, 832
- Original account of gastric ulcer, ii, 347

ABERCROMBIE, JOHN
- Sarcomata of both kidneys, ii, 695

ACKERMANN
- Chyluria, ii, 701

ACLAND, THEODORE
- Actinomycosis, i, 384

ADAMS, ROBERT, of Dublin
- Arthritis deformans, ii, 840

ADDISON, THOMAS (Works, New Syd. Soc.)
- Pungent heat of skin in pneumonia and scarlet fever, i, 46, 210; ii, 135
- Pathology of phthisis, i, 95; ii, 200
- Diarrhœa of enteric fever, i, 173
- Diphtheria, i, 259
- Phthisis in diabetes, ii, 718
- Chorea, i, 752
- Hysteria, i, 838 *et seq.*
- Peritoneal rub, ii, 164
- Pneumonic exudation, seat of, ii, 129
- Pleurisy in acute pneumonia, ii, 136
- Phthisis, pathology of, ii, 200
- Significance of melæna, ii, 340
- Uræmic coma, ii, 617
- Melasma suprarenale, ii, 733
- Idiopathic anæmia, ii, 772
- Keloid=morphœa, ii, 989

ADDISON and GULL
- Vitiligoidea=xanthelesma, ii, 506, 993

ADDISON, WILLIAM, of Brighton (Researches on Inflammation, 1843)
- Diapedesis of leucocytes, i, 53

AIRY, Sir GEORGE
- Migraine, i, 781 *et seq.*

AIRY, HUBERT
- Migraine, i, 780 *et seq.*

AITKEN (Sci. and Prac. of Medicine, 1872)
- Rubeola, i, 223
- Quarantine in plague, i, 305
- Delirium tremens, i, 889
- Relation of dysentery to malaria, ii, 394

ALDRIDGE
- Epilepsy, i, 801

ALIBERT (Monographie des Dermatoses, 1835; folio plates in his Description des Maladies de la peau, 1814)
- Arbre des Dermatoses, ii, 867
- Pityriasis nigra, ii, 907
- Causation of acne, ii, 945
- Tinea favosa, ii, 972
- Cheloid (*kéloide*), ii, 1014
- Rapid canities, ii, 1022

ALISON, of Edinburgh (Outlines of Pathology and Practice, 1844; Tweedie's Cycl.)
- Relation of tubercle to phthisis, i, 95
- Croup, ii, 25 *et seq.*

ALLBUTT, CLIFFORD
- Syphilitic cerebral arteritis, i, 556
- Athetosis, i, 576
- The optic disc and retina in cases of cerebral tumour, i, 614 *et seq.*
- Meningitis, i, 683 *et seq.*
- Auditory symptoms in meningitis, i, 704
- Muscular atrophy following hypertrophy, i, 923
- Cardiac dyspnœa, i, 931
- Diagnosis of œsophageal stricture by auscultation, ii, 315

ALLEY, of Dublin (1804)
- Eruptions from mercury, ii, 941

ALVARENGA, of Lisbon (quoted by Solly in Ziemssen's Cyclopædia)
- Black vomit in yellow fever, i, 357

ALVAREZ
- Rhinoscleroma, ii, 1008

AMMANN
- Hysteria, i, 824, 838

AMYOT
- Foot-and-mouth disease in man, i, 382

ANDERSON, MCCALL, of Glasgow (The Treatment of Diseases of the skin, 1872)
- Treatment of eczema, ii, 885
- Tinea imbricata, ii, 1012

ANDERSON, WILLIAM
- Nephrolithotomy, ii, 688

ANDRAL (Clinique Médicale), 1823–34
- Apoplectiform congestion of brain, i, 596
- Hypertrophy of brain, i, 647 *et seq.*
- Stenosis of trachea from ulcer, ii, 63
- Bronchorrhœa serosa, ii, 105
- Latent pleuritic effusion, ii, 172

ANDREW
Meningitis, i, 703
ANNESLEY (Researches into the Causes, &c., of the Diseases of India, 1841)
Aspect of patient in cholera, i, 281
Tropical dysentery, ii, 393
Hepatic abscess and dysentery, ii, 514
ANSTIE (Stimulants and narcotics, 1864; Neuralgia, 1871; Reynolds' System; Practitioner)
Neuralgia, i, 418 *et seq.*
Sciatica, i, 425 *et seq.*
Rachialgia, i, 476 *et seq.*
Compression-paraplegia, i, 482
Paralysis from lead, i, 507
Test for alcohol in urine, i, 594, *note*
Cerebral abscess, i, 641
Chorea, i, 741
Migraine, i, 783 *et seq.*
Hysteria, i, 825 *et seq.*
Hypochondriasis, i, 845 *et seq.*
Alcoholism, i, 883 *et seq.*
Delirium tremens, i, 888 *et seq.*
Angina pectoris, i, 911 *et seq.*
Gastralgia, ii, 332
Peritonitis from sewer-gas, ii, 468
Cirrhosis of liver and alcohol, ii, 525
Zona, ii, 1027
ARAGO
Migraine, i, 781
ARAN
Progressive muscular atrophy, i, 507
Tetany, i, 740
Paracentesis of pericardium, i, 948
Paralysis of the diaphragm, ii, 71
ARETÆUS
Tetanus, i, 759
Thorax of phthisis, ii, 234
Elephantiasis, ii, 991
Leprosy, ii, 1009
ARISTOTLE
Hydrophobia, i, 767
Theory of hysteria, i, 836
Arthritis in horses, ii, 846, *footnote*
ARNOLD, JULIUS, of Heidelberg
Emigration of leucocytes, i, 54
Origin of giant-cells, i, 82
Relation of tubercle to inflammation, i, 83
ASHBY, of Manchester
Synovitis and carditis in scarlatina, i, 214
ASHWELL (Practical Treatise on Diseases peculiar to Women, 1844)
Chlorosis, ii, 769
AUENBRÜGGER, of Vienna (Inventum novum ex percussione thoracis humani abstrusos interni pectoris morbos detegendi, 1761)
Percussion of thorax, ii, 75
AUSPITZ (Archiv, *now* Vierteljahresschrift, für Dermatologie und Syphilis; System der Hautkrankh., 1881; Ziemssen's Hdbh.
Histology of pock of smallpox, i, 231
Histology of syphilitic chancre, i, 309
Pityriasis rubra, ii, 908
Method of applying chrysarobin, ii, 915
Histology of lupus, ii, 996
Classification of deep dermatitis, ii, 1003
AUSTIN, T. J. (Practical account of General Paralysis, 1859)
General paralysis of the insane, i, 664
AVERY
Early attempt at laryngoscopy, ii, 1
AXEL-KEY
Myoma of skin, ii, 1014

B.

BABES, of Buda-Pesth (Ziemssen's Hdbh.)
Rhinoscleroma, ii, 1008
Histology of cheloid, ii, 1015
BABINGTON, B. G. (the younger)
Attempt at laryngoscopy, ii, 1
Epistaxis, ii, 282
Odour of diabetes, ii, 716
The blood in Bright's disease, ii, 622
BACON, MACKENZIE
Hydrocephalus of adults, i, 656
General paralysis of the insane, i, 666
BADHAM (Essay on Bronchitis, 1814)
Bronchitis, ii, 94
BAIERLACHER
Peripheral facial paralysis, i, 405
BAILLARGER (Notes to transl. of Griesinger)
General paralysis of the insane, i, 671
Hæmatoma of the dura mater, i, 710
Hereditary insanity, i, 748
BAILLIE, MATTHEW (Morbid Anatomy, 1797)
Hydrocephalus of childhood, i, 654
BAILLON (Opera Ballonii, *ob. ante*, 1600)
Pulsating empyema, ii, 177
Rheumatism, ii, 810
BAKER, Sir GEORGE (Essay on the Epidemical Colic of Devonshire, 1767)
Influenza, ii, 275
Lead-colic, ii, 371
BAKER, MORRANT (St Barth. Hosp. Rep.; Trans. Internat. Med. Congr., 1881, &c.)
Heredity of tumours, i, 122
Nephrotomy, ii, 688
Prurigo, ii, 906, *footnote*
Urticaria pigmentosa, ii, 936
Cheloid acne, ii, 956
Modified Coster's paste, ii, 968
Case of sclerodermia (?), ii, 990
BALFOUR, G. W., of Edinburgh (Clin. Lec. on Dis. of Heart and Aorta, 1876)
Irregular pulse, i, 899 *et seq.*
Angina pectoris, i, 911 *et seq.*
Anæmic murmurs, i, 969
BALLARD EDWARD (On a Localised Outbreak of Fever in Islington, 1871)
Epidemic of enterica from milk, i, 196
BALY (Gulstonian Lec., 1847)
Prevalence of phthisis at Millbank Penitentiary, ii, 236
Anatomy of dysentery, ii, 389, 394
BAMBERGER, of Vienna
Function-spasm, i, 721
Hysteria, i, 829
Gastric catarrh, ii, 322
Phlegmonous gastritis, ii, 324

BANCROFT, of Brisbane
Filaria sanguinis hominis, ii, 459
VON BÄRENSPRUNG, of Berlin, (Beiträge zur Anat. u. Phys. d. menschl. Haut, 1848)
Clinical thermometry, i, 37
Histology of pock of variola, i, 232
Parencentesis for pneumothorax, ii, 199
Zona, ii, 1027 *et seq.*
BARKER, A. E. J.
Nephrotomy, ii, 680, 688
BARKER, FORDYCE, of New York
Diarrhœa in puerperal peritonitis, ii, 464
BARKER, F., and CHEYNE, J. (Account of the Fever lately Epidemical in Ireland, 1821)
Contagion of typhus, i, 142
BARLOW, G. H. (Guy's Hosp. Reports)
Urine in intestinal obstruction, ii, 429
Renal pulse, ii, 611
BARLOW, H. C.
Enteric fever, i, 164
BARLOW, THOMAS
Malformation of larynx, ii, 58
Raynaud's disease, ii, 590
Rheumatic nodules, ii, 815
Acute rickets, ii, 855, *footnote*
Craniotabes and syphilis, ii, 859
Erythema and rheumatism, ii, 935
BARTELS, of Kiel (Ziemssen's Cyclopædia, Volkmann's Sammlung, &c.)
Cholera, i, 285 *et seq.*
Atelectasis of lungs, ii, 103
Fatal syncope from left pleuritic effusion, ii, 173
Albuminuria, ii, 599
Tube-casts, ii, 604
Bright's disease, ii, 607 *et seq.*
Theory of uræmia, ii, 620
Diaphoresis a cause of uræmia, ii, 623
Lardaceous disease in congenital syphilis, ii, 632
Lardaceous kidney, ii, 635 *et seq.*
Cirrhosis of kidneys, ii, 643 *et seq.*
Polyuria after obstructed ureter, ii, 671
BARTHOLIN, THOMAS, of Copenhagen (Hist. Anat. et. Med., 1654)
Plastic bronchitis, ii, 125
BARWELL
Nephrolithotomy, ii, 688
VON BASCH
Histology of pock of variola, i, 231
BASEDOW (Caspar's Wochenscrift, 1840 and 1848)
Exophthalmic goitre, i, 1009
BASSEREAU (Affections de la Peau symptomatiques de la Syphilis, 1852)
Relations of venereal sores to constitutional symptoms, i, 309
BASTIAN, CHARLTON (Paralysis from Brain Disease, 1875; Reynolds' System)
Crossed amblyopia, i, 574
Aphasia, i, 577 *et seq.*
Amnesic agraphia, i, 584
Cirrhosis of the lung, ii, 154 *et seq.*
BATEMAN (On Aphasia, 1874)
Aphasia, i, 577
BATEMAN, THOMAS (Practical Synopsis of Cutaneous Diseases, 1813)
Classification of diseases of skin, ii, 866
Prurigo, ii, 903
Pityriasis rubra, ii, 907, *et seq.*
Erythema nodosum, ii, 934
Molluscum contagiosum ii, 950
Sycosis, ii, 953
Porrigo lupinosa, ii, 971
Leprosy, ii, 1009
Frambœsia, ii, 1012
Fibroma molluscum, ii, 1013
BATEMAN, of Norwich
Sarcinæ in urine, ii, 583
BAUER (Ziemssen's Cyclopædia)
Tetanus, i, 759 *et seq.*
BAUMGARTEN
Myelitis, i, 450
BÄUMLER, of Freiburg (Ziemssen's Cyclopædia; Clinical Transactions, &c.)
Clinical thermometry, i, 38
Syphilis, i, 308 *et seq.*
Idiopathic pericarditis, i, 942
Paralysis of larynx from pressure on the vagus nerve, ii, 6
Hæmoptysis in phthisis, ii, 217
Sclerostomum duodenale, ii, 453
BAUWINKEL
Disseminated sclerosis, i, 551
BAXTER, E. B. (Reports to Privy Council, &c.)
Investigations on disinfectants, i, 35
Effect of fever on blood-discs, i, 48
General exfoliative dermatitis, ii, 908
Pemphigus foliaceus, ii, 920
BAYFORD, of Lewes
Dysphagia lusoria, ii, 313
BAYLE, GASPARD L. (Recherches sur la Phthisie pulmonaire, 1810)
Grey granulations, i, 80
Forms of phthisis, i, 80
Œdema glottidis, ii, 46
BAZIN (Leçons sur les Affections Cutanées, 1851–62)
Hydroa, ii, 922
Acne varioloformis, ii, 947, 952
Sycosis, ii, 955
Onychomycosis, ii, 971
Alopecia areata, ii, 975
Teigne pelade, ii, 975
Scrofulides, ii, 999
Cheloid, ii, 1015
BAZIRE (Notes to translation of Trousseau)
Locomotor ataxy, i, 532
Epilepsy, i, 794 *et seq.*
BEACH, FLETCHER
Hypertrophy of the brain, i, 648
Cretinism, i, 879
BEARD and ROCKWELL, of New York
Neurasthenia spinalis, i, 452
BEALE, LIONEL (Kidney Diseases, &c., 1869 Disease Germs, 1870)
Dumb-bell crystals in kidney, ii, 570
Urinary calculi, ii, 657 *et seq.*
Hæmaturia, ii, 663

BEAUMONT, WILLIAM, of Plattsburgh, U.S.A. (Exp. and Obs. on the Gastric Juice, 1833)
Gastric catarrh, ii, 320
BECK, MARCUS (Reynolds' System)
Consecutive Bright's disease, ii, 648
Nephrolithotomy, ii, 688
BECQUET
Moveable kidney, ii, 706
BEDNAR (Lehrb. d. Kinderkrankhn., 1865)
Laryngismus stridulus, ii, 14
BEHREND (Die Hautkrankheiten, 1879)
Medicinal eruptions, ii, 940
BEIGEL
Trichoptylose, ii, 977
BELL, Sir CHARLES
Peripheral facial paralysis, i, 401
Progressive muscular atrophy, i, 507
Thomsen's disease, i, 734
BELL, of Bradford
Woolsorters' disease, i, 370 *et seq.*
BENEDIKT (Nervenpathologie u. Electrotherapie 1874)
Oculo-motor paralysis, i, 413
Pseudo-hypertrophic paralysis, i, 529
Locomotor ataxy, i, 533
Choked optic disc, i, 615
Writers' cramp, i, 723 *et seq.*
Chorea, i, 741
BENNETT, HENRY
Hysteria, i, 836
BENNETT, HUGHES, of Edinburgh (Prin. and Prac. of Med., 1859; 'Practitioner')
Treatment of acute pneumonia, ii, 144
Cod-liver oil in phthisis, ii, 260
Leuchæmia, ii, 753
Ointments and lotions, ii, 835
BENNETT, Sir RISDON (Lumleian Lectures on Intrathoracic Growths, 1872)
Mediastinal growths, ii, 81 *et seq.*
BERGEON (Théorie des Bruits de la Resp., 1869)
Origin of respiratory sounds, ii, 85
BERNARD, CLAUDE (Nouvelle fonction du foie, 1853; Leçons sur le diabète, 1877)
Glycogenic function of the liver, ii, 721
BESNIER
Myoma of skin, ii, 1014
BEUMER
Hypertrophy of one kidney from atrophy of the other, ii, 673
Disease of a single kidney, ii, 673
BICHAT (Rech. sur la vie et la mort, 1805)
Modes of death, i, 8
Rapid canities, ii, 1022
BIDDER, of Dorpat
Columnar carcinoma, i, 119
BIDDER and SCHMIDT (Die Verdauungssäfte, 1852)
Fatty stools in jaundice, ii, 506
BIDENKAP, of Christiania
Syphilis, i, 309
BIERMER (Clin. Lect., New Syd. Soc.)
Enteric fever, i, 190
Cerebral abscess, i, 637 *et seq.*
Bronchiectasis, ii, 117
Plastic bronchitis, ii, 125 *et seq.*
Progressive pernicious anæmia, ii, 772
Retinal hæmorrhage in anæmia, ii, 776
BIESIADECKI, of Cracow
Erysipelas, i, 254
Histology of syphilitic lesions, i, 308
Histology of bullæ, ii, 919
BIETT (Abrégé des Maladies de la peau, by Cazenave and Schedel, 1838)
Syphilides, ii, 866
Nature of sycosis, ii, 955
Lupus erythematosus, ii, 1006 *et seq.*
Erythème centrifuge, ii, 1006
Cheloid, ii, 1015
BILLROTH, of Vienna (Lectures on Surgical Pathology, translation New Syd. Soc.)
Causation of pyrexia by injection of pus or decomposing matter, i, 44
Tumours, i, 97 *et seq.*
Erysipelas, i, 255
Pseudo-hypertrophic paralysis, i, 526
Extirpation of larynx, ii, 57
Excision of pylorus, ii, 366
BILHARZ
Endemic hæmaturia, ii, 702 *et seq.*
BIRCH-HIRSCHFELD (Ziemssen's Handbh.)
Spirochæta Obermeieri, i, 159
Tuberculosis after enteric fever, ii, 270
Lardaceous disease of mesenteric glands, ii, 631
BIRD, GOLDING (Urinary Deposits, 1853, Guy's Hosp. Reports)
Spasmodic torticollis, i, 716
Colouring matter in urine, ii, 575
Crystals of calcic oxalate, ii, 575
BIRD, GOLDING, C. H.
Electrolytic treatment of bedsore, i, 469
Case of chronic tetanus, i, 764
BLACKALL (Observations on the Nature and Cure of Dropsies, 1813)
Albuminuria in renal disease, ii, 591
BLACKLEY (Experimental Researches on Catarrhus æstivus, 1873)
Hay fever, ii, 284
BLANE, Sir GILBERT (Croonian Lectures on Muscular Motion, 1790)
Tetanus, i, 759
BLANDFORD (Lectures on Insanity, 1871)
General paralysis of the insane, i, 664
Hysterical insanity, i, 833
BLOXALL
Epidemic enterica at Sherborne, i, 196
BOECK, CÆSAR
Erythema and rheumatism, ii, 935
Lupus erythem. disseminatus, ii, 1008
Leprosy, ii, 1011
BOECK and DANIELSSEN, of Christiania
Leprosy, ii, 1011
BOERHAAVE, of Leyden
Clinical thermometry, i, 37
BOLL, FRANZ (1876)
Traumatic origin of cancer, i, 124
DU BOIS REYMOND, of Berlin
Migraine, i, 781 *et seq.*
BOLLINGER, of Munich (Ziemssen's Cycl.)
Bovine tuberculosis, i, 90
Anthrax, i, 369 *et seq.*
Glanders, i, 376 *et seq.*

Bollinger (*continued*)—
Actinomycosis, i, 382
Hydrophobia, i, 767 *et seq.*
Bonnet and Poincaré
General paralysis of the insane, i, 669
Bostock, John (1819)
Hay-fever, ii, 284
Böttcher
Local production of leucocytes, i, 55
Cerebral abscess, i, 637 *et seq.*
Bottini
Extirpation of larynx, ii, 58
Bouchard (De la Pathogénie des Hémorrhagies, 1869, &c.)
Cerebral hæmorrhage, i, 563 *et seq.*
Late rigidity, i, 574
Ulcer of tongue in pertussis, ii, 287
Bouchardat (Du Diabète Sucré, 1852)
Gluten bread in diabetes, ii, 730
Bouchut (Leçons cliniques sur les Maladies de l'Enfance, 1858)
Diphtheria, i, 273
Relation of craniotabes to rickets and congenital syphilis, ii, 859
Boudet
Causation of respiratory sounds, ii, 85
Boudin (Traité des fièvres intermittentes, 1842)
Ague, i, 350
Bouillaud (Traité de l'Encephalite, 1825; Maladies de la Cœur, 1835)
Localisation of language, i, 580
Bruit de diable, ii, 751
Bouillon-Lagrange
Hæmatoma of dura mater, i, 712
Bourneville (Editor of Charcot's Lectures)
Disseminated sclerosis, i, 547
Apoplexy, i, 585
Paralysis agitans, i, 730
Bowditch, of Boston (Consumption in New England, 1868; Thoracentesis, 1870)
Paracentesis thoracis, ii, 184
Soil in relation to phthisis, ii, 247
Bowles, of Folkestone
Relief of stertor in apoplexy, i, 586
Braidwood
Organisms in measles, i, 217
Bramwell, Byron
Arsenic in idiopathic anæmia, i, 778
Brand, of Stettin (Wasserbehandlung d. typhösen Fiebers, 1877)
Baths in enteric fever, i, 203
Bräuell
Bacillus anthracis, i, 368
Braun, Julius (Lehrb. d. Balneotherapie, 1873, transl. by Dr Weber)
Chronic spinal meningitis, i, 495
Mineral waters in bronchitis, ii, 124
Brenner, Rudolf (Electrotherapie, 1868)
Peripheral facial paralysis, i, 405
Bretonneau (Papers on Diphtheria, 1821–26, translation for New Syd. Soc., &c.)
Enteric fever, i, 164
Diphtheria, i, 259 *et seq.*
Croup and diphtheria, ii, 218 *et seq.*
Epilepsy, i, 811
Bricheteau (Traité de l'Hydrocephale aiguë des Enfans, 1829)
Tubercular meningitis, i, 675
Chorea, i, 752
Bright (Guy's Hospital Reports; Cases and Obs. illust. of Renal Disease, 1836)
Melanæmia, i, 346
Cerebral localisation, i, 621 *et seq.*
Hydrocephalus of childhood, i, 652
Spasmodic torticollis, i, 716
Chorea, i, 752 *et seq.*
Tetanus, i, 767
Migraine, i, 791
Syphilitic stenosis of trachea, ii, 63
Atelectasis of lungs, ii, 101
Fatty stools in jaundice, ii, 505
Large liver in early cirrhosis, ii, 524
Acute atrophy of liver, ii, 531
Abscess of spleen, ii, 591
Renal disease, ii, 743 *et seq.*
Brinton (Diseases of the Stomach, 1859; Intestinal Obstruction, 1867)
Ulcer of stomach, ii, 348 *et seq.*
Cancer of stomach, ii, 354 *et seq.*
Invagination of intestine, ii, 407
Pain in intestinal obstruction, ii, 423
Briscoe, of Chippenham
Foot-and-mouth disease, i, 382
Bristowe (Theory and Practice of Medicine; St Thomas's Hospital Reports, &c.)
Definition of disease, i, 4
Rötheln, i, 223 *et seq.*
Smallpox, i, 227
Diarrhœa during cholera, i, 299
Apoplexy, i, 603
Opium in meningitis, i, 706
Chorea, i, 746 *et seq.*
Tracheal stenosis from goitre, ii, 61
Paroxysmal dyspnœa from stenosis of trachea or bronchi, ii, 66
Physical examination of thorax, ii, 79
Subtympanitic note in pleuritic effusion, ii, 79, 170
Fungus in phthisical vomicæ, ii, 208
Obsolescence of miliary tubercle, ii, 269
Contagion of pertussis, ii, 289
Tachycardia, ii, 988
Briquet (Traité de l'Hystérie, 1859)
Hysteria, i, 825
Broadbent (Med.-Chir. Tr.; Croonian Lect.)
Alcoholic paralysis, i, 464
Hemiplegia, i, 568 *et seq.*
Aphasia, i, 577 *et seq.*
Cerebral localisation, i, 619
Chorea, i, 747 *et seq.*
Pulse, i, 898
Broca, Paul
Aphasia, i, 577 *et seq.*
Brockman
Paralysis from chronic plumbism, i, 507
Brodie, Sir Benjamin (Dis. of Joints, 1818–50; Dis. Urinary Organs, 1832–49)
Urine after spinal injury, i, 443
Uric acid a cause of hæmaturia, ii, 666
Nephrolithotomy, ii, 687
Syphilitic paraplegia, i, 464

Brodie (*continued*)—
Malformation of hip-joints, i, 528
Gonorrhœal synovitis, ii, 836
Brodhurst (Reynolds' System)
Gonorrhœal synovitis, ii, 835 *et seq.*
Broussais (Leçons sur les Phlegmasies gastriques, 1823)
Enteric fever, i, 164
Brown, Crichton (West Riding Asylum Reports, &c.)
Cerebral tubercle, i, 606
General paralysis of the insane, i, 671
Epilepsy, i, 798 *et seq.*
Brown, Crum (Journal of Anatomy, 1874)
Function of semicircular canals, i, 813
Brown, Thomas, of Glasgow, 1825
Rachialgia, i, 473
Brown-Séquard
Peripheral facial paralysis, i, 404
Sensory tracts of spinal cord, i, 43
Anæmia of spinal cord, i, 455
Paraplegia, i, 455 *et seq.*
Decussation of sensory and motor tracts of spinal cord, i, 471 *et seq.*
Spinal caries, i, 479
Hemiplegia, i, 567
Paralysis agitans, i, 734
Epilepsy, i, 803 *et seq.*
Rapid canities, ii, 1022
Brücke, of Vienna
Case of stomach cough, ii, 73
Brunniche
Typus inversus in tuberculosis, ii, 265
Brüns
Elephantiasis neuromatosa, ii, 1017
Brunton, Lauder
Case of atrophy, i, 516, *footnote*
Angina pectoris, i, 911
Glycogenic function of liver, ii, 723
Bryant
Tetanus, i, 761
Trephining in epilepsy, i, 806
Results of colotomy, ii, 428
Bryce (Inoculation of Smallpox, 1809)
Varicella, i, 244
Bryson (Report on the Fever in H.M.S. "Bann" on the Coast of Africa, 1824; Ophthalmic Hospital Reports, 1859)
Yellow fever, i, 360
Night blindness in scurvy, ii, 782
Buchanan, George (Reports to Privy Council; Reynolds' System)
Typhus, i, 134 *et seq.*
Enteric fever, i, 196 *et seq.*
Yellow fever, i, 359
Soil in relation to phthisis, ii, 248
Summer diarrhœa, ii, 380
Buchner
Microzymes, i, 25
Anthrax, i, 369
Budd, George (Diseases of Liver, 1845–57)
Acute atrophy of liver, ii, 532, 537
Abscess of liver, ii, 514 *et seq.*
Budd, William, of Bristol (Typhoid Fever, 1873, &c.)
Enteric fever, i, 188 *et seq.*
Cholera, i, 291
Budd (*continued*)—
Anthrax, i, 372
Paraplegia, i, 438 *et seq.*
Buhl, of Munich (Lungenentzündung, Tuberculose u. Schwindsucht, 1872, &c.)
Tubercle, i, 88 *et seq.*
Soil-water theory applied to enteric fever, i, 199
Diphtheria, i, 260 *et seq.*
Cholera, i, 284 *et seq.*
Anthrax, i, 369 *et seq.*
Bulkley, of New York
Herpes gestationis, ii, 934
Cubebs rash, ii, 939
Acne varioliformis, ii, 947
Burger
Pertussis, ii, 291
Burkart
Miliary tuberculosis, ii, 263 *et seq.*
Burton
"Blue line" of plumbism, ii, 372
Bury, of Manchester
Mollities ossium in an infant, ii, 862
Busch
Spasmodic torticollis, i, 720
Tubercle of choroid, ii, 267
Busk, Geo.
Erysipelas, i, 252
Diarrhœa, ii, 173, *footnote*
Butlin
Rupture of diaphragm simulating pneumothorax, ii, 196
Nephrolithotomy, ii, 688
Histology of pachydermia, ii, 992
Buzzard (Reynolds' System, &c.)
Galvanism in neuralgia, i, 427
Peripheral neuritis, i, 430 *et seq.*
Paralysis agitans, i, 731
Thomsen's disease, i, 734 *et seq.*
Diphtheritic paralysis, i, 274–275, *note*
Syphilitic paraplegia, i, 464
Cerebral localisation, i, 626
Syphilis of brain, i, 631
Charcot's joint disease, ii, 848
Scurvy, ii, 782 *et seq.*

C.

Cadge, of Norwich
Local prevalence of calculi, ii, 661
Callender
Granular degeneration of diaphragm in chronic bronchitis, ii, 105
Calmeil (La Paralysie chez les Alienés, 1826)
General paralysis of the insane, i, 663
Migraine, i, 790
Epilepsy, i, 808
Campbell, H. G.
Statistics of diabetes, ii, 721
Carré (Nouvelles recherches sur l'Ataxie, &c., 1865)
Locomotor ataxy, i, 542
Carswell, Sir Robert (Pathological Anatomy, plates, 1838)
Tubercle in phthisis, ii, 205 *et seq.*

CARTER, VANDYKE, of Bombay (On Leprosy and Elephantiasis, 1874, &c.)
Relapsing fever in India, i, 153, 162
Urinary calculi, ii, 657
Leprosy in India, ii, 1010, 1012
Leucodermia and leprosy, ii, 1022
CAVAFY (St George's Hosp. Rep.; Clin. Tr., &c.)
Leucocytes in leuchæmia, ii, 756
Urticaria pigmentosa, ii, 937
Eruptions from salicylic acid, ii, 941
Alcoholic and ethereal solution in ringworm, ii, 968
Case of lupus disseminatus, ii, 1008
Rhinoscleroma, ii, 1008
CAYLEY (Croonian Lectures, 1880, &c.)
Enteric fever, i, 190 *et seq.*
Fatal case of injecting iodine into the pleura, ii, 187
Gastric tube casts, ii, 320
Simple atrophy of liver, ii, 529
CAZEAUX (Art des Accouchements, 1845)
Pemphigus neonatorum, i, 333
CAZENAVE (Leçons sur les Maladies de la Peau, 1843)
Erysipelas, i, 255
Pemphigus foliaceus, ii, 921
Species of lupus, ii, 999
Lupus erythematosus, ii, 1006 *et seq.*
CECCARELLI, of Turin
Moveable kidney, ii, 707
CEELEY
Relation of variola and vaccinia, i, 242
CELSUS, AUR. CORNELIUS (De Medicina libri octo)
Ague, i, 339
Hydrophobia, i, 767
Acne, ii, 942
Alopecia, ii, 974
Elephantiasis, ii, 991, 1009
Vitiligo, ii, 1021
CHAMBERS, T. K. (Reynolds' System; The Indigestions, 1867)
Epilepsy simulating catalepsy, i, 793
Hysterical catalepsy, i, 830 *et seq.*
Dyspepsia, ii, 328 *et seq.*
Pyrosis, ii, 336
Habitual vomiting, ii, 337
Hæmatemesis, ii, 340 *et seq.*
CHAMPNEYS
Chloasma in uterine affections, ii, 1024
CHANOTIN
Endemic hæmaturia, ii, 702
CHAPMAN
Spinal icebag in epilepsy, i, 812
CHARCOT (Leçons sur les Maladies des Vieillards, 1868; sur les Mal. de Syst. Nerveux, 1872, transl. New Syd. Soc.; sur les Mal. du Foie et des Reins, 1877)
Paralysis of the fifth nerve, i, 415
Ankle-clonus, i, 440
Paraplegia, i, 444 *et seq.*
Anæmia of spinal cord, i, 454
Hæmorrhage into spinal cord, i, 465.
Compression-paraplegia, i, 478 *et seq.*
Spinal caries, i, 480
Meningeal tumours, i, 481
CHARCOT (*continued*)—
Spastic paraplegia, i, 488 *et seq.*
Acute atrophic paralysis, i, 498 *et seq.*
Progressive musc. atrophy, i, 509 *et seq.*
Lateral sclerosis, i, 513, 575 *et seq.*
Cervical pachymeningitis, i, 513
Bulbar paralysis, i, 520 *et seq.*
Pseudo-hypertrophic paralysis, i, 528
Locomotor ataxy, i, 535 *et seq.*
Disseminated sclerosis, i, 547 *et seq.*
Cerebral softening, i, 558
Cerebral hæmorrhage, i, 561 *et seq.*
Hemiplegia, i, 568 *et seq.*
Hemiopia, i, 574
Paralysis agitans, i, 729, *et seq.*
Epilepsy, i, 798
Paroxysmal vertigo, i, 813 *et seq.*
Hysteria, i, 823 *et seq.*
Cirrhosis of the lung, ii, 153
Hypertrophic cirrhosis of liver, ii, 528
Hysterical ischuria, ii, 669
Muscular tremor in uræmia, ii, 618
Arthropathie ataxique, ii, 848
Zona, ii, 1028 *et seq.*
CHARCOT and BOUCHARD
Miliary aneurysms a cause of cerebral hæmorrhage, i, 564
CHARLES, T. C.
Analysis of tissues in myxœdema, i, 882
CHAUSIT (Tr. élementaire des Maladies de la Peau après Cazenave, 1853)
Herpes gestationis, ii, 934
CHAUVEAU, of Lyons
Contagion, i, 22
Artificial production of chorea, i, 747
Causation of respiratory sounds, ii, 85
CHEADLE
Rubeola, i, 226
Acute rickets, ii, 855, *footnote*
CHESELDEN, 1688—1752
Plastic bronchitis, ii, 125
CHEVERS, NORMAN
Cause of dysentery, ii, 395
CHEYNE, JOHN, of Dublin (Essays on Diseases of Children, 1801; Hydrocephalus, 1808; Apoplexy and Lethargy, 1812)
Croup, ii, 18 *et seq.*
Plastic bronchitis, ii, 125
CHEYNE, JOHN and STOKES
Peculiar respiratory rhythm, i, 586, 974
CHEYNE, JOHN and BARKER (Fever, Tweedie's Library of Medicine, 1840)
Typhus, i, 142
CHEYNE, WATSON
Treatment of diphtheria, i, 278
Micrococci in purpura, ii, 790, *footnote*
CHIARI
Histology of sclerodermia, ii, 724
CHOMEL (Leçons de Clinique Médicale, 1834)
Lesions in enteric fever, i, 168
Erysipelas, i, 252
Peripheral neuritis, i, 430
CHOSSAT (Recherches sur l'inanition, 1843)
Artificial rickets, ii, 858
CHRISTISON, Sir ROBERT, of Edinburgh
Relapsing fever, i, 157

CHRISTISON (*continued*)—
Alcoholism, i, 889
Pigment in miners' phthisis, ii, 241
CHURCH (St Barth. Hosp. Reports)
Cerebral aneurysm, i, 560
Migraine, i, 784
Statistics of acute rheumatism, ii, 814
Xanthoma, ii, 994
CLARK, Sir ANDREW
Renal inadequacy, ii, 565
Treatment of hay fever, i, 285
CLARKE, FAIRLIE
Paralysis of hypoglossal nerve, i, 414
CLARKE, LOCKHART (Holmes's Surgery)
Origin of hypoglossal nerve, i, 522
Pseudo-hypertrophic paralysis, i, 529
General paralysis of the insane, i, 669
Atrophy of the brain, i, 672
Tetanus, i, 762
Origin of spinal accessory nerve, ii, 6
CLERC
Non-infecting chancre, i, 310 *et seq.*
CLOUSTON
Causation of dysentery, ii, 394, 395
COATS, of Glasgow (Manual of Pathology, 1883)
Histology of tetanus, i, 762
— of hydrophobia, i, 773
Calcareous disease of the heart, i, 941
COBBOLD (Entozoa, 1864)
Intestinal worms, ii, 445 *et seq.*
Filaria sanguinis hominis, ii, 459
Bilharzia hæmatobia, ii, 702
COHN, FERDINAND, of Breslau (Beiträge zur Biologie der Pflanzen, 1875)
Microphytes, i, 24
— in diphtheria, i, 263
Bacillus anthracis, i, 368
COHNHEIM (Vorlesungen über allgemeine Pathologie, 1877)
Pyrexia, i, 42 *et seq.*
Inflammation, i, 52 *et seq.*
Tubercle, i, 82 *et seq.*
Tumours, i, 100
Cholera, i, 283
Pseudo-hypertrophic paralysis, i, 526
Collapse of lung from stenosis of bronchus, ii, 64
Dyspnœa, ii, 70
Emphysema of the lungs, ii, 111
Pleurisy, ii, 167
Experimental pneumothorax, ii, 193
Tubercle of choroid, ii, 267
Hypertrophy of heart, i, 923
Uric acid deposits in kidneys of newborn children, ii, 573
Experiments on septic cystitis, ii, 580
Albuminuria, ii, 598
Theory of renal dropsy, ii, 607
The heart and pulse in Bright's disease, ii, 612
Histology of Bright's disease, ii, 615
Lardaceous disease of kidneys ii, 634
Changes in kidney after ligature of ureter, ii, 671
Double hydronephrosis, ii, 675
COHNHEIM (*continued*)—
Hydronephrosis from malposition of ureter, ii, 677
Anæmia myelogenica, ii, 757, 766
COLLES, of Dublin (Pract. Obs. on the Venereal Disease, 1837)
Law of congenital syphilis, i, 330
COLLEY, DAVIES
Anthrax, i, 372
Paracentesis thoracis ii, 184
Gonorrhœal arthritis, ii, 839
Molluscum contagiosum, ii, 952
COLLIE, ALEXANDER
Enteric fever, i, 189 *et seq.*
COLLINS, ROBERT, of Dublin
Epidemic meningitis, i, 693 *et seq.*
COOPER, Sir ASTLEY
Experimental anæmia of cord, i, 453
Gonorrhœal synovitis, ii, 835
COPLAND (Dict. of Pract. Med., 1844–58)
Rubeola, i, 223 *et seq.*
Stomach-cough, ii, 73
Gastric regurgitation, ii, 335
CORMACK, Sir JOHN
Croup and diphtheria, ii, 18
CORNIL
Erysipelas faucium, i, 254
Acute atrophic paralysis, i, 500
Chronic atrophic paralysis, i, 504
Choked optic disc, i, 614
Rhinoscleroma, ii, 1008
Bacillus lepræ, ii, 1010
CORNIL and RANVIER (Manuel d'histologie pathologique, 1869–76)
Tumours, i, 114 *et seq.*
Tuberculosis of bones, ii, 266
Lardaceous disease preceded by nephritis, ii, 634
Histology of gouty joints, ii, 799
CORRIGAN, Sir DOMINIC, of Dublin
Cirrhosis of the lung, ii, 152 *et seq.*
Origin of bruit de souffle, i, 957
Pulse, i, 966
CORVISART (Transl. of Auenbrügger, 1808; Essai sur les Maladies du Cœur, 1811)
Tetany, i, 736
Physical examination of thorax, ii, 76
DA COSTA, of Philadelphia (Medical Diagnosis, 1870)
Irritable heart, i, 904
COSTER
Iodine paste in ringworm, ii, 968
COTHOLENDY, of Réunion
Dengue, i, 364 *et seq.*
COTTON
Frequent pulse, i, 898
COUPLAND
Acute myelitis in glanders, i, 380
CRAMPTON, Sir PHILIP, of Dublin
Enormous pleuritic effusion, ii, 165
CREIGHTON, CHAS. (Phys. and Path. of the Breast, 1878; Bovine Tuberculosis, 1881; Journal of Micros. Science)
Tubercle and Perlsucht, i, 90
Cancer, i, 124
Micro-organisms, i, 262

CROCKER, RADCLIFFE
Bullous erythema, ii, 933
Molluscum contagiosum, ii, 952
Goa powder in ringworm, ii, 968
Histology of morphœa, ii, 990, *footnote*
Cases of xerodermia maligna, ii, 1019
Neurotic affections of the skin, ii, 1029
CROFT
Hydronephrosis, ii, 676 *et seq.*
Traumatic stricture of ureter, ii, 676
CROOKSHANK (Practical Bacteriology, 1886)
Microzoa in ague; 352, *footnote*
CRUVEILHIER (Anatomie Pathologique, 1864)
Pyæmia, i, 70
Compression-paraplegia, i, 478 *et seq.*
Hydatid of spine, i, 481
Progressive muscular atrophy, i, 507
Locomotor ataxy, i, 532
Disseminated sclerosis, i, 547
Secondary lateral sclerosis, i, 575
Hydrocephalus of childhood, i, 651
Induration ardoise, ii, 152
Point de côté in pleurisy, ii, 171
Gastric ulcer, ii, 347
Displacement of kidney, ii, 705
CULLEN, WM., of Edinburgh (Synopsis Nosologiæ, 1772)
Influenza a contagious catarrh, ii, 278
Abortive whooping-cough, ii, 286
Pyrosis, ii, 336
CURRIE, JAMES, of Liverpool (Medical Reports, 4th Ed., 1805)
Cold bathing in pyrexia, i, 203
CUNNINGHAM
Cholera, i, 291
CURLING
Absence of thyroid in cretinism, i, 878
CURSCHMANN (Ziemssen's Cyclopædia)
Smallpox, i, 227 *et seq.*
CYON (Die Lehre v. d. Tabes-dorsalis, 1867)
Locomotor ataxy, i, 535 *et seq.*
CZERMAK (Der Kehlkopfspiegel, 1860)
The laryngoscope, ii, 1
CZERNICKI
Effect of pneumothorax on phthisis, ii, 198
CZERNY, of Freiburg (Trans. Internat. Med. Congr., 1881)
Nephrectomy, ii, 674, 680, 687
Nephrotomy, ii, 688
Moveable kidney, ii, 706

D.

DALDY (Disease of the Right Side of the Heart, 1866)
Cardiac dyspnœa, i, 932
DANCE
Tetany, i, 736
DARWIN, FRANCIS
Intravesical ganglia, i, 443, footnote
DAVAINE (Traité des Entozoaires, 1877)
Bacillus anthracis, i, 35, 368, *et seq.*
Distribution of hydatid cysts, ii, 557
Hydatid of kidney, ii, 698
DAVIES, HERBERT
Blisters in acute rheumatism, ii, 823
DAVY, JOHN
Air of pneumothorax, ii, 192
DAVY, of Exeter
Atrophy of adrenals, ii, 738
DAVY and MACKERN
The blood in anæmia, ii, 750
DAX, of Sommières
Aphasia, i, 579
DAY, E. O.
Case of mollities ossium, ii, 862
DAY, of St Neots
Case of cerebral tumour, i, 612
DECHAMBRE
Senile atrophy of the lungs, ii, 110
DECROIX (Analogies entre la Choléra et la Peste Bovine, 1872)
Flesh of glandered animals, i, 376
DELAMARE
Gastric pains in locomotor ataxy, i, 535
DELLA CHIAJE
Osteo-arthritis from Pompeii, ii, 846
DEMARQUAY
Ulceration of trachea in glanders, ii, 63
DEMME, of Würzburg
Stenosis of trachea and bronchi, ii, 66
DESFOSSES (De la Hernie du Poumon, 1875)
Hernia of the lung, ii, 108
D'ESPINE
Hypertrophy of the brain, i, 647
DEVERGIE (Traité pratique des Maladies de la Peau, 1854)
Pityriasis pilaris, ii, 899
Pityriasis rubra, ii, 908
Trichoptylose, ii, 977
Ichthyosis, ii, 987
DICKINSON, W. H. (Diseases of the Kidney and Urinary Derangements, 1875, &c.)
Hydrocephalus, i, 649 *et seq.*
Vacuolation of the brain, i, 669
Histology of chorea, i, 748 *et seq.*
Tetanus, i, 762
Dysentery, ii, 398
Cirrhosis of the liver and alcohol, ii, 524
Lardaceous disease, ii, 639
Cirrhosis of kidney, ii, 646
Renal colic from aneurysm, ii, 669
Suppurative nephritis, ii, 684 *et seq.*
Diabetes, ii, 715 *et seq.*
The spleen in rickets, ii, 855
DICKINSON, of Workington
Trichiniasis in England, ii, 457
DIDAY (Tr. de la Syphilis des Nouveaux-nés, 1854, translation by New Syd. Soc.)
Congenital syphilis, i, 333
DIEBURG
Cheloid, ii, 1016
DITTRICH, of Erlangen (ü Lungenbrand im Folge von Bronchialerweiterung, 1850)
Tubercle i, 88
Laryngeal perichondritis, ii, 50 *et seq.*
Fœtid bronchitis, ii, 118 *et seq.*
DONKIN, A. S.
Skimmed milk in diabetes, ii, 729
DONKIN, H. B.
Paradoxical temperature, i, 41

Douglas, of Newbury
Rubeola, i, 224
Down, Langdon
Cretinism, i, 879
Croup, ii, 27
Dreschfeld, of Manchester
Cerebral gumma i, 607
— localisation, i, 625
Diabetic coma, ii, 719
Drewitt, F. G. D.
Cretinoid condition of adults, i, 882
Druitt, Robert
Paroxysmal hæmoglobinuria, ii, 587
Dubois (Traité des Accouchements, 1849)
Pemphigus neonatorum, i, 333
Du Bois Reymond
Migraine, i, 781 *et seq.*
Duchenne, of Boulogne (Traité de l'Électricité localisée, 1855–72; De la Paralysie musculaire pseudo-hypertrophique)
Peripheral paralysis, i, 392
Peripheral facial paralysis, i, 406
Acute atrophic paralysis, i, 497 *et seq.*
Chronic atrophic paralysis, i, 503
Progressive muscular atrophy, i, 507
Emporte-pièce, i, 508
Bulbar paralysis, i, 517 *et seq.*
Pseudo-hypertrophic paralysis, i, 525
Progressive muscular atrophy in children, i, 524
Locomotor ataxy, i, 530 *et seq.*
Paralysis agitans, i, 732
— of the diaphragm, ii, 70
Duckworth, Dyce (St Barth. Hosp. Rep.)
Cretinoid condition of adults, i, 881
Emphysema of lungs, ii, 112
Acute yellow atrophy of liver, ii, 534
Lardaceous liver, ii, 545
Charcot's joint disease, ii, 848
Acute pemphigus, ii, 919
Molluscum contagiosum, ii, 953
Detection of ringworm, ii, 963, *footnote*
Duffin
Hydatid of liver, ii, 556
Bullous erythema, ii, 933
Duffy, of Dublin
Iodic purpura, ii, 940
Dufour
Migraine, i, 781
Duncan, Matthews
Lupus of vulva, ii, 997
Duhring, of Philadelphia
Prickly heat, ii, 899
Lichen planus, ii, 902
Prurigo hiemalis, ii, 904
Neuroma, ii, 1014
Dukes, of Rugby
Rubeola, i, 224
Physiological albuminuria, ii, 599
Duménil
Peripheral neuritis, i, 430 *et seq.*
Dupuytren
Paralysis of hypoglossal nerve, i, 414
Durand-Fardel (Traité des Maladies des Vieillards, 1873)
Hemiplegia, i, 571
Apoplexy, i, 591
Duret
Anatomy of cerebral arteries, i, 558
Durham (Holmes' System; Guy's Reports)
Tetanus, i, 766
Tumours of the larynx, ii, 54 *et seq.*
Electrolysis for hydatid of liver, ii, 355
Malposition of kidney, ii, 705
Mollities ossium, ii, 861
von Dusch (transl. New Syd. Soc.)
Thrombosis of cerebral sinuses, i, 708

E.

Eales, of Birmingham
Albuminuric retinitis, ii, 601
Eastes, of Folkestone
Diphtheria, i, 264
Eberth (Bacterische Mykosen, 1872)
Diphtheria, i, 273
Rickets in utero, ii, 857
Ebstein (Ziemssen's Cyclopædia, &c.)
Typhus, i, 138
Renal calculus, peritonitis from, ii, 668
Tubercular disease of kidney, ii, 689
Renal cancer, ii, 694, 696
Moveable kidney, ii, 705 *et seq.*
Edis
Mucous cyst of larynx, ii, 54
Edmunds
Frequent pulse, i, 898
Edwards, Vertue
Non-contagiousness of phthisis, ii, 229
Eichhorst (Die progressive perniciöse Anämie, 1878; Handbuch d. speciellen Pathologie u. Therapie, 1887)
Peripheral neuritis i, 430
Myelitis, i, 451
Pseudo-hypertrophic paralysis, i, 528
The blood in anæmia, ii, 750
Parasitic nature of area, ii, 975
Eichstadt (1846)
Tinea vesicolor, ii, 972
Microsporon furfurans, ii, 711
Eimer (Die Blätternkrankheit, 1853)
Rose-rash in smallpox, i, 228
Eiselt (Prager Vierteljahrschr., 1861)
Melanotic sarcoma, i, 109
Eisenlohr
Plastic bronchitis, ii, 128
Eitner
Hæmoglobinuria, ii, 585
Ehrlich
Paroxysmal hæmoglobinuria, ii, 587
Elliotson (Med.-Chir. Trans., 1830)
Glanders, i, 376
Paralysis agitans, i, 734
Elsässer (Der weiche Hinterkopf, 1843)
Laryngismus stridulus, ii, 15
Craniotabes, ii, 856, 859
Elsberg, of New York
Syphilis of the larynx, ii, 45
Erb, of Heidelberg (Ziemssen's Cyclopædia)
Peripheral paralysis, i, 391 *et seq.*
Neuralgia, i, 418 *et seq.*
Sciatica, i, 425
Paraplegia, i, 437, 478

ERB (*continued*)—
Tendon-reflexes, i, 440
Myelitis, i, 447 *et seq.*
Neurasthenia spinalis, i, 452
Hyperæmia of spinal cord, i, 456
Case of concussion of cord, i, 460
Acute ascending paralysis, i, 461 *et seq.*
Hæmorrhage into spinal cord, i, 466
Hemiparaplegia, i, 472 *et seq.*
Chronic spinal meningitis, i, 495
Acute atrophic paralysis, i, 499 *et seq.*
Chronic atrophic paralysis, i, 503
Electric reactions in progressive muscular atrophy, i, 511
— in bulbar paralysis, i, 521
— in writers' cramp, i, 727
— in tetany, i, 737
Juvenile hereditary form of progressive muscular atrophy, i, 525
Pseudo-hypertrophic paralysis, i, 529
Locomotor ataxy, i, 533 *et seq.*
Disseminated sclerosis, i, 548 *et seq.*
Spasmodic torticollis, i, 716 *et seq.*
Function spasm, i, 722
Thomsen's disease, i, 735
Paralysis of the diaphragm, ii, 70
ERICHSEN (Concussion of the Spine, 1875)
Concussion of spinal cord, i, 460 *et seq.*
Rachialgia, i, 477
ESMARCH, of Kiel
Herpes from nervous lesion, ii, 1028
ESOFF
Ichthyosis, ii, 986
ESQUIROL (Des maladies mentales, 1838)
Epilepsy, i, 799, 808
ESTELLE
Characters of albumen in urine, ii, 595
EUGALENUS, SEVERINUS (De Scorbuto Morbo liber, 1588)
Scurvy, ii, 780
EULENBURG, of Greifswald (Ziemssen's Handbuch)
Paralysis agitans, i, 730 *et seq.*
Hysteria, i, 828
Angina pectoris, i, 911
Exophthalmic goitre, i, 1010
EWALD
Thickening of arteries in Bright's disease, ii, 613
EWART, COSSAR, of Edinburgh
Bacillus anthracis, i, 368
EWART, WILLIAM (Gulstonian Lect., 1882)
Anatomy of phthisis, ii, 207 *et seq.*

F.

FABER
Sea-voyage in phthisis, ii, 257
FALRET, JULES (Des Aliénés dangereux, 1869)
Paroxysmal insanity, i, 818 *et seq.*
FARR (Reports of Registrar-General)
Cholera, i, 298
Sex in relation to phthisis, ii, 255
General mortality in relation to that from influenza, ii, 276
FAUVEL (Recherches sur la Bronchite chez les Enfans, 1840)
Œdematous laryngitis, ii, 48
Gums in scurvy, ii, 781
FAYRER, Sir JOSEPH (Clin. and Path. Obs. in India, 1873)
Heat-stroke, i, 891
FEINBERG
Experimental reflex paraplegia, i, 458
FENWICK, BEDFORD
Tricuspid stenosis, i, 965
Atrophy of adrenals, ii, 738
FENWICK, SAMUEL (Morbid States of the Stomach and Duodenum, 1868, &c.)
Scarlatina, i, 212
Detection of pulmonary tissue in sputa of phthisis, ii, 214
Gastric tube casts, ii, 320
Chronic gastric catarrh, ii, 327
Pernicious anæmia, ii, 778
FERGUSON
Epidemic meningitis, i, 697
FERRIAR, of Manchester
Spasmodic croup, ii, 23
FERRIER (Functions of the Brain, 1876; Gulstonian Lectures, 1878)
Localisation of anosmia, i, 499
— of cerebral lesions, i, 572, 583, 620
Epilepsy, i, 801
"Snuff" for coryza, ii, 273
FIEDLER
Enteric fever, i, 201
FIELD
Spasmodic croup, ii, 22
FINLAYSON
Chorea, i, 742
Casts in jaundiced urine, ii, 605
FISCH
Albuminuria from abdominal pain and collapse, ii, 602
FLEISCHER
Theory of uræmia, ii, 621 *et seq.*
FLEMING, GEORGE
Anthrax, i, 375
FLINT, AUSTIN, of Philadelphia (Prin. and Pr. of Medicine, 1873)
Præsystolic murmur in aortic regurgitation, i, 967
Treatment of dysentery, ii, 401
Cholestearæmia, ii, 528
FLOURENS (Recherches sur la Système nerveux, 1842)
Cerebral localisation, i, 619
Functions on semicircular canals, i, 813
FOOTE, of Dublin
Chromidrosis, ii, 960
FORBES, Sir JOHN (Cases illustrating the use of the Stethoscope, 1824)
Percussion of thorax, ii, 76
FORDYCE, Sir WILLIAM (Fragm. Chir. et Med., 1784)
Hæmophilia, ii, 792
FORSTER, COOPER (Guy's Hosp. Reports)
Use of the term chancre, i, 312
Hydrophobia, i, 768
Trephining in epilepsy, i, 806
Introduction of gastrostomy, ii, 317

FORSTER, COOPER (*continued*)—
Obstruction of bowels for eighty-eight days, ii, 424
FOSTER, BALTHAZAR, of Birmingham
Diagnostic value of the course of the aortic murmur, i, 961
Lactic acid and rheumatism, ii, 817
FOSTER, MICHAEL (Text-book of Physiology, 1883)
Modes of dying, i, 9, 11
FOTHERGILL, JOHN (Account of the Sore-throat attended with Ulcers, 1748)
Diphtheria, i, 259
FOTHERGILL, SAMUEL (Account of the Tic douloureux, 1804)
Migraine, i, 788
FOULIS, DAVID, of Glasgow (Trans. Int. Med. Congress, 1881, vol. iii)
Extirpation of larynx, ii, 58
Cautery in granular pharyngitis, ii, 310
FOULIS, JAMES
Treatment of ringworm, ii, 967
FOURNIER (Leçons sur la Syphilis, 1873)
Syphilis, i, 308 *et seq.*
FOX, THOS. COLCOTT
Trichoclasia, ii, 977, footnote
Urticaria pigmentosa, ii, 936
Histology of lymphangioma, ii, 1017
FOX, CORNELIUS
Cough, ii, 72 *et seq.*
FOX, LONG
Chorea, i, 750 *et seq.*
Heat-stroke, i, 892 *et seq.*
FOX, TILBURY (Skin Diseases, 1873)
Lichen planus, ii, 902
Hydroa, ii, 921
"Xanthelasmoidea," ii, 936
Dysidrosis, ii, 961
Burmese ringworm, ii, 970
Tinea favosa, ii, 972
Fibroma fungoides, ii, 1017
FOX, WILSON (Artificial Production of Tubercle, 1868; Diagnosis and Treatment of Dyspepsia, 1867; Treatment of Hyperpyrexia, 1871, &c.)
Tubercle, i, 83 *et seq.*
Meningitis, i, 703
Pneumonia, ii, 137 *et seq*
Pyrexia in phthisis, ii, 212
Siderosis of lung, ii, 243
Acute gastric catarrh, ii, 319 *et seq.*
Atonic dyspepsia, ii, 327 *et seq.*
Gastric ulcer, ii, 348, 353
Cancer of stomach, ii, 361 *et seq.*
Treatment of hyperpyrexia, ii, 832
Blood-vessels in purpura, ii, 790
FRANCE
Diabetic cataract, ii, 717
FRAENKEL, of Breslau (Ziemssen's Hdbh.; Virchow's Archiv, &c.)
Inoculation of tubercle, i, 87
Syphilis of placenta, i, 331
The laryngoscope, ii, 3
Tubercular disease of larynx, ii, 40
Syphilitic disease of larynx, ii, 46
Tubercle of choroid, ii, 267
FRAENTZEL, of Berlin (Ziemssen's Hdbh.)
Pleurisy, ii, 168 *et seq.*
Pneumothorax, ii, 189 *et seq.*
Subcutaneous emphysema in phthisis, ii, 223
Coryza, ii, 272
Enlargement of heart, i, 928
FRANK, JOH. PETER (Interpretationes Clinicæ, 1812)
Bronchitis, ii, 94
FRANK, JOSEPH (Praxis Medicæ Universæ, 1811–43; Die Hautkrankheiten, 1843)
Prodroma of erysipelas, i, 252
Rachialgia, i, 474
Hydrocephalus, i, 650
Epilepsy, i, 795
FRERICHS, of Berlin (Die Bright'sche Nierenkrankheit, 1851; Diseases of the Liver, trans. New Syd. Soc., 1861, &c.)
Cholera, i, 285
Melanæmia in ague, i, 346
Cerebral hæmorrhage in Bright's disease, i, 564
Epidemic jaundice, ii, 497
Cirrhosis of liver, ii, 525 *et seq.*
Acholia from cirrhosis, ii, 528
Bright's disease, ii, 591
Theory of uræmia, ii, 620
FREUND
Thorax in emphysema, ii, 113
Cause of contracted thorax, ii, 234
FRIEDLÄNDER (ü locale Tuberculose, 1873)
Tubercle of the skin, i, 72
Arterial changes in Bright's diseaes, ii, 612
Giant-cells in lupus, ii, 996
FRIEDLEBEN (Die Physiologie der Thymusdrüse, 1858)
Laryngismus stridulus, ii, 14
Composition of bone in rickets, ii, 855
FRIEDREICH, of Heidelberg (Progressive Muskelatrophie, 1873; Virchow's Handbuch; Ziemssen's Cyclopædia)
Pseudo-hypertrophic paralysis, i, 527
Locomotor ataxy, i, 533 *et seq.*
Hereditary ataxic paraplegia, i, 545
Disseminated sclerosis, i, 549
Cerebral tumour, i, 613
Compression of left bronchus by dilated left auricle, ii, 63
Siderosis, ii, 243
FRISCH
Movement of bacillus anthracis, i, 368
Bacterium in rhinoscleroma, ii, 1008
FRITSCH and HITZIG
Cerebral localisation, i, 622
FÜRBRINGER
Salicin in retention of urine, i, 470
Physiological albuminuria, ii, 599
FULLER, HENRY WILLIAM
Plastic bronchitis, ii, 128
Alkalies in acute rheumatism, ii, 822
FULLER, THOMAS (Exanthematologia, 1730)
Varicella, i, 243

G.

GAIRDNER, of Glasgow (Path. Anat. of Bronchitis, 1850; Clinical Medicine, 1862)
Athetosis, i, 576
Chorea, i, 757
Collapse and emphysema, ii, 102
Rub, in emphysema, ii, 115, 176
Angina pectoris, i, 909 *et seq.*
Pityriasis rubra, ii, 908
Inoculation of leprosy, ii, 1011
GALABIN
Sphygmograph, i, 972
GALENUS, CLAUDIUS (ob. 200 A.D.)
Heat of fever, i, 37
The four signs of inflammation, i, 52
Thorax of phthisis, ii, 234
GALL (Craniologie, 1807)
Localisation of language, i, 580
GALTON, FR., and MAHOMED (Guy's Hosp. Rep. 1881)
The Physiognomy of Phthisis, ii, 233
GAMGEE, ARTHUR (Physiological Chemistry, 1880; Reynolds' System)
Glanders, i, 376 *et seq.*
Acetonæmia and Lipæmia in diabetes, ii, 719, *footnote*
GARCIA, MANUEL (Internat. Med. Cong., 1881, vol. iii, p. 197)
The laryngoscope, ii, 1
GARRETT, of Hastings
Throat cough, ii, 72
GARROD (Treatise on Gout and Rheumatic Gout, 1876; Reynolds' System)
Deposits of uric acid in cirrhotic kidneys, ii, 643
Theory of scurvy, ii, 785 *et seq.*
Gout, ii, 798 *et seq.*
Acute rheumatism, ii, 812
Arthritis deformans, ii, 841, *et seq.*
GAULE
Tubercle, i, 83
GAY
Intestinal obstruction from band, ii, 419
GAYTON
Vaccination, i, 240
GEBER
Rhinoscleroma, ii, 1008
GEDDINGS, of New York (k. k. Acad. d. Wissenschaften)
Histology of lupus erythema., ii, 1007
GEE (Reynolds' System; St Bart. Hosp. Reports; Auscultation and Percussion, 1870–77)
Scarlatina, i, 210 *et seq.*
Varicella, i, 244
Spastic paraplegia in children, i, 490
Tubercular meningitis, i, 676 *et seq.*
Laryngismus stridulus, ii, 16
Malformation of larynx, ii, 58
Physical examination of thorax, ii, 78
Atelectasis of lungs, ii, 103
Emphysema of lungs, ii, 113 *et seq.*
Physical signs of pneumonia, ii, 134
Thorax in pleurisy, ii, 168 *et seq.*
Signs of pneumothorax, ii, 196
GEE (*continued*)—
Bruit de pôt fêlé in effusion, ii, 222
Thorax in phthisis, ii, 233 *et seq.*
Large fusible calculus, ii, 661
Rickets, ii, 852 *et seq.*
GEISSLER
Herpes in pneumonia, ii, 138
GERHARD, of Philadelphia
Differentiation of typhus and enteric fevers, i, 164
GERHARDT (Lehrb. d. Kinderkrankh. 1861; 'Stimmbandlähmung,' Volkmann's Sammlung)
Thrombosis of cerebral sinuses, i, 708
Diseases of the larynx, ii, 5 *et seq.*
Stenosis of trachea, ii, 63 *et seq.*
Fœtid bronchitis, ii, 120
GERLACH, A. C. (Die Rinderpest, 1867)
Bovine tuberculosis, i, 90
Glanders, i, 379
GIBB (Diseases of the Throat, 1860)
Œdematous laryngitis, ii, 47
GIBBES, HENEAGE
Staining of tubercle bacillus, ii, 214
GIBERT
Pityriasis rosea, ii, 973
VON GIETL
Enteric fever, i, 190 *et seq.*
GILCHRIST (De Febre Anomala inter Dumfrisienses epidemica, 1774)
Nature of enteric fever, i, 165
GLASS, THOMAS
Large hydronephrosis, ii, 676
GLISSON, FRANCIS (De Rachitide, 1671)
Rickets, ii, 850
GÖLIS (Gehirnhöhlen-Wassersucht, 1815)
Hydrocephalus of childhood, i, 650
GOLTDAMMER
Hæmorrhage into spinal cord, i, 466
GOLTZ, of Strassburg
Reflex action of bladder, i, 441
Myelitis, i, 451
Cerebral localisation, i, 623
GOOCH, ROBERT
Spurious hydrocephalus, i, 687
"Child crowing," ii, 14
GOOD, MASON (System of Nosology, 1817)
Laryngismus stridulus, ii, 14
GOODALL
Hydrophobia, i, 768
GOODEVE (Reynolds' System)
Cholera, i, 279 *et seq.*
GOODHART (Guy's Hosp. Rep.; Diseases of Children, 1884, &c.)
Surgical scarlet fever, i, 30
Innocent tumours with cancer, i, 123
Infection of urine in paraplegia, i, 444
Syphilitic disease of arteries of spinal cord, i, 465
Rigidity from lesions of the brain, i, 590
Recovery from optic atrophy, i, 630
Statistics of chorea, i, 749
Rapid hypertrophy of heart, i, 922
Cervical ganglia in exophthalmic goitre, i, 1012
Syphilitic phthisis, ii, 239

GOODHART (*continued*)—
Intestinal casts, ii, 405
Connection of erysipelas with suppurative nephritis, ii, 684
Estimation of urea in suppurative nephritis, ii, 685
Moveable kidney, ii, 705
Treatment of leuchæmia, ii, 760
Ill-effects of salicylic acid, ii, 829
Cheloid in smallpox scars, ii, 1016
GOODSIR, of Edinburgh (Anatomical Memoirs, 1868)
Inflammation, i, 55
Sarcina ventriculi, ii, 363
GOOLDEN
Glycosuria from injury to head, ii, 726
GORDON
Epidemic meningitis, i, 694 *et seq.*
GORHAM
Inflation for invagination, ii, 413
GOWERS (Reynolds' System, &c.)
Peripheral facial paralysis, i, 402
Nature of "tendon-reflexes," i, 439
Pseudo-hypertrophic paralysis, i, 526
Locomotor ataxy, i, 537 *et seq.*
Hemiplegia, i, 573
Post-hemiplegic motor disorders, i, 576
Cerebral localisation, i, 625
Function-spasm, i, 721
Hydrophobia i, 773
Albuminuric retinitis, ii, 609 *et seq.*
Hæmocytometer, ii, 748
Hæmochromometer, ii, 749
Leucocythæmia, ii, 757 *et seq.*
VON GRÄFE
Oculo-motor paralysis, i, 411 *et seq.*
Vision in locomotor ataxy, i, 535
Changes in retina from cerebral tumour, i, 613 *et seq.*
Ocular affections in exophthamic goitre, i, 1011
Tubercle of choroid, ii, 267 *et seq.*
GRANCHER
Case of spread of tubercle, i, 93
GRAVES (Clinical Lectures, 1848)
Pupils in typhus, i, 135
Feeding in fever, i, 149
Hæmorrhage in enteric fever, i. 179
Scarlatina, i, 215
Ague, i, 342 *et seq.*
Peripheral neuritis, i, 430
Reflex paraplegia, i, 457
Chorea, i, 650
Exophthalmic goitre, i, 1009
Treatment of spurious croup, ii, 30
Opium in peritonitis, ii, 472
Grinding of teeth in lithæmia, ii, 490
Jaundice ii, 506
Rheumatism without arthritis, ii, 812
GRAY, of Oxford
Duration of chorea, i, 744
GREEN, HORACE, of New York
Granular pharyngitis, ii, 308 *et seq.*
GREEN, JONATHAN (Compendium of Diseases of the Skin, 1st ed., 1835)
Strophulus and lichen, ii, 900
Erythema nodosum, ii, 934
GREEN, T. H. (Introduction to Pathology)
Lymphoma, i, 114
Syphilitic phthisis, ii, 239
GREENFIELD, of Edinburgh (Reports to Privy Council; Atlas of Pathology, &c.)
Attenuated virus of anthrax, i, 30
Anatomy of yellow fever, i, 358
Anthrax of thoracic viscera, i, 373
Syphilitic phthisis, i,
Anatomy of Bright's disease, ii, 625
GREENHOW (Reports to Privy Council, 1859–60; Croonian Lectures on Addison's Disease, 1875; Chronic Bronchitis, 1869; Clin. Trans.)
Brassfounders' ague, i, 353
Diphtheria, i, 272
Production of phthisis by noxious trades, ii, 237
Diagnosis of chronic traumatic pneumonia from phthisis, ii, 240
Epidemic diarrhœa, ii, 379
Diabetes treated by skimmed milk, i, 729
Addison's disease, ii, 733 *et seq.*
Salicylic acid in rheumatism, ii, 830
GREGORY, GEORGE, of Edinburgh (Lectures on Eruptive Fevers, &c., 1843)
Varicella, i, 244
Tetanus, i, 761
Visual aura in epilepsy, i, 795
Duration of phthisis, ii, 226 *et seq.*
GREGORY, JAMES, of Edinburgh, 1797
Ague, i, 342
GRIESINGER ('Infectionskrankheiten,' Virchow's Handbuch, ii, 1857; Path. u. Ther. d. psychischen Krankheiten, 1861)
Relapsing fever, i, 155
"Bilious typhoid" fever, i, 158
Yellow fever, i, 359
Pseudo-hypertrophic paralysis, i, 526
Cerebral localisation, i, 625
General paralysis of the insane, i, 664
Hæmatoma of dura mater, i, 712
Hypochondriasis, i, 845
Cretinism, i, 877
Ætiology of pneumonia, ii, 141
Egyptian chlorosis, ii, 453
Endemic hæmaturia, ii, 702 *et seq.*
Anchylostomum duodenale, ii, 453
GRIFFIN, W. and DANIEL, of Limerick
Rachialgia, i, 473 *et seq.*
GRISOLLE (Traité de la Pneumonie, 1864)
Ætiology of pneumonia, ii, 141
GRUBY, DAVID (Gaz. Médicale, 1842)
Microsporon mentagraphytes, ii, 955
Parasitic nature of area, ii, 975
GUBLER, ADOLPHE
Locomotor ataxy, i, 538
Hemiplegia alterna, i, 571
Quinsy, ii, 307
GUÉRIN, JULES (Mém. sur les Difformités du Système Osseux)
Relation of craniotabes to rickets and congenital syphilis, ii, 859
Rachitis in utero, ii, 857

GUERSENT (Memoir on Diphtheria, 1835, transl. by Dr Semple for New Syd. Soc.)
Meningitis, i, 680
Croup and diphtheria, ii, 18
GUILLEMARD
Endemic hæmaturia, ii, 704
GUENEAU DE MUSSY
Delirium of typhus, i, 135
GUINIER (Le Gargarisme Laryngien, 1868)
Effectual method of gargling, ii, 309
GULL, Sir WILLIAM (Guy's Hosp. Rep.; Reynolds' System; Med.-Chir. Trans., &c.)
Enteric fever, i, 188
Oculo-motor paralysis, i, 413
Paraplegia, i, 435 *et seq.*
Myelitis, i, 449
Anæmia of spinal cord, i, 453
Concussion of spinal cord, i, 459
Paraplegia from spinal caries, i, 479
Chronic spinal meningitis, i, 494
Paralysis from lead, i, 506
Syringomyelus, i, 515
Retarded intelligence from cerebral tumour, i, 613
Cerebral localisation, i, 625
— abscess, i, 636, *et seq.*
Epilepsy, i, 799
Hysteria, i, 823 *et seq.*
Anorexia nervosa v. hysterica, i, 835
Cretinoid condition of adults ("myxœdema"), i, 881 *et seq.*
Gastric concretions, ii, 367
Treatment of tapeworm, ii, 445
Albuminuria of adolescents, ii, 599
Reaction of sweat in rheumatism, ii, 813
Treatment of acute rheumatism, ii, 823
GULL and SUTTON
Arterio-capillary fibrosis, i, 565, ii, 613
Duration of acute rheumatism, ii, 823
GUNTZ (Das Syphilitische fieber, 1873)
Syphilis, i, 315
GUTTMANN (Lehrb. d. klin. Untersuchungsmethoden f. die Brust u. Unterleibsorganen, 1872)
Auscultation, ii, 83 *et seq.*
Fœtid bronchitis, ii, 118
"Rub" from pleural tubercles, ii, 176
GUY (Public Health, 1870)
Impure air a cause of phthisis, ii, 236
"The sweating sickness," ii, 813, *note*

H.

HABERSHON (Lumleian Lectures, 1876; Dis. of Abdomen, 1878; Guy's Hosp. Rep.)
Case of adult hydrocephalus, i, 661
Case of spasmodic torticollis, i, 718
Treatment of paralysis agitans, i, 734
Glossitis in chorea, i, 745
Stomach-cough, ii, 73
Addison's disease, ii, 740
Arsenic in purpura, ii, 791
DE HAEN, of Vienna (Opuscula medico-physica, 1777)
Use of clinical thermometer, i, 37
Smallpox, i, 230
Plastic bronchitis, ii, 125
HAIGHT, of New York
Histology of erysipelas, i, 254
— of zona, ii, 1025
HAHN
Moveable kidney, ii, 707
HALDANE, of Edinburgh
Renal calculi of calcic carbonate, ii, 660
HALL, MARSHALL (Lect. on Nervous System and its Diseases, 1837–41; Prone and Postural Respiration in Drowning, 1857)
Artificial respiration, i, 13
Spurious hydrocephalus, i, 687 *et seq.*
Chorea, i, 741.
Laryngismus stridulus, ii, 16
HALLIBURTON
Analysis of Tissues in Myxœdema, i, 882
HAMILTON, of Aberdeen (Practitioner, 1879–80; Pathology of Bronchitis, &c., 1883)
Tubercle, i, 81 *et seq.*
Bronchitis, ii, 97 *et seq.*
Definition of phthisis, ii, 202
Localisation of phthisis, ii, 202
Lipæmia and fat-embolism in diabetes, ii, 721
HAMMOND, of New York (Diseases of the Nervous System, 1871)
Anæmia of spinal cord, i, 455
Reflex paraplegia, i, 456
Rachialgia, i, 474
Progressive muscular atrophy, i, 507
Athetosis, i, 576
HAMBURGER
Diagnosis of œsophageal stricture by auscultation, ii, 315
HANCOCK
Spasmodic torticollis, i, 717
HÄNISCH (Ziemssen's Cyclopædia)
Yellow fever, i, 357
Nasal polypi a cause of asthma, ii, 296
HANNOVER, of Copenhagen (Das Epithelioma, 1852)
Epithelioma, i, 118
HANSEN, of Bergen
Bacillus lepræ, ii, 1010
HARDY (Leçons sur les Maladies de la Peau, 1858–64; Maladies de la Peau, 1886)
Syphilodermia, i, 318
Alkaline lotions in eczema, ii, 886
Strophulus volaticus, ii, 900
Treatment of scabies at St Louis, ii, 926
Parasitic sycosis, ii, 955
— nature of area, ii, 975, 976
Scrofulides, ii, 999
Treatment of lupus, ii, 1004
HARE (quoted by Cheyne)
Morbus strangulatorius, ii, 19
HARE, C. J.
Treatment of hysterical fit, i, 841
Enormous cystic kidney, ii, 651

HARE, C. J. (*continued*)—
Hydronephrosis from tortuous ureter, ii, 677
HARLEY, GEORGE (The Urine and its Derangements, 1872)
Paroxysmal hæmoglobinuria, ii, 586
HARLEY, JOHN (Reynolds' System; St. Thomas's Hosp. Rep., 1876, &c.)
Rose rash in typhus, i, 133
Typhoid stools, i, 174
Actinomycosis, i, 383
Spasmodic torticollis, i, 716 *et seq.*
Hysterical cough, ii, 74
Endemic hæmaturia, ii, 703 *et seq.*
VAN HARLINGEN, of Philadelphia
Medicinal rashes, ii, 941
Sclerodermia, ii, 990, *footnote*
HASLAM (Observations on Insanity, 1798)
General paralysis, i, 663
HASLUND, of Copenhagen
Iodide of potassium in psoriasis, ii, 916
HASSE (Kr. des Nervenapparat., 1855)
Disseminated sclerosis, i, 548
The pigment in miners' phthisis, ii, 241
HAUGHTON, of Dublin
Hypertrophy of bladder in diabetes, ii, 720
HAWKINS, CÆSAR
Compression-paraplegia, i, 484
HAYEM
Hæmorrhage into spinal cord, i, 465
The blood in anæmia, ii, 748
HAYGARTH (Clinical History of Diseases, vol. i, 1805)
Nodosity of the joints, ii, 840
HAYWARD
Nephrolithotomy, ii, 688
HEARNE, of Southampton
Treatment of torticollis, i, 719
HEBERDEN, WILLIAM, the elder (Commentarii de morborum historia, 1802)
Varicella, i, 243 *et seq.*
Local treatment of diphtheria, i, 277
Angina pectoris, i, 908
Whooping-cough in old age, ii, 289
Hæmoptatic drugs, ii, 666, *footnote*
Digitorum nodi, ii, 842
HEBERDEN, WILLIAM, the younger (Observations on the Plague, &c., 1801)
Plague, i, 303
HEBRA, FERDINAND (Acute Exantheme und Hautkrankheiten, 1860–74)
Roseola variolosa, i, 228, 229
Eczema a dermatitis, ii, 872
The continuous bath, ii, 883
Lichen ruber, ii, 901
Prurigo gravis, ii, 905 *et seq.*
Frequency of pemphigus, ii, 921
Parasitic sycosis, ii, 955
Non-parasitic nature of area, ii, 975
Lupus, ii, 997 *et seq.*
VON HEBRA, HANS (Die kr. Veränderungen d. Haut, 1884)
Scrofuloderma papulosum, ii, 901
Lichen planus, ii, 902
Pityriasis rubra, ii, 907
VON HEBRA, HANS (*continued*)—
Histology of pityriasis rubra, ii, 909
Erythema and pemphigus, ii, 927
Sycosis frambœsiformis, ii, 950
Ichthyosis congenita, ii, 988
VON HEINE, JACOB (1840)
Acute atrophic paralysis, i, 497
Angina pectoris, i, 911
HEINZE (Monograph, Leipzig, 1879)
Tubercle of the larynx, ii, 38 *et seq.*
HEITLER
Statistics of cured phthisis, ii, 208
HELLER (Ziemssen's Cyclopædia)
Epidemic meningitis, i, 693
HELMHOLTZ
Vowel sounds, ii, 91
Treatment of hay-fever, ii, 285
HEMPTENBACHER
Pseudo-hypertrophic paralysis, i, 528
HENDERSON, of Edinburgh, 1842
Relapsing fever, i, 153 *et seq.*
HENLE, of Göttingen
Anatomy of laryngeal adductors, ii, 10
HENOCH (Beiträge z. Kinderheilkunde, 1861)
Laryngismus stridulus, ii, 16
HERING, THEODOR (Histol. u. experim. Studien ü. die Tuberculosis, 1873)
Tubercle and inflammation, i, 83
HERMANN (Physiologie, 1872)
Ligature of renal arteries, i, 283
Amount of pressure necessary to prevent secretion of urine, ii, 671
HERSCHELL, Sir JOHN
Migraine, i, 781
HERTWIG
Foot-and-mouth disease in man, i, 381
Inoculation of influenza, ii, 279
HERTZ, of Amsterdam (Ziemssen's Hdbh.)
Ague, i, 339 *et seq.*
Melanæmia, i, 347
Emphysema, ii, 106
Gangrene of lungs, ii, 161
Mediastinal tumours, i, 1003
HERTZBERG
Locomotor ataxy, i, 532
HESSE
Mediastinal tumours, i, 1004
HEUBNER, of Leipzig (Ziemssen's Hdbh.)
Syphilis of spinal cord, i, 464
Gumma of spinal meninges, i, 482
Syphilitic disease of cerebral arteries, i, 556 *et seq.*
Anatomy of cerebral arteries, i, 607
Apoplexy, i, 600 *et seq.*
Gumma of brain, i, 607 *et seq.*
Anatomy of dysentery, ii, 388
VON HEUSINGER
Erythema from quinine, ii, 940
HEWETT, PRESCOTT
Hæmatoma of dura mater, i, 710
HEWITT, GRAILY
Uterine flexion and hysteria, i, 836
HEYDENREICH (Klin. u. micr. Unters. ü. d. Parasiten des Rückfallstyphus, 1877)
Spirochæta Obermeieri, i, 159

HICKS, BRAXTON (Obstetrical Trans.; Guy's Hosp. Reports)
Alteration of type in disease, i, 21
Puerperal scarlatina, i, 210
Epidemic of diphtheria in obstetric ward of Guy's Hospital, i, 266
HIGGINBOTTOM, of Nottingham (Essay on the Use of Nitrate of Silver, 1826–65)
Lunar caustic in erysipelas, i, 257
HIGGINS, CHARLES (Guy's Hosp. Reports)
Optic neuritis from cerebral tumour, i, 630
HILLIER (Diseases of Children, 1868; Holmes's System of Surgery)
Chorea, i, 744 *et seq.*
Croup and diphtheria, ii, 18
Histology of lichen planus, ii, 902
HILTON ('Rest and Pain;' Guy's Hosp. Rep.)
Relation between cutaneous and deep nerve supply, i, 419
Rachialgia, i, 474
Arrest of growth after disease of a joint, i, 499
Congenital malformation of hip, i, 528
Hydrocephalus of childhood, i, 651
Cause of vomiting in phthisis, ii, 213
Epigastric pain due to spinal disease, ii, 334
Post-mortem blisters, ii, 920
HINTON (Guy's Hosp. Reports)
Examination of internal ear in paroxysmal vertigo, i, 815
HIPPOCRATES
Effect of fever on chorea, i, 746
Tetanus, i, 757
Hypochondriasis, i, 843
Auscultation, ii, 82
Peripneumonia, ii, 129
Pleuritic creaking, ii, 163
Succussion splash, ii, 195, footnote
Phthisis ab hæmoptoë, ii, 215
Dysentery, ii, 388
Chlorosis, ii, 769
HIRSCH, AUGUST (Handb. d. Historisch-geographischen Pathologie, 1859–64; Meningitis cerebrospinalis epidemica, 1866)
Erysipelas, i, 250
Plague, i, 305
Ague, i, 347 *et seq.*
Dengue, i, 364 *et seq.*
Epidemic meningitis, i, 694 *et seq.*
Pertussis, ii, 289
Distribution of dysentery, ii, 393
Distribution of rheumatism, ii, 816
HIRSCHBERG (Klin. Beobachtungen aus der Augenheilanstalt, 1874)
Conjunctival diphtheria, i, 267
HIRT (Ziemssen's Handbuch)
Bronchitis from irritant gases, ii, 122
HISLOP
Foot-and-mouth disease in man, i, 382
HITZIG ('Localisation im Grosshirn,' Volkmann's Sammlung, 1877; Ziemssen's Handbuch.)
Chronic spinal meningitis, i, 495
Peripheral facial paralysis, i, 406
HITZIG (*continued*)—
Locality of lesion in hemiplegia, i, 572
Late rigidity of hemiplegia, i, 575
Cerebral localisation, i, 620
Cerebral abscess, i, 642
General paralysis of the insane, i, 664
HODGKIN (Guy's Hosp. Reports)
Anæmia lymphatica, ii, 761
HOFFMANN, C. E. E. (Path. anat. Veränderungen beim Abdominaltyphus, 1869)
Enteric fever, i, 168 *et seq.*
HOFMEIER
Hæmoglobinuria from potassic chlorate, ii, 585
HOLMES
Pulsation of renal carcinoma, ii, 696
HOMAN and HERTWIG
Incubation of dysentery, ii, 396
HOME, of Edinburgh (Inquiry into the Nature, Causes, and Cure of Croup, 1765)
Croup, ii, 18 *et seq.*
HOPE
Mitral contraction, i, 963
HOPPE-SEYLER
Removal of cerebral tumour, i, 633
Analysis of synovia in arthritis deformans, ii, 844
HORSLEY, VICTOR
Experimental myxœdema, i, 882
HOUSSARD
Hæmatoma of dura mater, i, 710
HOWARD, of New York
Artificial respiration, i, 13
HOWSE (Guy's Hosp. Reports)
Scarlatina and septicæmia, i, 21
Excision of rib for empyema, ii, 188
Gastrostomy, ii, 317
Nephrolithotomy, ii, 688
HUDSON, of Dublin (Lectures on Fever, 1868)
Typhus, i, 147
Friction-sound in emphysema, ii, 115
HUGHES (Guy's Hosp. Reports)
Chorea, i, 749 *et seq.*
Hydrophobia, i, 767
Pneumothorax in phthisis, ii, 198
Treatment of peritonitis, ii, 472
HUGUENIN (Ziemssen's Handbuch)
Enteric fever conveyed by meat, i, 198
Origin of compound granule-masses in cerebral softening, i, 559
Red softening of brain, i, 635
Cerebral abscess, i, 636 *et seq.*
Hydrocephalus of childhood, i, 649
— of adults, i, 662 *et seq.*
Meningitis, i, 676 *et seq.*
Hæmatoma of dura mater, i, 709
HULKE (Treatise on the use of the Ophthalmoscope, 1861; Ophthal. Hosp. Rep., &c.)
Neuro-retinitis from cerebral tumour, i, 616
Rodent ulcer, ii, 1019
HUME, of Newcastle
Rodent ulcer, ii, 1020

HUNTER, DOUGLAS
Anatomy of sunstroke, i, 893
HUNTER, JOHN
Specimen of bowel affected with typhoid ulceration, i, 165
Paralysis from plumbism, i, 506
"Bronchial polypi," ii, 125
Relation of pressure to hypertrophy and to atrophy, ii, 981
HUNTER, WILLIAM
Case of irritable stomach, ii, 338
Case of congenital cardiac disease, i, 980, 981, *footnote*
HUSS, of Stockholm
Alcoholic phthisis, ii, 238
HUTCHINSON, JOHN (Todd's Cyclo., 1852)
Spirometer, ii, 93, 234
HUTCHINSON, JONATHAN (Inherited Syphilis, 1863; Reynolds' System; Clinical Lectures; London Hosp. Reports)
Contagious inflammation, i, 26
Tumours, i, 109
Varicella, i, 246
Varicella-prurigo, i, 246; ii, 904
Erysipelas, i, 251
Pupils in collapse, i, 282
Syphilis, i, 309 *et seq.*
Local temperature after lesion of nerve-trunks, i, 398
Reflex action of bladder after cured spina bifida, i, 442
Hemiplegia, i, 572
Optic neuritis after meningitis, i, 704
Ear affection in meningitis, i, 704
Migraine, i, 784
Intestinal casts, ii, 405
Invagination of intestine, ii, 412
Family distribution of gout, ii, 803
Arthritic diathesis, ii, 816
Arthritis deformans, ii, 841
Lichen planus and leucoplacia, ii, 903
Prurigo, ii, 904
Pityriasis rubra, ii, 908
Bullous dermatitis with sore fingers and mouth, ii, 921
Chiro-pompholyx, ii, 921
Arsenic in pemphigus, ii, 922
Eruptions allied to chilblains, ii, 934
Cases of favus, ii, 972
Lupus erythematosus, ii, 1007
Rhinoscleroma, ii, 1008
Ætiology of leprosy, ii, 1012
Fibroma molluscum, ii, 1014
Cheloid, ii, 1016
Lupus lymphaticus, ii, 1016
Zona, ii, 1026
HÜTER, of Greifswald (Pitha's Handbuch)
Erysipelas, i, 251
Diphtheria, i, 262
Deformity from paralysis, i, 499
Cardiac infarctus, i, 936
HUXHAM (Essay on Fevers, 1764)
Enteric fever, i, 165
HYDE, J. N., of New York
Rash from cannabis indica, ii, 939

I.

IMMERMANN (Ziemssen's Handbuch)
Anæmia, ii, 778
Scurvy, ii, 783
Purpura, ii, 788 *et seq.*
Hæmophilia, ii, 792 *et seq.*
VON INS (Arch. f. Exp. Path., 1876)
Experiments on causation of pneumonokoniosis, ii, 292
IRVINE
Enteric fever, i, 170 *et seq.*
ISRAEL, JAMES, of Berlin
Actinomycosis, i, 382, 383
ITARD (a pupil of Bayle, 1803)
Pneumothorax, ii, 189

J.

JACCOUD (Les Paraplegies; Traité de Pathologie interne, 1875)
Myelitis, i, 445
Reflex paraplegia, i, 457
Hæmorrhage into spinal cord, i, 465
Progressive muscular atrophy, i, 509
Locomotor ataxy, i, 533 *et seq.*
Atrophy of optic disc, i, 617
Chorea, i, 741 *et seq.*
Addison's disease, ii, 740
JACKSON, HUGHLINGS (Reynolds' System; London Hosp. Rep., &c.)
Hemiplegia, i, 569 *et seq.*
Hemiopia, i, 573
Aphasia, i, 576 *et seq.*
Apoplexy, i, 585 *et seq.*
Cerebral tubercle, i, 605
Cerebral localisation, i, 617 *et seq.*
— tumour, i, 628 *et seq.*
Tetany, i, 737
Embolic theory of chorea, i, 754
Epilepsy, i, 797 *et seq.*
Paroxysmal insanity, i, 818
JACKSON, SCORESBY
Aphasia, i, 578
JACKSON, of Boston
Heredity of emphysema, ii, 110
JACOB, of Dublin
Rodent ulcer, ii, 1019
JACOBSON, of Königsberg (Virchow's Archiv, 1870)
Local temperature in inflammation, i, 59
Conjunctival diphtheria, i, 267
JAFFÉ
Fœtid bronchitis, ii, 118
JEBB (Select Cases of Paralysis, 1783)
Hysterical catalepsy, i, 830
JEFFRIES
Tic douloureux, i, 421
JENNER, EDWARD
Vaccination, i, 240
JENNER, Sir WILLIAM (Typhoid and Typhus Fevers, 1850; Lectures on Rickets in Medical Times, 1850; Diphtheria, 1861; Emphysema in Reynolds' System, &c.)
Heredity of tumours, i, 123
Typhus rash, i, 133 *et seq.*

JENNER (*continued*)—
Relapsing fever, i, 153
Enteric fever, i, 165 *et seq.*
Febricula, i, 207
Diphtheria, i, 264 *et seq.*
Hypertrophy of the brain, i, 647
Croup and diphtheria, ii, 18 *et seq.*
Compression of trachea by abscess, ii, 30
Emphysema, ii, 108 *et seq.*
Tubercular and scrofulous diatheses, ii, 232
Cancerous peritonitis, ii, 479
Addison's disease, ii, 740
Hæmophilia, ii, 793, 795
Rickets, ii, 852 *et seq.*

JESSOP, of Leeds
Nephrectomy for renal cancer, ii, 698

JOFFROY
Bulbar paralysis, i, 521
Locomotor ataxy, i, 541

JOHNSON, GEORGE (Epidemic Diarrhœa and Cholera, 1870; Lumleian Lectures, 1877; Medical Lectures and Essays, 1887)
Causation of diphtheria, i, 265
Coincidence of diphtheria and scarlet fever, i, 265
Pathology of cholera, i, 288
Paralysis of larynx from pressure on the vagus nerve, ii, 6
Croup and diphtheria, ii, 18
Tests for albuminuria, ii, 596
Physiological albuminuria, ii, 599

JOHNSON, JAMES (Influence of Tropical Climates, 1813)
Prickly heat, ii, 899

JOHNSON, of Dublin
Laryngismus stridulus, ii, 17

JOLLY, of Strassburg (Ziemssen's Hdbh.)
Hysteria, i, 822 *et seq.*

JONES, BENCE (Contributions to the Chemistry of the Urine, 1845)
Excretion of urea in chorea, i, 742
Phosphates in delirium tremens, i, 886
Treatment of chyluria, ii, 702
Glycosuria in apparent health, ii, 716
Ætiology of diabetes, ii, 723

JONES, HANDFIELD (Clin. Obs. on Functional Nervous Disorders, 1864)
Paralysis agitans, i, 733
Chorea, i, 742
Pyrosis, ii, 336

JONES, of Jersey
Digitalis in delirium tremens, i, 890

JÖRG, E. (Die Fötuslunge u. s. w., 1835)
Atelectasis of lungs, ii, 101

JÜRGENSEN, of Tübingen ('Kl. Studien ü. d. Behandlung des Abdominaltyphus,' 1866; Ziemssen's Handbuch)
Enteric fever, i, 177 *et seq.*
— and febricula, i, 208
Pneumonia, ii, 131 *et seq.*
Broncho-pneumonia, ii, 148 *et seq.*
Acute miliary tuberculosis, ii, 263
Test for lardacein, ii, 630

K.

KALTENBACH
Pyelitis after parturition, ii, 682

KAPOSI (Hebra's Hautkrankheiten, 1870; Syphilis der Haut, 1874)
Syphilis, i, 315 *et seq.*
Histology of lichen scrofulosus, ii, 901
Dermatitis papillomatosa capillitii, ii, 956
Non-parasitic nature of area, ii, 975
Area a tropho-neurosis, ii, 976
Trichorrhexis nodosa, ii, 977
Histology of linear atrophy, ii, 991
Lupus erythematosus disseminatus, ii 1007
Rhinoscleroma, ii, 1008
Leprosy, ii, 1009
Elephantiasis teleangiectodes, ii, 1017
Xerodermia maligna, ii, 1018

KEKULÉ
Composition of lardacein, ii, 630

KELLY
Soil in relation to phthisis, ii, 252

KEY, ASTON (Guy's Hosp. Rep.)
Compression-paraplegia, i, 478
Tetanus, i, 760
Treatment of bunion, ii, 983

KIDD, P.
Diphtheritic paralysis, i, 274

KING, WILKINSON (Guy's Hosp. Rep.)
Compression-paraplegia, i, 478
Sphygmograph, i, 970
Compression of left bronchus by dilated left auricle, ii, 63
Position of diverticulum ilei, ii, 420

KIRKES
Embolism, i, 71
Chorea, i, 742

KLEBS (Handbuch der pathol. Anat., 1870)
Tubercle, i, 84 *et seq.*
Bacillus of ague, i, 351
Cerebral tumour, i, 609
Scarlatinal nephritis, ii, 625
Myoma of skin, ii, 1014

KLEIN (Anatomy of Lymphatic System; Reports to Privy Council)
Fission of leucocytes i, 61
Tubercle, i, 82
Cholera-bacillus, i, 292
Scarlatinal nephritis, ii, 625

KLEMM
Experimental reflex paralysis, i, 458

KNAPP, of New York
Cerebral localisation, i, 618
Paroxysmal vertigo, i, 813 *et seq.*

KÖBNER
Osteitis in congenital syphilis, i, 332
Fungus of eczema marginatum, ii, 970
Bacillus lepræ, ii, 1010

KOCH, of Berlin
Infective micrococci, i, 24
Bacillus tuberculosis, i, 83 *et seq.*
"Comma-bacillus" of cholera, i, 291
Bacillus anthracis, i, 368
Bacilli in phthisical sputa, ii, 201

KOCHER, of Bern
Cretinism after excision of thyroid, i, 882

KOHN, MORITZ (*vide* KAPOSI)

KOHTS, of Strassburg (Virchow's Archiv, 1874)
Cough, ii, 72 *et seq.*
— from irritation of pleura, ii, 172
VAN DER KOLK, SCHROEDER (Obs. Anat. et Path., 1826)
Lymphatics in false membranes, i, 67
— in new growths, i, 100
Relation between cutaneous and deep nerve supply, i, 419
Epilepsy, i, 800 *et seq.*
KÖNIG, of Rostock (Pitha's Handbuch)
Erysipelas, i, 250
KOPP
Thymic asthma, ii, 14
KÖSTER
Tubercle, i, 82
Tumours, i, 100 *et seq.*
Calcareous infiltration of heart, i, 941
KRAUS (Das perforirende Geschwür im Duodenum, 1865)
Statistics of duodenal ulcer, ii, 351
KRETSCHY
Plastic bronchitis, ii, 126
KREUSER (Die Compl. d. acuten Rheumatismus, 1866)
Hyperpyrexia in rheumatism, ii, 819
KRISHABER (Internat. Med. Congr., 1881)
Bulbar paralysis, i, 518
Tubercle of larynx, ii, 42
Tumours of larynx, ii, 56
Sonde œsophagienne à demeure, ii, 316
KRYLOW
Fatty degeneration of heart, i, 940
KÜCHENMEISTER (Manualon Parasites, 1857)
Intestinal entozoa, ii, 438 *et seq.*
KÜHNE, of Heidelberg
Cholera, i, 287
Composition of lardacein, ii, 630
KÜLZ (Beiträge zur Path. u. Ther. des Diabetes, 1874)
Lactic acid and rheumatism, ii, 818
KUSSMAUL, of Strassburg (Clin. Lectures in Volkmann's Sammlung)
Bulbar paralysis, i, 517 *et seq.*
Paracentesis thoracis, ii, 183
Chalicosis, ii, 243
Washing out dilated stomach, ii, 366
Diabetic coma, ii, 719 *et seq.*
KUSSMAUL and TENNER (On the Nature and Origin of Epileptic Convulsions, transl. Syd. Soc.)
Epilepsy, i, 802
KÜTTNER
Relapsing fever, i, 158
KYBER, of Dorpat (Unters. ü. d. Amyloide Degeneration, 1871)
Lardaceous disease, ii, 631

L.

LAACHE, of Christiania (Die Anämie, 1883)
Pernicious anæmia, ii, 776
LAENNEC (Traité de l'Auscultation médiate, 1819, translated by Forbes, 1821)
Tubercle, i, 79 *et seq.*
LAENNEC (*continued*)—
Hypertrophy of the brain, i, 647
Frémissement in mitral stenosis, i, 958
Tubercle of larynx, ii, 37
Discovery of auscultation, ii, 81 *et seq.*
Catarrhe sec, ii, 105
Emphysema of lungs, ii, 106 *et seq.*
Pneumonic crepitation, ii, 134
Gangrene of lungs, ii, 159
Pleurisy, ii, 164 *et seq.*
Pneumothorax, ii, 189 *et seq.*
Phthisis, ii, 200, *et seq.*
Cirrhosis of liver, ii, 523
LAMB, W. H. (Guy's Hosp. Reports)
Croup and albuminuria, ii, 26
LANCEREAUX (Traité de la Syphilis, 1866)
Syphilis, i, 307
Angina pectoris, i, 911
LANCISI (Opera, 1718)
Ague, i, 339
LANDAU
Floating kidney, ii, 706
LANDENBERGER
Scarlatina, i, 212
LANDRY (Traité des Paralysies, 1859)
Acute ascending paralysis, i, 461 *et seq.*
LANE, W. A.
Laryngoscopic view of trachea, ii, 65
LANGHANS
Tubercle, i, 81 *et seq.*
Primary carcinoma of trachea, ii, 63
LANKESTER, E. RAY
Pleomorphism, i, 25, *footnote*
LARREY, D. J.
Tetanus, i, 760 *et seq.*
LASÈGUE
Hysteria, i, 824
Palatal ecchymosis in scurvy, ii, 781
Histology of scurvy, ii, 785
LASSAR
Inflammation, i, 58 *et seq.*
LATHAM, PETER MERE (Clinical Lectures on Diseases of the Heart, 1845; Collected Works, New Syd. Soc., 1876)
Frequent pulse, i, 898
Angina pectoris, i, 908
Rhonchus, ii, 87
Dry bronchitis, ii, 99
LATHAM, P. W., of Cambridge (Nervous or Sick Headache, 1873)
Migraine, i, 783 *et seq.*
LAVERAN
Tuberculosis of joints, ii, 266
LAWSON, HENRY
Sciatica, i, 423 *et seq.*
Choked optic disc, i, 615
LAWSON, ROBERT
Yellow fever, i, 360
LAYCOCK, of Edinburgh
Rheumatic diathesis, ii, 816
Rapid canities, ii, 1022
LEBERT, of Breslau (Traité des Mal. Scrofuleuses et Tuberculeuses, 1849; Traité d'Anatomie Pathologique, 1857–60; Rückfalls- und Fleck-typhus, 1870, &c.)
The "tubercle corpuscle," i, 81
Tumours, i, 97 *et seq.*

LEBERT (*continued*)—
Typhus, i, 132 *et seq.*
Relapsing fever, i, 156 *et seq.*
Cholera, i, 281 *et seq.*
Cerebral abscess, i, 610
— tumour, i, 612
Plastic bronchitis, ii, 127 *et seq.*
Essential anæmia, ii, 772
Acute rheumatism, ii, 812, 823
Arthritis deformans, ii, 846
Keratosis intra-uterina, ii, 988
Cheloid, ii, 1015
LEE, HENRY (Holmes's System of Surgery)
Syphilis, i, 310
LEES, D. B.
Malformation of larynx, ii, 58
LEES and BARLOW
Rickets, craniotabes, and congenital syphilis, ii, 859
LEFFERTS, of New York
Fibroma of larynx, ii, 54
LEGG, WICKHAM (Hæmophilia, 1872; St Barth. Hosp. Reports, &c.)
Artificial pyrexia, i, 50
Myocarditis, i, 937
Cardiac aneurysm, i, 938
Sarcoma of stomach, ii, 356
Effects of ligature of bile-duct, ii, 505
Atrophy of adrenals, ii, 738
Hæmophilia, ii, 792 *et seq.*
Purpura rheumatica, ii, 1025
LEGGATT
Yellow fever, i, 357
LEGROS
Artificial production of chorea, i, 747
LELOIR
Neurotic affections of the skin, ii, 1029
LÉPINE
Chronic diffused atrophic paralysis, i, 504
LESSER
Symmetrical leucodermia, ii, 1022, *note*
LETZERICH
Fungus in pertussis, ii, 290
LEUBE, of Jena
Anthrax, i, 370, 373
Disseminated sclerosis, i, 549
Physiological albuminuria, ii, 599
Amount of albumen in pyuria, ii, 681
LEUCKART (Die menschlichen Parasiten, 1863
Intestinal entozoa, ii, 436 *et seq.*
LEWIN
Chronic catarrhal laryngitis, ii, 35
LEWIS, T. R., of Calcutta (Hæmatozoon, 1872)
Spirochæta Obermeieri, i, 160
Filaria sanguinis hominis, ii, 459
Comma-bacillus, i, 292
LEWIS, BEVAN
The nerves in general paralysis, i, 669
LEWISSON
Reflex paraplegia, i, 457
LEYDEN, of Berlin (Klinik d. Rückenmarkskrankheiten, 1874; Volkmann's Sammlung)
Pyrexia, i, 47 *et seq.*
LEYDEN (*continued*)—
Paraplegia, i, 439 *et seq.*
Myelitis, i, 445 *et seq.*
Anæmia of spinal cord, i, 454 *et seq.*
Concussion of spinal cord, i, 460
Tubercle of spinal cord, i, 466
Compression paraplegia, i, 478 *et seq.*
Progressive muscular atrophy, i, 509
Bulbar paralysis, i, 518
Pseudo-hypertrophic paralysis, i, 527
Locomotor ataxy, i, 539
Meningitis, i, 697 *et seq.*
Laryngismus stridulus, i, 787
Phthisis i, 956 *et seq.*
Reflex cough, ii, 73
Fœtid bronchitis, ii, 118
Asthma, ii, 297
LICHTHEIM (Volkmann's Sammlung)
Experiments on plugging of the bronchi, ii, 67
Atelectasis of lungs, ii, 102 *et seq.*
Experiments on the elasticity of the lung, ii, 102, 166
Cause of renal dropsy, ii, 607
LIEBERMEISTER, of Tübingen (Handbh. d. Path. u. Ther. d. Fiebers, 1875, and in Ziemssen's Handbuch)
Specific diseases, i, 20 *et seq.*
Pyrexia, i, 43 *et seq.*
Enteric fever, i, 168 *et seq.*
Yellow fever, i, 358
Plague i, 303 *et seq.*
Dysentery, ii, 396
Warm baths in acute nephritis ii, 654
LIEUTAUD (Précis de la Médecine pratique, 1776)
Fibroma of larynx, ii, 54
LIND, JAMES (Treatise on the Scurvy, 1753)
Scurvy, ii, 780
LIOUVILLE (La Généralisation des Anevrismes miliaires, 1871)
Miliary aneurysms, i, 565
LISTER, Sir JOSEPH (Path. Trans.)
Lactic acid fermentation, i, 23
LITTEN, of Berlin
Relapsing fever i, 158 *et seq.*
LIVEING, EDWARD (Megrim, Sick Headache, and some Allied Disorders, 1873)
Paroxysmal neuroses, i, 778
Migraine, i, 780 *et seq.*
Paroxysmal vertigo, i, 816
Hiccough, ii, 339
LIVEING, ROBERT (Gulstonian Lectures on Leprosy, 1873; Handbook of Diseases of the Skin, 2nd ed., 1887)
Rötheln or rubeola, i, 223 *et seq.*
Lichen planus, ii, 902
Pityriasis rubra, ii, 909
Herpes gestationis, ii, 934
Linear atrophy of skin, ii, 991
Leprosy, ii, 1009, 1011
Cheloid, ii, 1016
Elephantiasis teleangiectodes, ii, 1017
Neurotic origin of area, ii, 1029
LOCOCK, Sir CHARLES
Epilepsy, i, 810

Löffler
Cases illustrating cerebral localisation, i, 626
Longmore
Heat-stroke, i, 891
Löschner (Aus dem Kinderspital in Prag, 1860)
Histology of scarlatina, i, 212
Lossen, of Heidelberg
Nephrectomy for renal cancer, ii, 698
Louis (Researches on Phthisis, transl. by Dr Walshe for the New Syd. Soc., 1844; Recherches sur la fièvre typhoide, 1841)
Tubercle, i, 79 *et seq.*
Enteric fever, i, 164 *et seq.*
Yellow fever, i, 357
Tubercle of larynx, ii, 37 *et seq.*
Emphysema of lungs, ii, 107 *et seq.*
Lucæ, of Berlin
Peripheral facial paralysis, i, 404
Lucas, Clement
Case of foreign body in bronchus, ii, 64
Albuminuria and flat foot, ii, 600
Nephrolithotomy, ii, 688
Tubercular disease of kidney, ii, 691
Lücke ('Geschwülsten,' Pitha's Handbuch)
Melanotic sarcoma, i, 109
Carcinoma, i, 116
Luschka, of Tübingen (Der Kehlkopf des Menschen, 1871)
Anatomy of adductors of larynx, ii, 10
Lutz, of Würtzburg
Pseudo-hypertrophic paralysis, i, 528
Luys (Leçons sur les Maladies du Système Nerveux, 1875)
Locomotor ataxy, i, 538

M.

McBride
Foot-and-mouth disease, i, 381
McCarthy
Lupus erythematosus, ii, 1007
Macculloch (Essay on Remittent and Intermittent Diseases, 1828)
Migraine, i, 787
Macdonald
Yellow fever, i, 357
McDonnell, of Dublin
Reflex movements in paraplegia, i, 439
McGhie
Pityriasis rubra acuta, ii, 908
Mackenzie, Fr. (London Hosp. Reports)
Cholera, i, 282
Mackenzie, Stephen
Melanæmia, i, 346
Chorea and rheumatism, i, 754
Filaria sanguinis hominis, ii, 460
Idiopathic anæmia, ii, 776
Case of purpura, ii, 786
Iodic purpura, ii, 788
Mackenzie, Morell (Manual of the Diseases of the Throat and Nose, 1880–84; and Diseases of Larynx in Reynolds' System)
Diseases of the larynx, ii, 3 *et seq.*
Mackenzie (*continued*)—
Throat cough, ii, 72
Age most liable to quinsy, ii, 306
Granular pharyngitis, ii, 309, 310
Maclachlan (Practical Treatise on the Diseases of Advanced Life, 1863)
Paralysis agitans, i, 732
Maclagan, T. J.
Enteric fever, i, 185
Salicin in rheumatic fever, ii, 825
Maclaren
Paroxysmal insanity, i, 817 *et seq.*
Maclean (Reynolds' System)
Ague, i, 339 *et seq.*
Remittent fever, i, 345
Heat-stroke, i, 891, *et seq.*
Dysentery, ii, 392 *et seq.*
Abscess of liver, ii, 517 *et seq.*
Macpherson, of Calcutta
Delirium tremens, i, 889
Magendie
Intestinal absorption in cholera, i, 289
Hydrophobia, i, 771
Magnan
Hypertrophy of the brain, i, 647
Mahomed (Guy's Hosp. Reports, &c.)
Case of paradoxical temperature, i, 41
Renal pulse, ii, 613 *et seq.*
Prealbuminuric stage in Bright's disease, ii, 628
Cardiac symptoms in renal cirrhosis, ii, 646
Maier (Lehrbuch d. path. Anat. 1871)
Histology of bulbar paralysis, i, 522
Maingault (De la Paralysie diphthéritique, 1860)
Effects of tonsillitis, ii, 307
Malassez (De la Numération des Globules rouges, 1873)
Compte-globule, ii, 748
Parasitic nature of area, ii, 975
Malmsten, of Stockholm
Trichophyton tonsurans, ii, 962
Major
General paralysis of the insane, i, 669
Manson, of Canton
Filaria sanguinis hominis, ii, 459, 700
Manx
Tubercle of choroid, ii, 267
Marc (De la Folie, &c., 1840)
Feigned epilepsy, i, 807
Marcet, Alexander (Essay on Calculous Disorders, 1817)
Xanthin calculus, ii, 659
Marcet, William (On Chronic Alcoholic Intoxication, 1862)
Zinc in alcoholism, i, 885
Marchiafava
Bacillus malariæ, i, 352
Marsh, Howard
Nephrotomy, ii, 687
Marshall
Fracture of ribs mistaken for pleurodynia, ii, 75
Spontaneous perforating point of empyema, ii, 175

MARSON (Reynolds' System)
Smallpox, i, 229 *et seq.*
MARTIN, Sir RANALD
Nitro-hydrochloric acid bath, ii, 491
MARTIN, of Berlin
Nephrectomy for renal sarcoma, ii, 698
MASCHKA, of Prague
Hydrophobia, i, 774
MAURER
Meningitis, i, 697
MAYNE, of Dublin
Cirrhosis of the lung, ii, 153
MECKEL
Lardaceous reaction with iodine, ii, 630
MEDING
Hyperpyrexia in rheumatism, ii, 832
À MEEK'REN (Obs. Med-Chir., 1682)
Dermatolysis, ii, 993
MEINEL
Silica in lung, ii, 243
MENDELSSOHN (Der Mechanismus der Respiration und Circulation, 1845)
Emphysema of the lungs, ii, 108
MÉNIÈRE
Paroxysmal vertigo, i, 813 *et seq.*
MERKEL, G. (Ziemssen's Handbuch)
Epidemic meningitis, i, 696
Pneumonoconiosis, ii, 243 *et seq.*
MERSON (West Riding Asylum Reports)
Epilepsy i, 798
MERYON (Researches on the various Forms of Paralysis, 1864)
Pseudo-hypertrophic paralysis, i, 528
MEYER, WM., of Copenhagen (Med.-Chir. Trans.; Intern. Med. Cong., 1881)
Acute atrophic paralysis, i, 502
Cerebral abscess, i, 636 *et seq.*
Hysteria, i, 835
Adenoid growths in pharynx, ii, 310
MEYER-HÜNI
Anatomy of pertussis, ii, 290
MICHAUD (Sur la Meningite et la Myelite, 1871)
Myelitis, i, 451
Spinal caries, i, 480
Compression-paraplegia, i, 484 *et seq.*
MICHEL
Galvanic cautery in pharyngitis, ii, 310
Course of fibres in chiasma, i, 574
MIDDELDORPFF
Harpoon for examination of muscle during life, i, 508
MILNE-EDWARDS, ALPHONSE
Artificial rickets, ii, 858
MILROY (Reynolds' System)
Plague, i, 303
MITCHELL, WEIR, of Philadelphia
Peripheral paralysis, i, 392 *et seq.*
Massage, i, 516, *footnote*
Post-hemiplegic chorea, i, 576
Function spasm, i, 720 *et seq.*
Hysteria, i, 838
Experiments on diabetic cataract, ii, 717
MITCHELL (Med.-Ch. Trans., 1813)
Torticollis from diseased teeth, i, 717
MOERS and MÜCK
Urine in mollities ossium, ii, 864
MÖHL, of Copenhagen
Varicella and variola, i, 244
MÖLLENDORFF
Migraine, i, 783 *et seq.*
MOREAU
Intestinal secretion, i, 287
MOREHEAD (Disease in India, 1856)
Heat-stroke, i, 892 *et seq.*
Abscess of liver, ii, 515 *et seq.*
MORGAGNI (De Sedibus et Causis Morborum per Anatomen indagatis, 1761)
"Bronchial polypi," ii, 125
Pneumothorax, ii, 189
Earthy concretions in phthisis, ii, 219
MORGAN, of Dublin
Syphilis, i, 312
DE MORGAN (Holmes' System, &c.)
Cancer, i, 99
Erysipelas, i, 250 *et seq.*
Spasmodic torticollis, i, 720
MORRIS, HENRY (Surgical Dis. of Kidney)
Hydronephrosis, ii, 675 *et seq.*
Nephrolithotomy, ii, 687
MORRIS, MALCOLM
Piedra, ii, 977
Peculiar papular eruption in diabetes, ii, 994, *footnote*
MORRISON
Molluscum contagiosum, ii, 952
MORROW, of New York
Eruptions from quinine, ii, 940
MORTON, RICHARD (Pyretologia, 1694)
Scarlatina, i, 209
Phthisis ab hæmoptoë, ii, 215
MORTON, THOMAS
Ulceration of tongue in pertussis, ii, 287
MOSLER (Path. u. Ther. d. Leukämie, 1872; Ziemssen's Cyclopædia)
Typhus, i, 146
Leuchæmia, ii, 759
MOUTARD-MARTIN
Anæmia of spinal cord, i, 454
MOXON (Wilks and Moxon's Pathology; Guy's Hosp. Rep.; Croonian Lectures)
Clinical nomenclature of disease, i, 5
Cancer i, 101
Morbid anatomy of enterica, i, 167
Spleen in relapsing fever, i, 258
Cholera, i, 285 *et seq.*
"Glossy skin," i, 399
Peripheral facial paralysis, i, 402
Influence of the circulation upon the nervous system, i, 455
Spinal caries, i, 479
Muscles in paralysis from lead, i, 506
Insular sclerosis, i, 547 *et seq.*
Aphasia, i, 579 *et seq.*
Dual function of the brain, i, 581
Red softening of the brain, i, 634
Ependyma in hydrocephalus, i, 652
Spasmodic torticollis, i, 719 *et seq.*
Tetany, i, 736 *et seq.*
Collapse of lung from pleuritic effusion, ii, 167
Double pneumothorax after tracheotomy, ii, 190
Localisation of phthisis, ii, 202 *et seq.*

MOXON (*continued*)—
Cancer of stomach, ii, 356 *et seq.*
Lymphosarcoma of intestine, ii, 382
Theory of jaundice, ii, 507
Urinary reaction from potassium iodide, ii, 569
Albuminuria of adolescents, ii, 600
Suppurative nephritis, ii, 683
Addison's disease, ii, 738
Latent deposit of urate of soda in the joints in gout, ii, 801
Anatomy of acute rheumatism, ii, 811
MÜLLER, JOHANNES (Der Feinere Bau u. die Formen d. kr. Geschwülste, 1838)
Tumours, i, 97
MUNK, of Berlin
Cerebral localisation, i, 623
MURCHISON (Treatise on the Continued Fevers of Great Britain, 1862; Croonian Lectures, 1874, &c.)
Typhus, i, 132 *et seq.*
Relapsing fever, i, 153 *et seq.*
Enteric fever, i, 169 *et seq.*
Febricula, i, 207
Age most liable to scarlatina, i, 210
Rubeola, i, 223
Erysipelas, i, 252
Paroxysmal vertigo, i, 815
Hæmatemesis, ii, 342, 343
Gastric ulcer, ii, 346
Gastro-colic fistula, ii, 363
Lithæmia, ii, 489 *et seq.*
Case of villous growth of renal pelves and bladder, ii, 665
MURPHY, SHIRLEY
Enteric fever, i, 189
MURRAY, of Newcastle
Cure of abdominal aneurysm, i, 996
MURRELL
Nitroglycerine, i, 917
MUSCROFT, of Pontefract
Glanders, i, 376
MUSSER, of Philadelphia
Idiopathic anæmia, ii, 772

N.

VON NÄGELI (Die niederen Pilze in ihrer Bez. zu den Infections-Krankheiten, 1877)
Pathogenetic microphytes, i, 24 *et seq.*
Cholera, i, 297
NÄGELI, W.
Bacterium lactis, i, 25
NAUNYN, of Königsberg
Myelitis, i, 451
Causation of anæmic bruits, i, 969
NEISSER (Ziemssen's Handbuch)
Classification of lupus, ii, 1003
Bacillus lepræ, ii, 1010
Xerodermia, ii, 1019
NEUMANN, ISIDOR (Lehrbuch der Hautkrankheiten, 1869, transl. by Dr Pullar)
Histology of lichen planus, ii, 902
NEUMANN (*continued*)—
Histology of milium, ii, 949
Onychomycosis, ii, 971
Fungus in area, ii, 975
Tubercular nature of lupus, ii, 1004
Lupus erythematosus, ii, 1007
NETTLESHIP
Urticaria pigmentosa, ii, 936
NIELLY
Yellow fever, i, 357
Dengue, i, 366
VON NIEMEYER, FELIX, of Tübingen (Lehrbuch d. sp. Path. u. Ther. 1865, transl. in America, 1869; Clin. Lect. on Pulm. Consumption, transl. New Syd. Soc., 1870)
Marasmus in pyrexia, i, 48
Tubercle, i, 79
Epidemic meningitis, i, 696
Croup, ii, 26
Acute bronchitis, ii, 99
Pleurisy, ii, 167
Phthisis, ii, 215 *et seq.*
Chlorosis, ii, 769
NIEMEYER, PAUL (Grundriss der Percussion und Auscultation, 1871)
Nomenclature of sounds, ii, 80, 85
NIÈPCE (Traité du Goître et du Crétinisme, 1851)
Cretinism, i, 877
NORTON
Gumma of larynx, ii, 45
NOTHNAGEL, of Jena (Ziemssen's Hdbuch.)
Thrombosis of cerebral arteries, i, 556
Apoplexy, i, 588 *et seq.*
Epilepsy i, 794 *et seq.*
Angina pectoris, i, 912
NUSSBAUM
Pathology of albumuinuria, ii, 598

O.

OBERMEIER, OTTO (Centralblatt f. Med. Wissensch., x, 1873; Virch. Arch., liv)
Spirillum of relapsing fever, i, 159
Vicarious epistaxis, ii, 282
OBERNIER, of Bonn (Ziemssen's Cyclopædia)
Cerebral tubercle, i, 605
— tumour, i, 613 *et seq.*
OERTEL, of Munich (Ziemssen's Cyclopædia)
Diphtheria i, 261 *et seq.*
Experimental membranous tracheitis, ii, 19 *et seq.*
Uræmia, ii, 620 *et seq.*
OERTMANN
Thermometry, i, 37, *footnote*
OGLE, JOHN
Aphasia, i, 580
Hydrophobia, i, 769
OGLE, William (Med.-Chir Trans.)
Anosmia, i, 416 *et seq.*
Aphasia, i, 577
OLIVER, of Harrogate
Danger of injection of morphia, i, 429
Tests for albumen in urine, ii, 597

OLLIVIER (Traité des Maladies de la Moelle Épinière, 1837)
Paraplegia, i, 442 *et seq.*
Myelitis, i, 445 *et seq.*
Hyperæmia of spinal cord, i, 455
Rachialgia, i, 474
Syringomyelus, i, 515
ONIMUS and LEGROS (Traité d'Électricité médicale, 1872)
Artificial production of chorea, i, 747
OPITZ
Scurvy in Rastadt, ii, 783
OPPOLZER, of Vienna
Paralysis of the diaphragm, ii, 70
ORD
Myxœdema, or adult cretinism i, 882
Uric acid crystals, ii, 570
Indigo calculus, ii, 659
Cirrhosis of kidney and gout, ii, 640
Relation of arthritis to ovaritis ii, 844
ORDENSTEIN (Sur la paralysie agitante et la sclérose en plaques généralisie, 1867)
Tremors in insular sclerosis, i, 549
ORTH, of Bonn
Erysipelas, i, 250
OSLER, of Philadelphia
Ulcerative endocarditis, i, 77, 952
OTT
Paralysis of abductors of larynx, ii, 9
OTTER
Health-resorts in phthisis, ii, 257
Microzoa in ague, i, 352
OTTO, ADOLPH, of Breslau
Hæmophilia, ii, 792

P.

PAGENSTECHER, of Wiesbaden (Atlas of Ophthalmic Pathology, text transl. by Dr Gowers, New Syd. Soc., 1873)
Changes in optic disc in cerebral tumour, i, 615 *et seq.*
PAGET, Sir JAMES (Lect. on Surgical Pathology, 1853–70; Clin. Lec., 1875)
Specific disease, i, 20
Inflammation, i, 65 *et seq.*
Chronic pyæmia, i, 78
Tumours, i, 102 *et seq.*
Traumatic brachial paralysis, i, 389
"Glossy skin," i, 398
Paralysis of hypoglossal nerve, i, 414
Case of suppression of urine, ii, 670
Charcot's joint disease, ii, 848
Herpes facialis, ii, 1026
PAGET Sir GEORGE, of Cambridge
Case of muscular power in diabetes, ii, 727
PAPAVOINE (Propositions sur les Tubercles chez les Enfans, 1830)
Tubercular meningitis, i, 675
PARKER, R. W.
Traumatic membranous laryngitis, ii, 19
PARKES (Reynolds' System; Croonian Lectures, 1871; Manual of Hygiene, &c.)
Tetanus, i, 44
Contagion of typhus, i, 142
PARKES (*continued*)—
Ague, i, 346 *et seq.*
Yellow fever, i, 359 *et seq.*
Effects of breathed air, ii, 235
Influenza, ii, 274 *et seq.*
Anatomy of dysentery, ii, 389
Milk in scurvy, ii, 784
PARKINSON (Essay on the Shaking Palsy 1817)
Paralysis agitans, i, 729 *et seq.*
PARROT, JULES (Clinique des Nouveau-nés, 1877)
Bones in congenital syphilis, i, 332
Natiform skull, i, 335
Relation of craniotabes to syphilis and rickets, ii, 859
PARRY, CALEB, of Bath (Inquiry into Angina Pectoris, 1799; Collected Writings, 1825)
Migraine, i, 785 *et seq.*
Epilepsy, i, 809
Cold applications in gout, ii, 809
PASTEUR
Alcoholic and butyric fermentation, i, 23
Attenuation of contagion, i, 30, 375
Prophylaxis of anthrax, i, 375
Treatment of hydrophobia, i, 775
PATERSON, of Leith
Rubeola, i, 223 *et seq.*
Hodgkin's disease, ii, 765
PAUL, F. T., of Liverpool
Treatment of hyperpyrexia, ii, 833
PAVY
Salicylates for ammoniacal urine, i, 470
Chronic diffused cerebritis, i, 646
Hydrocephalus of adults, i, 660
Estimation of uric acid, ii, 574
Cyclic albuminuria, ii, 600
Diabetes, ii, 709, 722 *et seq.*
PAYNE, J. F. (Manual of General Path., 1888; St Thomas's Hospital Reports, &c.)
Cancerous thrombi in heart, i, 101
Local use of lead in dermatitis, ii, 884
Lichen circumscriptus, ii, 898
Psoriasis and pityriasis rubra, ii, 914
Febrile pemphigus, ii, 919
Lupus erythematosus, ii, 1006, *footnote*
Rhinoscleroma, ii, 1008
PEACOCK (Tables of Weights of Organs, 1845; The Influenza of 1847–48; Weight and Dimensions of the Heart, 1863; Lectures on Fever)
Febricula, i, 207
Hypertrophy of the heart, i, 921
Dissecting aneurysm, i, 988
Plastic bronchitis, ii, 128
Chalicosis, ii, 243
Influenza, ii, 275
Obstruction by fæcal masses, ii, 409
PEARSON
Origin of pigmentation of lung, ii, 241
PEITAVY
Excision of rib in paracentesis, ii 188
PENNINGTON, Sir ISAAC
Hydrophobia, i, 774
PERLS
Emphysema of lungs, ii, 111

PERRY, of Glasgow
Typhus, i, 141
PETER, MICHEL (Leçons de Clinique Médicale, 1877)
Local temperature in inflammation, i, 59
Diphtheria, i, 264
Angina pectoris, i, 911
Croup, ii, 29
Local temperature in pleurisy, ii, 172
PETHERTON
Cretinism in Somerset, i, 877
VON PETTENKOFER, of Münich (Verbreitungsart der Cholera, 1871)
Soil-water theory of cholera, i, 296
PETERS
Acetonæmia, ii, 719
PFLEGER
Erysipelas, i, 254
PICK, of Prague
Contagion of syphilis, i, 311
PICOT and D'ESPINE (Maladies de l'Enfance, 1873)
Hypertrophy of the brain, i, 647
PIERRET
Pseudo-hypertrophic paralysis, i, 529
Locomotor ataxy, i, 539
PINCUS, of Berlin
Treatment of alopecia, ii, 975
PINEL, PHILIPPE (Traité sur l'Aliénation mentale, 1804, transl. by Dr Davis)
Vicarious hæmoptysis, ii, 219
PIORRY (De la Percussion Médiate, 1828)
Migraine, i, 783 *et seq.*
Percussion of thorax, ii, 76
Pleurisy, ii, 169
PLENCK, of Vienna (Doctrina de Morbis Cutaneis, 1780)
Classification of skin diseases, ii, 866
The cure of acne, ii, 945
PLINY (C. Plinius Secundus, ob. A.D. 70)
Zona, ii, 1025
PLUMBE, SAMUEL (Practical Treatise on Diseases of the Skin, 1824)
Erythema nodosum, ii, 934
Epilation in sycosis, ii, 955
POINCARÉ and BONNET (Recherches sur la Paralysie Générale, 1869)
General paralysis of the insane, i, 669
POLAND, ALFRED (Holmes' System; Guy's Hosp. Reports)
Glanders, i, 379
Tetanus, i, 758 *et seq.*
POLAND, JOHN (Path. Trans.)
Anthrax, i, 373
POLLENDER
Bacillus anthracis, i, 368
Prognosis in phthisis, i, 969 *et seq.*
POLLOCK, GEORGE
Case of gastric concretion, ii, 367
POLLOCK, J. E. (Elements of Prognosis in Phthisis, 1865)
Phthisis, ii, 226 *et seq.*
Fistula in ano, ii, 227
Suckling as a cause of phthisis, ii, 237
Tea in phthisis, ii, 255
PONFICK, of Rostock
Tubercle, i, 92
Actinomycosis, i, 383
Tubercle of choroid, ii, 267
POORE (Text-book of Electricity, 1876)
Paralysis of serratus magnus, i, 391
Writers' cramp, i, 723, *et seq.*
Malformation of larynx, ii, 59
POSNER
Albuminuria, ii, 598
POTT, PERCIVAL (Remarks on Palsy of the lower limbs found to accompany Curvature of the Spine, 1779, &c.)
Abscess of liver, i, 74
Spinal caries, i, 478
POWELL, DOUGLAS
Mechanism of pneumothorax, ii, 195
Pulmonary aneurysms, ii, 217
POWER, WM. HY. (Reports to Privy Council)
Epidemic enterica from milk, i, 198
PRÉVOST
Hemiplegia, i, 570
PROUT (The Nature and Treatment of Stomach and Renal Diseases, 1821–43)
Biliary colic, ii, 511
Renal calculi, ii, 668 *et seq.*
Chyluria, ii, 699 *et seq.*
Diabetes, ii, 713 *et seq.*
Theory of rheumatism, ii, 817
PURSER, of Dublin
Onychomycosis, ii, 971
Favus in a cat, ii, 972
PURVES, LAIDLAW
Histology of inflammation, i, 54

Q.

QUAIN
Hæmatoma of dura mater, i, 711
Fibroid disease of the heart, i, 933
Fatty degeneration of the heart, i, 940
QUINCKE, of Bern ('Perniciöse Anämie,' in Volkmann's Sammlung)
Estimate of amount of blood, ii, 748
Iron in the tissues in anæmia, ii, 750

R.

RADCLIFFE, C. B. (Lectures on Epilepsy, &c., 1864; Reynolds' System)
Hyperæmia of spinal cord, i, 456
Hysterical paraplegia, i, 459 *et seq.*
Rachialgia, i, 476 *et seq.*
Locomotor ataxy, i, 544
Spasmocic torticollis, i, 719
Chorea, i, 745
Epilepsy, i, 794
RADCLIFFE, NETTEN (Reports to Privy Council)
Enteric fever, i, 198
Cholera, i, 290 *et seq.*

RAINEY, of St Thomas's Hospital
Histology of emphysema, ii, 107
Dumb-bell crystals, ii, 570
RAKE, BEAVEN, of Trinidad
Leprosy, ii, 104
RALFE (Inquiry into the General Pathology of Scurvy, 1877)
Urine in scurvy, ii, 786
RAMAZZINI (De Morbis Artificum, 1703)
Phthisis from noxious trades, ii, 239
RAMMELL
Report on sanitary condition of Banbury and Salisbury, ii, 248 *et seq.*
RAMSKILL (Reynolds' System)
Paralysis agitans, i, 734
Epilepsy, i, 805
Paroxysmal vertigo, i, 812 *et seq.*
RANSOM, of Nottingham (Reynolds' System)
Intestinal worms, ii, 449
RANSOME, of Manchester (Measurement of Movements of Chest, 1869)
Stethometer, ii, 93
RANVIER (Traité technique d'Histologie, 1875)
Inflammation, i, 61
RASMUSSEN, of Copenhagen (Edin. Med. Jour., Nov., 1868)
Pulmonary aneurysm in phthisis, ii, 217
RATJEN
Syphilitic stenosis of trachea, ii, 64
RAYER (Tr. des Mal. de la Peau, 1826; Tr. des Mal. des Reins, 1839)
Smallpox, i, 228
Case of suppression of urine, ii, 670
Hydronephrosis, ii, 674, 676
Strophulus, ii, 900
RAYMOND, PAUL
Urticaria pigmentosa, ii, 936
RAYNAUD, MAURICE (De l'Asphyxie locale et de la Gangrène symmetrique des Extrémités, 1862)
Symmetrical gangrene, ii, 590, 930
VON RECKLINGHAUSEN, of Strassburg (Die Lymphgefässe, 1862; Virchow's Archiv)
Inflammation, i, 52 *et seq.*
Diphtheritic inoculation, i, 263
Lymph-channels between pleuræ and peritoneum, ii, 180
Neuromata, ii, 1014
REDTENBACHER
Urine in ague, i, 340
REES, OWEN (Croonian Lectures, 1856; Guy's Hosp. Reports)
Acid and alkaline urine, ii, 581
Renal pulse, ii, 611
Encystment of renal calculi, ii, 686
Peculiarities of diabetic sugar, ii, 724
Lemon-juice in rheumatism, ii, 823
REHDER
Cause of renal dropsy, ii, 607
REHN
Relation of craniotabes, congenital syphilis and rickets, ii, 859
REID, J. C.
Treatment of collapse, i, 17
REID, JAMES (Infantile Laryngismus, 1849)
Laryngismus stridulus, ii, 15
REINHARDT (in Traube's Beiträge)
Tubercle, i, 95
REMAK, ROBERT, of Berlin (Diagnostische u. Pathogenetische Untersuchungen, 1845
Carcinoma, i, 120
Progressive muscular atrophy, i, 517
Apoplexy, i, 604
Acute pneumonia, ii, 137
REVERDIN, of Geneva
Cachexia strumipriva, i, 882
REYNAUD
Tactile fremitus, ii, 92
The pleuritic rub, ii, 164
REYNOLDS, RUSSELL (articles in his System of Medicine, 1866–78, &c.)
Nature of disease, i, 6
Hysterical paraplegia, i, 459
Electricity in hemiplegia, i, 604
Spasmodic torticollis, i, 716
Writers' cramp, i, 724
Chorea, i, 747
Epilepsy, i, 793 *et seq.*
Hysteria, i, 822 *et seq.*
RHAZES (Abubekr Ar-razi, of Khorassan, fl. A.D. 900; Treatise on Smallpox and Measles, transl. by Dr Greenhill, 1848)
Morbilli, i, 217
Variola, i, 227
RIBBERT
Albuminuria, ii, 598
RICHARDSON, B. W.
Intermittent pulse, i, 900
Lactic acid and rheumatism, ii, 817
RICKARDS, of Birmingham
Replacement of kidney by fat after destruction by calculus, ii, 674
RICORD (Traité des Mal. vénériennes, 1851)
Syphilis, i, 307 *et seq.*
RIEGEL, of Cologne (Ziemssen's Cyclopædia)
Paralysis of laryngeal abductors, ii, 7
Diseases of trachea and bronchi, ii, 62
Bronchitis, ii, 95 *et seq.*
Plastic bronchitis, ii, 125
Duration of catarrhe sec, ii, 105
Cause of "catching cold," ii, 121
RIETZ
Experimental tracheitis, ii, 19
RILLIET and BARTHEZ (Maladies des Enfants, 1843–55)
Acute atrophic paralysis, i, 497
Cerebral tubercle, i, 606
Hydrocepalus, i, 650
Tubercular meningitis, i, 681 *et seq.*
Chorea in pregnancy, i, 746
Croup and diphtheria, ii, 18 *et seq.*
Pertussis, ii, 289
Invagination of intestine, ii, 410
RINDFLEISCH, of Würzburg (Lehrbuch der Path. Gewebelehre, 1866, transl. by Dr Baxter; and in Ziemssen's Handbuch)
"Epithelial infection," i, 67
Fibroid change in tubercle, i, 85
Melanotic sarcoma, i, 108
Histology of smallpox, i, 232
Diphtheria, i, 266 *et seq.*

RINDFLEISCH (*continued*)—
Cerebral softening, i, 559
— tubercle, i, 606
Red softening of brain, i, 634
Cerebral abscess, i, 639
Meningitis, i, 679
Bronchitis, ii, 98
Emphysema of lungs, ii, 107 *et seq.*
Acute pneumonia, ii, 130 *et seq.*
Anthracosis, ii, 242
Abscess of liver, ii, 516
Circulation in cirrhosis, ii, 523, 526
Fatty liver, ii, 542
Embolism from renal cancer, ii, 697
Pathology of Addison's disease, ii, 739
Histology of rickets, ii, 854
— of mollities ossium, ii, 864
— of lupus, ii, 996
Myoma of skin, ii, 1014

RINGER (Reynolds' System; Handbook of Therapeutics, 1869)
Measles, i, 218 *et seq.*
Ague, i, 340
Laryngismus stridulus, ii, 17
Relation of urea to sugar excreted in diabetes, ii, 715
Hyperpyrexia in rheumatism, ii, 819
Actæa racemosa in osteo-arthritis, ii, 847
Coster's paste in ringworm, ii, 968

RISCH
Œdematous laryngitis, ii, 47

RISEL
Addison's disease, ii, 740

RITTER VON RITTERSHAIN (Die Path. u. Ther. der Rachitis, 1863)
Rickets, ii, 853 *et seq.*

RIVOLTA
Actinomycosis, i, 382

ROBERTS, SYDNEY, of Philadelphia
Charcot's joint disease, ii, 848

ROBERTS, WILLIAM, of Manchester (Essay on Wasting Palsy, 1858; Pract. Treatise on Urinary and Renal Diseases, 1865; and articles in Reynolds' System)
Progressive muscular atrophy, i, 507
Pseudo-hypertrophic paralysis, i, 528
Diabetes insipidus, ii, 562 *et seq.*
Urinary deposits, ii, 572 *et seq.*
Hæmoglobinuria, ii, 587 *et seq.*
Aseptic bacilluria, ii, 582
Albuminuria, ii, 596 *et seq.*
Cystic disease of kidneys, ii, 651
Urinary calculi, ii, 657 *et seq.*
Suppression of urine, ii, 670 *et seq.*
Chyluria, ii, 701
Moveable kidney, ii, 706
Diabetes, ii, 709 *et seq.*

ROBERTSON, ARGYLL, of Edinburgh
Pupils in tabes dorsalis, i, 536

ROBIN (Traité du Microscope, 1871, &c.)
Histology of progressive muscular atrophy, i, 512
Psammoma, i, 608

ROCHAUX (Recherches sur l'Apoplexie, 1833)
Cerebral softening, i, 565

ROESCH
Œnomania, i, 884

ROESINGH
Reflex paraplegia, i, 458

ROGER, HENRI (Recherches cliniques sur les Maladies de l'Enfance, 1872)
Chorea, i, 750
Pertussis, ii, 291

ROKITANSKY, of Vienna (Lehrbuch der Path. Anat. 1849–61)
Cerebral hæmorrhage, i, 563
Yellow softening of brain, i, 610
Hydrocephalus of childhood, i, 657
Emphysema of lungs, ii, 106
Cyanosis inimical to phthisis, ii, 254
Gastric ulcer, ii, 344
Lardaceous disease, ii, 630, *footnote*

ROLLET (Traité des Maladies vénériennes, 1865)
Syphilis, i, 310

ROLLO, JOHN (Cases of Diabetes Mellitus, 1798)
Animal diet in diabetes, ii, 728

ROMBERG, of Berlin (Lehrbuch der Nervenkrankheiten, 1851; tr. by Dr Sieveking)
Paralysis of the fifth nerve, i, 414
Tic douloureux, i, 421
Rachialgia, i, 474 *et seq.*
Progressive muscular atrophy, i, 507
Locomotor ataxy, i, 530 *et seq.*
Cerebral tumour, i, 612
Chorea, i, 741
Epilepsy, i, 795
Hysteria, i, 836
Hypochondriasis, i, 843 *et seq.*
Asthmatic aura, ii, 292

ROSE, of Zurich
Tetanus, i, 758 *et seq.*
Tracheal stenosis from goitre, ii, 60

ROSE, COOPER
Fluid of hydronephrosis, ii, 675

ROSENBERG
Nephrotomy, ii, 687

ROSENTHAL, MORITZ (Hdbh. d. Diagnostik u. Therapie d. Nervenkrankheiten, 1870)
Chorea, i, 741 *et seq.*
Hysterical catalepsy, i, 830
Cause of "catching cold," ii, 121

ROSSBACH (Int. Med. Congr., 1881)
Tubercle of the larynx, ii, 42
Anatomy of pertussis, ii, 290

ROSTAN
Asthma, ii, 292

ROY, of Cambridge
Anthrax, i, 375

ROYER and DAMASCHINO
Acute atrophic paralysis, i, 500

RÜHLE, of Bonn (Ziemssen's Handbuch)
Emaciation in phthisis, ii, 211 *et seq.*
Acute miliary tuberculosis, ii, 265 *et seq.*

RÜHLMANN
Paralysis of abductors of larynx, ii, 8

RUNEBERG
Albuminuria, ii, 599

RYLE
Rubeola, i, 224, *footnote*

RYNECK, of Gratz
Experiments on inflammation, i, 57

S.

SALISBURY, of Ohio, U.S.A.
Contagium of ague, i, 351
SALKOWSKI
Urobilin ii, 567
Estimation of uric acid in urine, ii, 572
Tests for albumen in urine, ii, 595
Uræmia, ii, 623, *footnote*
SALTER, HYDE (Asthma, 1860, and in Reynolds' System)
Plastic bronchitis, ii, 125
Asthma, ii, 294 *et seq.*
Purpura, ii, 788
SALTER, S. J. A.
Reflex paralysis, i, 390
Reflex neuralgia, i, 421
Migraine, i, 789
SÄMISCH, of Bonn
Cerebral localisation, i, 618
SANDERS, of Edinburgh
Peripheral facial paralysis, i, 404
Amnesic aphasia, i, 582
"Waxy" disease, ii, 630, *footnote*
Lipæmia and fat-embolism in diabetes, ii, 721
SANDERSON, BURDON (Reports to Privy Council; Holmes' Surgery)
Contagion, i, 22
Inflammation, i, 55
Tubercle, i, 81 *et seq.*
Diphtheria, i, 261 *et seq.*
Cholera, i, 291
Epidemic meningitis, i, 692 *et seq.*
SANGSTER
Urticaria pigmentosa, ii, 936
Molluscum contagiosum, ii, 952
Histology of lymphangioma, ii, 1017
SANKEY
General paralysis of the insane, i, 664
SANNÉ (Étude sur le Croup après la Trachéotomie, 1869)
Diphtheria in boys and girls, ii, 28
Tracheotomy in croup, ii, 32
SAPPEY
Portal circulation in cirrhosis, ii, 526
SAUNDBY, of Birmingham
Renal pulse, ii, 514
Treatment of hæmaturia, ii, 666
SAUNDERS, WILLIAM, of Greenwich
Delirium tremens, i, 887
SAUVAGES (Nosologia methodica juxta Sydenhami mentem, 1768)
Measles, i, 217
Scleotyrbe festinans, i, 731
Alopecia areata, ii, 975
SAVAGE, G. H. (Guy's Hosp. Reports)
Chronic diffused cerebritis, i, 647
Insanity, i, 846–875
SAVORY
Erysipelas, i, 250
SCANZONI, of Würzburg
Hysteria, i, 838
SCARPA, of Pavia (Sull' Aneurisma, 1804, transl. by Wishart)
Aneurysm, i, 988
SCHÄFER
Cerebral localisation, i, 623
SCHECK
Paralysis of recurrent laryngeal, ii, 5
SCHENCK
Whooping-cough, ii, 286
SCHIFF, of Florence
Sensory tracts of spinal cord, i, 436
Experimental glycosuria, ii, 725
SCHIFFER
Anæmia of spinal cord, i, 453
SCHMIDT, C. C.
Hydrocephalus of childhood, i, 650
Fluid of renal dropsy, ii, 608
The bones in mollities ossium, ii, 863
SCHMIDT, HERMANN
Choked disc from cerebral tumour, i, 615
SCHNITZLER (Wiener Klinik., i, 6, 1875)
Mediastinal abscess, ii, 62
Emphysema of the lungs, ii, 110
SCHÖNLEIN, of Berlin (Müller's Archiv, 1836; Allg. u. spec. Path. u. Ther., 1837)
Peliosis rheumatica, ii, 813
Achorion Schönleinii, ii, 971
Urticaria pigmentosa, ii, 1025
SCHOTTIN
Cutaneous excretion of urea in cholera, ii, 622
SCHRADER, of Hamburg
Hydrophobia, i, 772
SCHRÖTTER, of Vienna (Ziemssen's Hdbuch.)
Laryngeal perichondritis, ii, 52
Laryngoscopic view of trachea, ii, 66
SCHÜLE
General paralysis of the insane, i, 667
SCHÜLEIN
Relation of internal to external temperature in ague, i, 45
Surface temperature in pneumonia, measles, and scarlatina, i, 46; ii, 135
SCHULTZ
Salivation in bulbar paralysis, i, 520
SCHULZE, E.
Locomotor ataxy, i, 542
SCHUNK
Oxaluric acid in health, ii, 576
SCHÜPPEL, of Tübingen (Untersuch ü. Lymphdrüsentuberkulose, 1871, &c.)
Tubercle, i, 81 *et seq.*
Syringomyelus, i, 515
Locomotor ataxy, i, 540
SCHWANN, of Liège (Micr. Untersuchungen, 1839, transl. by Mr Spencer Smith, 1847)
Cellular nature of animal tissues, i, 52
SCHWEIGGER
Choked disc from tumor cerebri, i, 615
SCHWIMMER (Ziemssen's Handbuch)
Lupus erythematosus, ii, 1006, *footnote*
Rhinoscleroma, ii, 1008
SCUDAMORE, Sir CHARLES (Treatise on Gout and Gravel, 1823)
Relation of diabetes to gout, ii, 715
Gout, ii, 798 *et seq.*
SEATON (Reports to Privy Council; Clin. Trans.)
Consequence of enteric fever, i, 193
Epidemic febricula, i, 208

SEATON (*continued*)—
Vaccination, i, 242
SEDGWICK, WM. (Nature of Cholera, 1866)
The collapse of cholera, i, 288
SÉE, GUSTAVE (De la Chorée, &c., 1851)
Chorea, i, 744 *et seq.*
SEELIGMÜLLER
Thomsen's disease, i, 734 *et seq.*
SEITZ, of Giessen
Acute bronchitis, ii, 99
"Metamorphosing murmur," ii, 222
SELTMAN
Pneumonokoniosis, ii, 240
SEMON, FELIX
Diseases of the larynx, ii, 5 *et seq.*
SEMPLE
Croup and diphtheria, ii, 18
SENATOR, of Berlin (German Clin. transl. New Syd. Soc.; Ziemssen's Handbuch)
Diphtheria, i, 259 *et seq.*
Dyspnœa, ii, 69
Experiments on glycosuria, ii, 265
Acute rheumatism, ii, 813 *et seq.*
Gonorrhœal synovitis, ii, 835
Arthritis deformans, ii, 846
Rickets, ii, 855
SHATTUCK, of Boston, U.S.A.
Enteric fever, i, 164
SHAW, L. E. (Guy's Hosp. Rep.)
Statistics of paralysis agitans, i, 732,
— of valvular disease, i, 984, 985
— of intestinal obstruction, ii, 434
Hæmorrhage from salicylates, ii, 830
SHOEMAKER, of Philadelphia
Oleates, ii, 966, footnote
SIBSON (Reynolds' System)
Statistics of cardiac inflammation in rheumatism, ii, 814 *et seq.*
SIEVEKING (transl. of Romberg)
Histrionic spasm, i, 715
SILVESTER, H. R. (True Physiological Method of Restoring, &c., 1858)
Artificial respiration, i, 13
SIMON, GUSTAV, of Heidelberg (Die Hautkrankheiten, 1848; Chirurgie der Nieren, 1876)
Histology of measles, i, 219
— of clavus, ii, 982
— of leucodermia, ii, 1022
SIMON, JOHN (Holmes' System; Reports to Privy Council; Lectures on Pathology)
Heat-production in inflammation, i, 59
Tubercle, i, 87
Cancer, i, 101
Yellow fever, i, 361
Cholera, i, 282 *et seq.*
Cysts in cirrhosis of kidney, ii, 643
SIMON, THEODOR, of Hamburg
Smallpox, i, 228
Syringomyelus, i, 467, 515
SINKLER
Acute atrophic paralysis, i, 501
SKODA, of Vienna (Abhandlung ü. Percussion u. Auscultation, 1850, translated by Markham)
Disseminated sclerosis, i, 548
Consonance in respiratory sounds, ii, 88
SKODA (*continued*)—
Turpentine inhalations in gangrene of the lung, ii, 162
Subtympanitic percussion-note (Skodaic resonance) in pleurisy, ii, 170
SMITH, ALDER (Ringworm, its Diagnosis and Treatment, 1885)
Treatment of ringworm, ii, 967 *et seq.*
SMITH, ARCHIBALD
Yellow fever, i, 361
Mountain air in phthisis, ii, 258
SMITH, EUSTACE (Disease in Children, 1884)
Convalescence from scarlatina, i, 216
Morbilli sine catarrho, i, 220
Acute miliary tuberculosis, ii, 263
Whooping-cough, ii, 286
Treatment of infantile scurvy, ii, 787
Rickets, ii, 855 *et seq.*
Quinine and guaiacum in eczema, ii, 891
SMITH, FRANK, of Sheffield
Hammer-palsy, i, 728
Red atrophy of liver, ii, 535
SMITH, SHINGLETON, of Clifton
Heat-stroke, i, 892
Sympathetic in exophthalmos, i, 1012
SMITH, TYLER (Manual of Obstetrics, 1858)
Puerperal eclampsia, i, 806
SMITH, WALTER, of Dublin
Phosphates in urine, ii, 567
SNELLEN
Paralysis of the fifth nerve, i, 415
SNOW, JOHN (The Mode of Communication of Cholera, 1849–55)
Water supply and cholera, i, 293
SOCOLEFF
Bronchitis, ii, 97
SOLLY, SAMUEL
Mollities ossium, ii, 864
SOMMERBRODT
Artificially produced pneumonia, ii, 142
Allorrhythmia, i, 901
SOTNITSCHEWSKY
Thickening of arteries in Bright's disease, ii, 613
SOUTHEY (Clin. Trans., &c.)
Raymond's disease, ii, 590
Febrile pemphigus, ii, 919
SPARKS
Acute pemphigus, ii, 919
SPÄTH
Locomotor ataxy, i, 540
SPEAR (Loc. Gov. Reports, 1880, 1883)
Anthrax, i, 373
SPENCER, W. H., of Clifton
Acute tuberculosis, i, 90
SPENDER
Constipation, ii, 376
Early symptoms of osteoarthritis, ii, 843
SQUIRE, BALMANO (Reynolds' System)
Scarification in acne, ii, 981
— in lupus, ii, 1005
SQUIRE, WILLIAM (Reynolds' System)
Rötheln, i, 223 *et seq.*
Diphtheria, i, 276
Incubation of whooping-cough, i, 1022
ST CYR
Tinea favosa, ii, 972

STANLEY, EDWARD
Reflex paraplegia, i, 456
STEDMAN
Dengue, i, 363 *et seq.*
STEELE, J. C., of Guy's Hospital
Relapsing fever, i, 153
STEFFEN (Ziemssen's Handbuch)
Laryngismus stridulus, ii, 14 *et seq.*
Broncho-pneumonia, ii, 148
Pertussis, ii, 287
STEINER, of Prag. (Ziemssen's Handbuch)
Croup, ii, 28 *et seq.*
STEINBURG, of Copenhagen
Syphilitic cerebral artereitis, i, 556
STENSON, NICOLAUS (De Musculis et Glandulis, 1664)
Experimental paraplegia, i, 453
STEVENSON, THOS. (Guy's Hosp. Rep.)
Diagnosis between poison and apoplexy, i, 593
Reddish sweat in tetanus, i, 759
Strychnia and brucia in urine, i, 763
Fatty degeneration of heart, i, 940
Lead in the viscera, ii, 374
STEWART, A. P.
Recognition of enteric fever, i, 164
STEWART, CHARLES
Histology of lymphangioma, ii, 1017
STEWART, GRAINGER, of Edinburgh (Pract. Treatise on Bright's Disease, 1868, &c.)
Peripheral neuritis, i, 430
Success of repeated paracentesis, ii, 530
Albuminuria in acute yellow atrophy of the liver, ii, 534
Lardaceous disease of kidneys, ii, 635
Urine in cirrhosis of kidneys, ii, 643
Treatment of acute nephritis, ii, 654
STILLÉ, of Philadelphia (Epidemic Meningitis)
Cerebro-spinal meningitis, i, 697
STILLING, B. (Untersuchungen ü. die Spinal-Irritation, 1840)
Hyperæmia of spinal cord, i, 455
Rachialgia, i, 473
STIRLING, A. N.
Albuminuria in boys, ii, 600
STOCKER, JAMES, of Guy's Hospital
Prognosis in fever, i, 135
Odour of smallpox, i, 228
Treatment of an epileptic fit, i, 809
— of hysteria, i, 842
STOCKVIS
Albuminuria and blood-pressure, ii, 599
STOFFEN
Moveable kidney in children, ii, 706
STOKES, of Dublin (Dis. of the Heart and Aorta, 1854; Lect. on Fever, 1874)
Typhus, i, 136
Exophthalmic goitre, i, 1010
Pulmonary auscultation in laryngeal disease, ii, 86
Thorax in emphysema, ii, 113
Acute pneumonia, ii, 133
STONE (Croonian Lectures, 1879; St Thomas's Hosp. Reports)
Hypertrophy of heart, i, 922
Physical cause of ægophony, ii, 91
STÖRCK (Pitha's Handbuch; Volkmann's Sammlung)
Diseases of the larynx, ii, 4 *et seq.*
Asthma, ii, 297
STOWERS, J. H.
Scarification in acne rosacea, ii, 981
— in lupus, ii, 1005
STRATTON
"Anthracosis," ii, 241
STRICKER, of Vienna (Vorlesungen ü. allgemeine u. experimentelle Pathologie, 1877)
Inflammation, i, 54
STURGES, OCTAVIUS (Chorea, 1881; Natural History of Pneumonia, 1877)
Pathology of chorea, i, 749
Pneumonia, ii, 141
SUTTON, H. G. (Medical Pathology, 1887; Guy's Hospital Reports, &c.)
Arterio-capillary fibrosis, i, 613
Cerebral abscess, i, 636
Treatment of rheumatism, ii, 823
SUTTON, J. B.
Phthisis in quadrumana, ii, 255, *note*
SUTTON, THOS., of Greenwich (Tracts on Delirium Tremens, &c., 1813)
Delirium tremens, i, 885, 887
VAN SWIETEN (Commentaria in Boerhaavii Aphorismos, 1765)
Clinical thermometer, i, 37
SYDENHAM, THOS., ob. 1689 (Opera omnia Syd. Soc., 1844)
Scarlatina, i, 209
Measles, i, 217 *et seq.*
Smallpox, i, 230
Chorea, i, 740
Hysteria, i, 839
Peripneumonia notha, ii, 94
Pertussis, ii, 291
Scurvy, ii, 780
Gout, ii, 797 *et seq.*
SYMONDS, C. J.
Intubation of œsophagus, ii, 316
Herpes and rigors, ii, 932, *footnote*

T.

TAIT, LAWSON
Cholecystotomy, ii, 511
TAPPEINER
Experiments on tubercle, i, 91
TAYLOR, FREDK. (Guy's Hosp. Rep., &c.)
Myelitis, i, 446
Tubercle of spinal cord, i, 467
Syringomyelus, i, 467, 515
Rachialgia, i, 476
Chronic spinal meningitis, i, 494
Cerebral localisation, i, 619
Chronic diffused cerebritis, i, 647
Meningitis, i, 703
Chorea, i, 745
Tetanus, i, 767
Hydrophobia, i, 773
Pulsation of liver, i, 965
Cardiac sounds transmitted by a vomica, ii, 222

TAYLOR, FREDERICK (*continued*)—
Cutaneous excretion of urea in Bright's disease, ii, 622
Cases of Addison's anæmia, ii, 772
Bullous erythema, ii, 935
Histology of ringworm, ii, 964
TAYLOR, R. W., of New York
Xerodermia, ii, 1019
TEALE, J. W., of Scarboro' (Clin. Tr., 1875)
Paradoxical temperature, i, 41
TEALE, THOS. PRIDGIN, sen., of Leeds (Treatise on Neuralgic Diseases, 1829)
Rachialgia, i, 473
TERRILLON (De l'Expectoration albumineuse après la Thoracentèse, 1873)
Profuse expectoration after paracentesis thoracis, ii, 185
THIERSCH, CARL, of Leipzig (Der Epithelialkrebs, 1865)
Melanotic sarcoma, i, 109
Cancer, i, 118 *et seq.*
Experiments on cholera, i, 291
THIN (Edin. Med. Jour.; Proc. Royal Soc.; Path. and Tr. of Ringworm, 1888)
Histology of corneitis, i, 61
Blood-vessels in purpura, ii, 791
Molluscum contagiosum, ii, 952, *note*
Bromidrosis, ii, 960
Locality of ringworm-fungus, ii, 964
Bacterium decalvans, ii, 976
Histology of lupus, ii, 996
Lupus erythematosus, ii, 1007
Bacillus lepræ, ii, 1011
Rodent ulcer, ii, 1020
THIRIAL (Gazette méd. de Paris, 1845)
Sclerema, ii, 988
THOMA, of Heidelberg (Ueberwanderung farbloser Blutkörperchen, 1873; Virchow's Archiv, &c.)
Phenomena of inflammation in warm-blooded animals, i, 57
Histology of cirrhosis of kidney, ii, 643
Histology of leprosy, ii, 1010
THOMAS, of Leipzig (Ziemssen's Handbuch)
Scarlatina, i, 211 *et seq.*
Measles, i, 218 *et seq.*
Rötheln, i, 224 *et seq.*
Varicella, i, 244 *et seq.*
THOMAS, WILLIAM, of Birmingham
Excision of rib in paracentesis, ii, 188
THOMPSON, JOHN ASHBURTON (Free Phosphorus in Medicine, 1874)
Phosphorus in neuralgia, i, 427
THOMPSON, JOSEPH, of Nottingham
Peritonitis from hydronephrosis, ii, 679
THOMPSON, REGINALD
Phthisis ab hæmoptoë, ii, 216 *et seq.*
THOMSEN, of Copenhagen (Arch. f. Psychiatrie, 1876)
Peculiar nervous affection, i, 734
THOMSON, JOHN, of Edinburgh (Account of the Varioloid Epidemic in Scotland, 1820)
Relation of varicella to variola, i, 243
THORNE (Reports of Privy Council)
Enteric fever, i, 194 *et seq.*
THORNER (Deutsch. Archiv, 1878)
Pertussis, ii, 291
THUDICHUM (Report to Privy Council)
Trichina spiralis, ii, 456 *et seq.*
TIBBITS
Myelitis, i, 469
TILESIUS, W. G., of Leipzig
Pemphigus not vesicular, ii, 918
Fibroma molluscum, ii, 1013
TODD (Cycl. Anat. and Phys., vol. iii; Clinical Lectures, 1854, 1860)
Distinction between paralysis and defective coordination, i, 530
Cerebral softening, i, 565
Hemiplegia, i, 568 *et seq.*
Choreic hemiplegia, i, 746
Migraine, i, 791
Epilepsy, i, 798
Hysteria, i, 825
Alcohol in acute pneumonia, ii, 144
TOMES (System of Dental Surgery, 1859–73)
Reflex neuralgia, i, 421
Epilepsy from dental exostoses, i, 805
TOMMASI-CRUDELI, of Rome
Bacterium of ague, i, 351
TOOTH (St Barth. Hosp. Rep.)
Cases of Erb's form of progressive muscular atrophy, i, 525
TOPINARD (De l'Ataxie locomotrice, 1864)
Pains in locomotor ataxy, i, 535, *et seq.*
TOUISSANT, of Toulouse
Attenuated virus, i, 30
TOYNBEE
Ear-cough, ii, 73
TRAUBE, of Berlin (Zusammenhang von Herz und Nieren-Krankheiten, 1856; Gesammelte Beiträge, 1871–1878)
Clinical thermometer, i, 37
Pyrexia, i, 42
Pulsus alternans, i, 901
Râles, ii, 87
Bronchitis, ii, 105 *et seq.*
Post-mortem metallic echo, ii, 195
Phthisis, ii, 216 *et seq.*
Catheterism a cause of putrefaction of urine in paraplegia, ii, 580
Pulse in Bright's disease, ii, 615
Theory of uræmia, ii, 620
Urine in lardaceous disease, ii, 635
Pulse in renal colic, ii, 667, *footnote*
TRAVERS, BENJ. (Inquiry concerning Constitutional Irritation, 1826)
Glanders, i, 377
TREITZ, of Prague
Retroperitoneal hernia, ii, 420
Uræmia, ii, 620
TRIPIER
Artificial rickets, ii, 858
VON TRÖLTSCH, of Würzburg (Krankheiten des Ohres, 1873, transl. by Hinton)
Cerebral abscess from otorrhœa, i, 637
TROUSSEAU (Clinique Médicale de l'Hôtel Dieu, 1865, transl. by Dr Bazire)
Enteric fever, i, 169 *et seq.*
Scarlatina, i, 212, *et seq.*
Measles, i, 218 *et seq.*
Rötheln, i, 223 *et seq.*

TROUSSEAU (*continued*)—
Smallpox, i, 229 *et seq.*
Varicella, i, 244 *et seq.*
Erysipelas, i, 252
Diphtheria, i, 264 *et seq.*
Ague, i, 343 *et seq.*
Peripheral facial paralysis, i, 403
Neuralgia, i, 418 *et seq.*
Point apophysaire of neuralgia, i, 475
Progressive muscular atrophy, i, 507
Bulbar paralysis, i, 517 *et seq.*
Locomotor ataxy, i, 532 *et seq.*
Hemiplegia, i, 574
Aphasia, i, 577 *et seq.*
Apoplexy, i, 589 *et seq.*
Apoplectiform cerebral congestion, i, 596
Hydrocephalus of childhood, i, 653
Meningitis, i, 682 *et seq.*
Spasmodic tic, i, 715
Paralysis agitans, i, 730
Tetany, i, 736 *et seq.*
Chorea, i, 741 *et seq.*
Hydrophobia, i, 770
Migraine, i, 772
Epilepsy, i, 794 *et seq.*
Paroxysmal insanity, i, 819
Angina pectoris, i, 911
Exophthalmic goitre, i, 1009 *et seq.*
Croup and diphtheria, ii, 18 *et seq.*
Tubercle of the larynx, ii, 37
Œdematous laryngitis, ii, 48 *et seq.*
Syncope in pleuritic effusion, ii, 173
Bruit d'airain in pneumothorax, ii, 195
Acute phthisis, ii, 224
Whooping-cough, ii, 286
Asthma, ii, 293
Orchitis after mumps, ii, 303
Diabetes from tumour of fourth ventricle, ii, 726
Exercise in diabetes, ii, 732
Gout, ii, 798 *et seq.*
Arthritis and cardiac disease, ii, 846
Zona, ii, 1027
TSCHIRJEW
"Tendon-reflexes," i, 440
TUCHMANN
Instrumental closure of ureter in diagnosis of renal disease, ii, 402
TUCKWELL, of Oxford
Chorea, i, 743 *et seq.*
Nerves in Addison's disease, ii, 740
TUFNELL, of Dublin (The Successful Treatment of Internal Aneurysm)
Aneurysm, i, 993
TUKE, HARRINGTON
Causation of cerebral abscess, i, 638
TULP, NICHOLAS (Obs. Med., 1652)
"Bronchial polypi," ii, 125
TÜRCK (Practische Anleitung zur Laryngoscopie, 1860)
Primary lateral sclerosis, i, 513
Lateral sclerosis secondary to cerebral lesions, i, 575
The laryngoscope, ii, 1
Atrophy of the cryco-thyroid, ii, 5
Laryngoscopic view of trachea, ii, 65
Retinitis in Bright's disease, ii, 609
TURNER, F. C.
Actinomycosis, i, 383
Fibroid disease of heart, i, 933
TURNER, of Samoa
Elephantiasis, ii, 992
TWEEDIE (Clinical Illust. of Fever, 1830)
Typhus, i, 144
TWINING (Clinical Illustrations of the Diseases of Bengal, 1835)
Dengue, i, 364

U.

UNDERWOOD (Treatise on the Diseases of Children, 1784–1846)
Acute atrophic paralysis, i, 497
UNNA, of Hamburg
Lichen planus acuminatus, ii, 901
Treatment of lichen planus, ii, 903
URTEL
Rickets in utero, ii, 857

V

VALENTINER and FÜRBRINGER
Sulphate of lime calculus, ii, 579
VALLEIX (Traité des Nevralgies, 1841)
Peripheral facial paralysis, i, 403
Neuralgia, i, 418
VALSALVA (Opera, 1740)
Bleeding in aneurysm, i, 993
VEIEL, of Cannstadt
Lupus erythematosus, ii, 1006, *footnote*
VELPEAU (Traité des Maladies du Sein, 1854, transl. by Mitchell Henry, New Syd. Soc.)
Scirrhous cancer of skin, ii, 1019
VERITÉ
Acne kéloidique, ii, 956
VERNEUIL
Histology of rodent ulcer, ii, 1020
VEYSSIÈRE (Recherches sur l'Hémianesthesie, 1874)
Anæsthesia with hemiplegia, i, 601
VIDAL
Pityriasis rosea, ii, 973
Area a tropho-neurosis, ii, 976
Scarification in lupus, ii, 1005
Xerodermia, ii, 1019
VIDDIUS, VIDUS, of the Collége de France (fl. 1550)
Varicella, i, 243
VILLEMIN (Études sur la Tuberculose, 1868)
Tubercle, i, 87
VIRCHOW (Handbuch der spec. Pathologie und Therapie, Bd. i, 1854; Die Cellularpathologie, 1858; Gesammelte Abhandlungen, 1862; Die krankhaften Geschwülste, 1863–67)
Causes of disease, i, 7
Pyrexia, i, 47
Inflammation, i, 52 *et seq.*
Phlegmonous phlebitis, i, 70
Tubercle, i, 80 *et seq.*

VIRCHOW (*continued*)—
Tumours, i, 97 *et seq.*
Origin of typhus, i, 144
Diphtheria, i, 260
Glanders, i, 376 *et seq.*
Reflex action in paraplegia, i, 439
Myelitis after fever, i, 449
Gumma of spinal meninges, i, 482
Cerebral tumour, i, 608 *et seq.*
Hypertrophy of brain, i, 647
Hæmatoma of dura mater, i, 709
Premature synostosis and idiocy, i, 876
Cretinism, i, 876 *et seq.*
Tubercle of the larynx, ii, 38
Goitre, ii, 60
Compression of left bronchus by dilated left auricle, ii, 63
Fœtid bronchitis, ii, 118
Miners' phthisis, ii, 241
Gastric ulcer, ii, 347
Primary sarcoma of stomach, ii, 356
Diphtheritic form of dysentery, ii, 390
Trichina spiralis, ii, 455, 456
Bright's disease, ii, 592
Lardaceous reaction ii, 630
Congenital cystic disease of kidneys, ii, 651
Pathology of Addison's disease, ii, 739
Leuchæmia, ii, 753 *et seq.*
The heart and vessels in chlorosis, ii, 770
Histology of mollities ossium, ii, 864
Molluscum contagiosum, ii, 951
Histology of lupus, ii, 995
Leprosy, ii, 1010
Fibroma molluscum, ii, 1013
Myoma of skin, ii, 1014
Elephantiasis teleangiectodes, ii, 1017
Epithelioma of skin, ii, 1019, *footnote*

VOGEL, ALFRED (Lehrbuch der Kinderkrankheiten, 1863)
Rötheln, i, 226
Hydrocephalus of childhood, i, 650
Tracheal and bronchial stenosis from scrofulous glands, ii, 62

VOGEL, AUGUST, of Munich (Ziemssen's Handbuch)
Single ulcer of the palate, ii, 299

VOGEL, JULIUS, of Giessen (Virchow's Hndb.)
Quantitative estimation of albumen in urine, ii, 597

VOGEL, of Dorpat
Case of pneumothorax, ii, 190, 192

VOIT and HOFFMAN
Acid phosphates in urine, ii, 570

VOIT and OERTEL
Uræmia, ii, 620 *et seq.*

VOLKMANN, of Halle (Clinical Lectures, New Syd. Soc.; Pitha's Handbuch, &c.)
Erysipelas, i, 249 *et seq.*
Acute atrophic paralysis, i, 498 *et seq.*
Causation of paralytic deformity, i, 499
Gangrene of lungs from otitis, ii, 160
Treatment of lupus, ii, 1005

VOLTOLINI, RUDOLPH, of Vienna
Deafness after meningitis, i, 704
Nasal polypi and asthma, ii, 296

VULPIAN (Mal. du Système nerveux, 1877)
Hemiplegia, i, 570

W.

WACHSMUTH
Bulbar paralysis, i, 517

WADE, of Birmingham
Diphtheria, i, 273

WADHAM
Aphasia, i, 580

WAGNER, ERNST, of Leipzig (Handbuch d. allgemeinen Pathologie; transl. by Drs van Duyn and Seguin, of New York)
Causes of disease, i, 7
Collapse, i, 18
Smallpox, i, 233
Histology of diphtheria, i, 261
Cholera-typhoid, i, 285
Bacillus anthracis, i, 374
Bright's disease, ii, 593 *et seq.*
Albuminuria, ii, 598
Tube casts, ii, 604
Uræmia, ii, 616 *et seq.*
Renal eclampsia, ii, 617
Cirrhosis of kidney, ii, 642 *et seq.*

WAGSTAFF
Elephantiasis, with lymphatic fistula, ii, 992

WALDENBURG
Emphysema of the lungs, ii, 109
Compressed air in bronchitis, ii, 123
Plastic bronchitis, ii, 125 *et seq.*

WALDEYER, of Strassburg (Volkmann's Sammlung)
Tumours, i, 99 *et seq.*
Origin of gastric carcinoma, ii, 357

WALDEYER and CARMALT
Amœboid movements in cells of carcinoma and sarcoma, i, 99

WALKER, ELIZA
Epilepsy from cerebral embolism, i, 599

WALLER, AUGUSTUS, of Birmingham, and afterwards of Geneva
Inflammation, i, 53
Degeneration of nerve trunks, i, 395

WALLER, AUGUSTUS, jun.
"Tendon-reflexes," i, 440

WALSHE (Diseases of the Lungs and of the Heart, 1851–73)
Hemiplegia, i, 569
Irregular pulse, i, 898
Mediastinal tumours, i, 1004
Phrenic paralysis after diphtheria, ii, 70
Emphysema of the lungs, ii, 111
Plastic bronchitis, ii, 125 *et seq.*
Pneumonia, ii, 136 *et seq.*
Gangrene of the lungs, ii, 159 *et seq.*
Pleurisy, ii, 164 *et seq.*
Pneumothorax, ii, 190 *et seq.*
Phthisis, ii, 211 *et seq.*
Asthma, ii, 294 *et seq.*

WALTHER
Moveable kidney, ii, 706

WALTON, HAYNES
Organic matrix of calculus, ii, 658

WARD, STEPHEN
Prognosis of dysentery, ii, 400
Treatment of dysentery, ii, 403
WARE, of Boston, U.S.A.
Delirium tremens, i, 886 *et seq.*
WARREN, the younger, of Boston, U.S.A.
Histology of cheloid, ii, 1015
WATSON, P.H., of Edinburgh
Extirpation of the larynx, ii, 57
WATSON, Sir THOMAS (Lectures on the Principles and Practice of Physic, 1871)
Syncope, i, 14
Specific disease, i, 27
Enteric fever, i, 192
Scarlatina, i, 212 *et seq.*
Measles, i, 218 *et seq.*
Smallpox, i, 228 *et seq.*
Erysipelas, i, 249 *et seq.*
Malaria and ague, i, 341 *et seq.*
Peripheral facial paralysis, i, 401
Tic douloureux, i, 421
Sciatica, i, 424
Cerebral hæmorrhage, i, 561 *et seq.*
Apoplexy, i, 603
Hydrocephalus of adults, i, 659
Meningitis, i, 675
Spurious hydrocephalus, i, 687
Spasmodic tic, i, 715
Chorea, i, 741
Tetanus, i, 759 *et seq.*
Hydrophobia, i, 768 *et seq.*
Migraine, i, 791
Epileptic aura, i, 794
Hysteria, i, 822 *et seq.*
Delirium tremens, i, 887 *et seq.*
Croup, ii, 20 *et seq.*
Case of Brunel, ii, 68
Rhonchus, ii, 87
Plastic bronchitis, ii, 125
Hæmothorax, ii, 178
Influenza, ii, 274 *et seq.*
Congenital whooping-cough, ii, 289
Gastralgia, ii, 332 *et seq.*
Pyrosis, ii, 336
Hæmatemesis, ii, 341 *et seq.*
Cancer of stomach, ii, 360, 364
Jaundice from emotion, ii, 497
Diabetes insipidus, ii, 562
Suppression of urine, ii, 671
Opium in diabetes, ii, 732
Gout, ii, 806 *et seq.*
Herpes of scalp, ii, 1027
Neuralgia from zona, ii, 1027
WEBBER
Chronic atrophic paralysis, i, 504
WEBER, E. H., of Leipzig
Detection of anæsthesia, i, 397
WEBER, HERMAN (Transl. of Braun's Baths and Waters ; Croonian Lect., 1885.)
Case of crossed hemiplegia, i, 571
Fatty heart, i, 940
Phthisis ab hæmoptoë, ii, 216
Contagiousness of phthisis, ii, 230
Health-resorts in phthisis, ii, 257
Sclerostomum duodemale, ii, 452
Ætiology of diabetes, ii, 726
WEBER, OTTO (Pitha's Handbuch)
Pyrexia, i, 44
Cancer, i, 120
WEIGERT (Volkmann's klin. Vorträge)
Tubercle, i, 92
Cardiac infarctus, i, 936
Cause of casts in urine, ii, 605
Anatomy of cirrhosis of kidney, ii, 642
WEIL
Lymphosarcoma of mediastinum, ii, 62
WELLS, SOELBERG
Tubercle of choroid, ii, 267
WELLS, Sir SPENCER (Abdominal Tumours)
Electrolytic treatment of bedsore, i, 470
Recovery from tubercular peritonitis, ii, 477
Ascites and ovarian dropsy, ii, 483
Nephrolithotomy, ii, 688
Treatment of hydronephrosis, ii, 680
Inflation of colon in diagnosis of renal tumour, ii, 696
WELLS, W. C. (1792)
Contagious erysipelas, i, 249
Albuminuria, ii, 591
WENZEL
Ague, i, 349
WERLHOF (Opera Medica, 1775)
Purpura (morbus maculosus), ii, 788
WERNHER, of Giessen
Cerebral localisation, i, 625
WERTHEIM
Membranous tracheitis, i, 62
WEST, CHARLES (Diseases of Infancy and Childhood, 1848–74)
Hydrocephalus of childhood, i, 653
Meningitis, i, 684 *et seq.*
Chorea, i, 756
Rarity of tetany in London, i, 738
Croup, ii, 24 *et seq.*
WEST, SAMUEL
Paracentesis pericardii, i, 948
Pneumothorax, ii, 191, *footnote*
WESTPHAL (Archiv für Psychiatrie, &c.)
"Tendon-reflexes," i, 440
Myelitis, i, 449
Acute ascending paralysis, i, 462 *et seq.*
Syringomyelus, i, 515
Locomotor ataxy, i, 534 *et seq.*
Disseminated sclerosis, i, 551
General paralysis, i, 668, *et seq.*
WHIPHAM
Paralysis of laryngeal abductors, ii, 9
Cardiac hypertrophy, i, 939
WHITTLE, of Belfast
Chyluria, ii, 701, *footnote*
WHITE, WM. HALE
Statistics of cerebral tumours, i, 610
Thomsen's disease, i, 734 *et seq.*
Histology of hydrophobia, i, 773
Results of exophthalmic goitre, i, 1013
Glycosuria in the insane, ii, 727, *note*
WHYTT, of Edinburgh (Observations on the Dropsy in the Brain, 1768)
Meningitis, i, 675 *et seq.*
Nervous cough, ii, 74

WICKE
Duration of chorea, i, 744
WIDERHOFER
Stenosis of trachea and bronchi, ii, 62
WILKS (Lectures on Pathological Anatomy, 1859; Lectures on Diseases of the Nervous System, 1878; Guy's Hosp. Rep.)
Tubercle of serous membranes, i, 94
Tumours, i, 102 *et seq.*
Marks on nails after fevers, i, 212
Roseola of smallpox, i, 228
Roseola choleraica, i, 284 *et seq.*
Visceral syphilis, i, 324
Alcoholic paraplegia, i, 463
Recovery from acute myelitis, i, 468
Spinal caries, i, 479
Gumma of spinal meninges, i, 482
Pathology of club-foot, i, 499
Simulation of bulbar paralysis, i, 518
Locomotor ataxy, i, 531 *et seq.*
Syphilitic disease of cerebral arteries, i, 556
Cerebral gumma, i, 607
Chronic diffused cerebritis, i, 646
External hydrocephalus, i, 652
General paralysis of the insane i, 670
Atrophy of the brain, i, 671
Hæmatoma of dura mater, i, 710
Treatment of torticollis, i, 719
Chorea, i, 742 *et seq.*
Coloured sweat in tetanus, i, 759
Migraine, i, 782 *et seq.*
Epilepsy, i, 793 *et seq.*
Paroxysmal vertigo, i, 814 *et seq.*
Hysteria, i, 826 *et seq.*
Cretinoid condition of adults, i, 881
Delirium tremens, i, 888 *et seq.*
Morbid anatomy of heat-stroke, i, 893
Frequent pulse, i, 898
Valvular diseases of heart, i, 953—978
Embolism, i, 1008
Syphilitic stenosis of trachea, ii, 63
Event of pneumonia, ii, 132, 905
Broncho-pneumonia after burns, ii, 148
Thorax of phthisis, ii, 234
Sarcoma of stomach, ii, 356
Tumour of pylorus, ii, 356
Origin of typhilitis, ii, 385, 386
Pyæmia and jaundice, ii, 498
Lardaceous liver, ii, 545
Bright's disease, ii, 592 *et seq.*
Lardaceous disease of kidneys, ii, 629
Cystic disease of kidneys, ii, 651
Diabetic phthisis, ii, 718
Addison's disease, ii, 733, *et seq.*
Hodgkin's disease, ii, 761 *et seq.*
Pityriasis rubra, ii, 908
Red sweat, ii, 960
Trichoclasia, ii, 977
Linear atrophy of skin, ii, 991
WILLAN, ROBERT (Cutaneous Diseases, 1808)
Anatomical classification, ii, 866
Eczema a vesicular disease, ii, 872
Pityriasis nigra, ii, 907
Porrigo decalvans, ii, 975
Leprosy, ii, 1009
Purpura urticans, ii, 1024
WILLIAMS, C. J. B. (Principles of Medicine, 1856; Pathology and Diagnosis of Disease of the Chest, 1840)
Nature of tubercle, i, 95
Apyrexia in phthisis, ii, 211
Duration of phthisis, ii, 226
Albuminuria in phthisis, ii, 228
Origin of phthisis in catarrh, ii, 246
Age in phthisis, ii, 254
Sex in phthisis, ii, 255
WILLIAMS, CHAS. THEODORE (The Climate of the South of France, 1867; Pulmonary Consumption, 1887)
Bacilli in grinders' phthisis ii, 241, *note*
The influence of climate on pulmonary consumption, ii, 258
Case of typhlitis, ii, 385
WILLIAMS, ROBERT (Elements of Medicine: on Morbid Poisons, 1836)
Influenza, ii, 275
WILLIS, THOMAS (Opera Omnia, 1682)
General paralysis of the insane, i, 663
Discovery of glycosuria, ii, 713
Hypochondriasis, i, 843
WILSON
Sea-voyages in phthisis, ii, 257
WILSON, ERASMUS (Diseases of the Skin, 1847-67)
Lichen planus, ii, 901
Exfoliative dermatitis, ii, 907
Xerodermia, ii, 949
Molluscum corpuscles, ii, 951
Trichoclasia, ii, 977
Pathology of ichthyosis, ii, 987
WINCKEL
Epidemic hæmoglobinuria among infants, ii, 585
WINDLE, of Dublin
Statistics of autopsies in diabetes, ii, 721
VON WINIWATER, of Vienna
Inflammation, i, 56
WINTERNITZ
Pellagra, ii, 1012
WINTRICH (Krankheiten der Respirationsorgane, 1854)
Physical examination of thorax, ii, 78
Diagnosis of pleuritic effusion, ii, 169
Abscess from gastric ulcer simulating pneumothorax, ii, 197
WITHERING, of Birmingham (Account of the Scarlet Fever and Sore-throat, 1779)
Scarlatina i, 209
WOOLASTON, WILLIAM (ob. 1828)
Migrane, i, 781
Cystine calculus, ii, 578
WOOD, KINDER
Hysterical ecstasy, i, 832
WOODMAN, BATHURST (Notes in transl. of Wunderlich on Temperature)
Scarlatina, i, 213
Erysipelas, i, 253
Temperature in chorea, i, 742
WOROSCHILOFF
Motor tracts of spinal cord, i, 435
WORTHINGTON
Œsophageal regurgitation, ii, 313

WRIGHT
Tetany and spasmodic ergotism, i, 739
WUCHERER, of Bahia
Filaria in chylous urine, ii, 459, 700
WUNDERLICH, of Leipzig (Das Verhalten der Eigenwärme in Krankheiten, 1868, transl. by Dr Woodman, New Syd. Soc.; Volkmann's Sammlung)
Clinical thermometry, i, 37, 39
Pyrexia, i, 37 *et seq.*
Temperature in typhus, i, 133
— enteric fever, i, 171
— smallpox, i, 236
— ague, i, 341
Hyperpyrexia in hysteria, i, 835
Temperature in pneumonia, ii, 137
Recovery from acute miliary tuberculosis, ii, 270
Temperature in rheumatism, ii, 812
WURZEL
Chorea during pregnancy, i, 752
VON WYSS, of Zürich
Cholera i, 284
Glanders, i, 377 *et seq.*

Y.

YANDELL, of Kentucky, U.S.A.
Malaria and cutaneous diseases, ii, 913
YEATS
Diphtheria, i, 266; ii, 20
YOUATT (The Horse, 1831; The Dog, 1851)
Glanders, i, 380
Hydrophobia, i, 767 *et seq.*

Z.

ZAHN
Granular degeneration of diaphragm in chronic bronchitis, ii, 105
ZANCAROL
Bilharzia hæmatobia, ii, 702
ZENKER, of Erlangen (Veranderüngen d. Muskeln im Typhus abdominalis, 1864)
The muscles in enteric fever, i, 176
Pneumonokoniosis, ii, 259
Trichina spiralis, ii, 456
ZIEGLER (Exp. Unters. ü. die Herkunft der Tuberkelelemente, 1875; Volkmann's Sammlung; Pathologische Anatomie, translated by Dr MacAlister)
New tissues in inflammation, i, 66
Tubercle, i, 66, 82
Lardaceous disease, ii, 630, 631
VON ZIEMSSEN, of Munich (Articles in his Cyclopædia)
Diseases of the larynx, ii, 5 *et seq.*
Emphysema of lungs, ii, 108
Ætiology of acute pneumonia, ii, 141
Broncho-pneumonia, ii, 148 *et seq.*
Wet-pack in Bright's disease, ii, 654
Epidemic meningitis, i, 690 *et seq.*
Chorea, i, 741 *et seq.*
ZIMMERMANN
Pyrexia, i, 46
ZOPF (Die Spaltpilze, 1885)
Classification of microphyta, i, 24
Pleomorphism, i, 25, *footnote*
ZÜLZER (Ziemssen's Handbuch)
Typhus, i, 146
Smallpox, ii, 227 *et seq.*
Erysipelas, i, 252 *et seq.*
ZURADELLI (Del Crampo degli Scrittore, 1857)
Writers' cramp, i, 725

PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE.

Catalogue B] *London*, 11, *New Burlington Street*
May, 1888

SELECTION

FROM

J. & A. CHURCHILL'S GENERAL CATALOGUE

COMPRISING

ALL RECENT WORKS PUBLISHED BY THEM

ON THE

ART AND SCIENCE OF MEDICINE

N.B.—As far as possible, this List is arranged in the order in which medical study is usually pursued.

J. & A. CHURCHILL publish for the following Institutions and Public Bodies:—

ROYAL COLLEGE OF SURGEONS.

CATALOGUES OF THE MUSEUM.

Twenty-three separate Catalogues (List and Prices can be obtained of J. & A. CHURCHILL).

GUY'S HOSPITAL.

REPORTS BY THE MEDICAL AND SURGICAL STAFF.
Vol. XXIX., Third Series. 7s. 6d.

FORMULÆ USED IN THE HOSPITAL IN ADDITION TO THOSE IN THE B.P. 1s. 6d.

LONDON HOSPITAL.

PHARMACOPŒIA OF THE HOSPITAL. 3s.

CLINICAL LECTURES AND REPORTS BY THE MEDICAL AND SURGICAL STAFF. Vols. I. to IV. 7s. 6d. each.

ST. BARTHOLOMEW'S HOSPITAL.

CATALOGUE OF THE ANATOMICAL AND PATHOLOGICAL MUSEUM. Vol. I.—Pathology. 15s. Vol. II.—Teratology, Anatomy and Physiology, Botany. 7s. 6d.

ST. GEORGE'S HOSPITAL.

REPORTS BY THE MEDICAL AND SURGICAL STAFF.
The last Volume (X.) was issued in 1880. Price 7s. 6d.

CATALOGUE OF THE PATHOLOGICAL MUSEUM. 15s.

SUPPLEMENTARY CATALOGUE (1882). 5s.

ST. THOMAS'S HOSPITAL.

REPORTS BY THE MEDICAL AND SURGICAL STAFF
Annually. Vol. XVI., New Series. 7s. 6d.

MIDDLESEX HOSPITAL.

CATALOGUE OF THE PATHOLOGICAL MUSEUM. 12s.

WESTMINSTER HOSPITAL.

REPORTS BY THE MEDICAL AND SURGICAL STAFF.
Annually. Vol. III. 6s.

ROYAL LONDON OPHTHALMIC HOSPITAL.

REPORTS BY THE MEDICAL AND SURGICAL STAFF.
Occasionally. Vol. XII., Part I. 5s.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

TRANSACTIONS.
Vol. VII. 12s. 6d.

MEDICO-PSYCHOLOGICAL ASSOCIATION.

JOURNAL OF MENTAL SCIENCE.
Quarterly. 3s. 6d. each, or 14s. per annum.

PHARMACEUTICAL SOCIETY OF GREAT BRITAIN.

PHARMACEUTICAL JOURNAL AND TRANSACTIONS.
Every Saturday. 4d. each, or 20s. per annum, post free.

BRITISH PHARMACEUTICAL CONFERENCE.

YEAR BOOK OF PHARMACY.
In December. 10s.

BRITISH DENTAL ASSOCIATION.

JOURNAL OF THE ASSOCIATION AND MONTHLY REVIEW OF DENTAL SURGERY.

On the 15th of each Month. 6d. each, or 7s. per annum, post free.

A SELECTION

FROM

J. & A. CHURCHILL'S GENERAL CATALOGUE,

COMPRISING

ALL RECENT WORKS PUBLISHED BY THEM ON THE ART AND SCIENCE OF MEDICINE.

N.B.—*J. & A. Churchill's Descriptive List of Works on Chemistry, Materia Medica, Pharmacy, Botany, Photography, Zoology, the Microscope, and other Branches of Science, can be had on application.*

Practical Anatomy: A Manual of Dissections. By CHRISTOPHER HEATH, Surgeon to University College Hospital. Seventh Edition. Revised by RICKMAN J. GODLEE, M.S. Lond., F.R.C.S., Teacher of Operative Surgery, late Demonstrator of Anatomy in University College, and Surgeon to the Hospital. Crown 8vo, with 24 Coloured Plates and 278 Engravings, 15s.

Wilson's Anatomist's Vade-Mecum. Tenth Edition. By GEORGE BUCHANAN, Professor of Clinical Surgery in the University of Glasgow; and HENRY E. CLARK, M.R.C.S., Lecturer on Anatomy at the Glasgow Royal Infirmary School of Medicine. Crown 8vo, with 450 Engravings (including 26 Coloured Plates), 18s.

Braune's Atlas of Topographical Anatomy, after Plane Sections of Frozen Bodies. Translated by EDWARD BELLAMY, Surgeon to, and Lecturer on Anatomy, &c., at, Charing Cross Hospital. Large Imp. 8vo, with 34 Photolithographic Plates and 46 Woodcuts, 40s.

An Atlas of Human Anatomy. By RICKMAN J. GODLEE, M.S., F.R.C.S., Assistant Surgeon and Senior Demonstrator of Anatomy, University College Hospital. With 48 Imp. 4to Plates (112 figures), and a volume of Explanatory Text. 8vo, £4 14s. 6d.

Harvey's (Wm.) Manuscript Lectures. Prelectiones Anatomiæ Universalis. Edited, with an Autotype reproduction of the Original, by a Committee of the Royal College of Physicians of London. Crown 4to, half bound in Persian, 52s. 6d.

Anatomy of the Joints of Man. By HENRY MORRIS, Surgeon to, and Lecturer on Anatomy and Practical Surgery at, the Middlesex Hospital. 8vo, with 44 Lithographic Plates (several being coloured) and 13 Wood Engravings, 16s.

Manual of the Dissection of the Human Body. By LUTHER HOLDEN, Consulting Surgeon to St. Bartholomew's Hospital. Edited by JOHN LANGTON, F.R.C.S., Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital. Fifth Edition. 8vo, with 208 Engravings. 20s.

By the same Author.

Human Osteology. Seventh Edition, edited by CHARLES STEWART, Conservator of the Museum R.C.S., and R. W. REID, M.D., F.R.C.S., Lecturer on Anatomy at St. Thomas's Hospital. 8vo, with 59 Lithographic Plates and 75 Engravings. 16s.

Also.

Landmarks, Medical and Surgical. Fourth Edition. 8vo. [*In the Press.*

The Student's Guide to Surgical Anatomy. By EDWARD BELLAMY, F.R.C.S. and Member of the Board of Examiners. Third Edition. Fcap. 8vo, with 81 Engravings. 7s. 6d.

The Anatomical Remembrancer; or, Complete Pocket Anatomist. Eighth Edition. 32mo, 3s. 6d.

Diagrams of the Nerves of the Human Body, exhibiting their Origin, Divisions, and Connections, with their Distribution to the Various Regions of the Cutaneous Surface, and to all the Muscles. By W. H. FLOWER, C.B., F.R.S., F.R.C.S. Third Edition, with 6 Plates. Royal 4to, 12s.

General Pathology.
An Introduction to. By JOHN BLAND SUTTON, F.R.C.S., Sir E. Wilson Lecturer on Pathology, R.C.S.; Assistant Surgeon to, and Lecturer on Anatomy at, Middlesex Hospital. 8vo, with 149 Engravings, 14s.

Atlas of Pathological Anatomy.
By Dr. LANCEREAUX. Translated by W. S. GREENFIELD, M.D., Professor of Pathology in the University of Edinburgh. Imp. 8vo, with 70 Coloured Plates, £5 5s.

A Manual of Pathological Anatomy. By C. HANDFIELD JONES, M.B., F.R.S., and E. H. SIEVEKING, M.D., F.R.C.P. Edited by J. F. PAYNE, M.D., F.R.C.P., Lecturer on General Pathology at St. Thomas's Hospital. Second Edition. Crown 8vo, with 195 Engravings, 16s.

Post-mortem Examinations:
A Description and Explanation of the Method of Performing them, with especial reference to Medico-Legal Practice. By Prof. VIRCHOW. Translated by Dr. T. P. SMITH. Second Edition. Fcap. 8vo, with 4 Plates, 3s. 6d.

The Human Brain:
Histological and Coarse Methods of Research. A Manual for Students and Asylum Medical Officers. By W. BEVAN LEWIS, L.R.C.P. Lond., Medical Superintendent, West Riding Lunatic Asylum. 8vo, with Wood Engravings and Photographs, 8s.

Manual of Physiology:
For the use of Junior Students of Medicine. By GERALD F. YEO, M.D., F.R.C.S., Professor of Physiology in King's College, London. Second Edition. Crown 8vo, with 318 Engravings, 14s.

Principles of Human Physiology. By W. B. CARPENTER, C.B., M.D., F.R.S. Ninth Edition. By HENRY POWER, M.B., F.R.C.S. 8vo, with 3 Steel Plates and 377 Wood Engravings, 31s. 6d.

Elementary Practical Biology:
Vegetable. By THOMAS W. SHORE, M.D., B.Sc. Lond., Lecturer on Comparative Anatomy at St. Bartholomew's Hospital. 8vo, 6s.

Histology and Histo-Chemistry of Man. By HEINRICH FREY, Professor of Medicine in Zurich. Translated by ARTHUR E. J. BARKER, Assistant Surgeon to University College Hospital. 8vo, with 608 Engravings, 21s.

A Text-Book of Medical Physics,
for Students and Practitioners. By J. C. DRAPER, M.D., LL.D., Professor of Physics in the University of New York. With 377 Engravings. 8vo, 18s.

Medical Jurisprudence:
Its Principles and Practice. By ALFRED S. TAYLOR, M.D., F.R.C.P., F.R.S. Third Edition, by THOMAS STEVENSON, M.D., F.R.C.P., Lecturer on Medical Jurisprudence at Guy's Hospital. 2 vols. 8vo, with 188 Engravings, 31s. 6d.

By the same Authors.

A Manual of Medical Jurisprudence. Eleventh Edition. Crown 8vo, with 56 Engravings, 14s.

Also.

Poisons,
In Relation to Medical Jurisprudence and Medicine. Third Edition. Crown 8vo, with 104 Engravings, 16s.

Lectures on Medical Jurisprudence. By FRANCIS OGSTON, M.D., late Professor in the University of Aberdeen. Edited by FRANCIS OGSTON, Jun., M.D. 8vo, with 12 Copper Plates, 18s.

The Student's Guide to Medical Jurisprudence. By JOHN ABERCROMBIE, M.D., F.R.C.P., Lecturer on Forensic Medicine to Charing Cross Hospital. Fcap. 8vo, 7s. 6d.

Influence of Sex in Disease.
By W. ROGER WILLIAMS, F.R.C.S., Surgical Registrar to the Middlesex Hospital. 8vo, 3s. 6d.

Microscopical Examination of Drinking Water and of Air. By J. D. MACDONALD, M.D., F.R.S., Ex-Professor of Naval Hygiene in the Army Medical School. Second Edition. 8vo, with 25 Plates, 7s. 6d.

Pay Hospitals and Paying Wards throughout the World. By HENRY C. BURDETT. 8vo, 7s.

By the same Author.

Cottage Hospitals — General, Fever, and Convalescent: Their Progress, Management, and Work. Second Edition, with many Plans and Illustrations. Crown 8vo, 14s.

Hospitals, Infirmaries, and Dispensaries: Their Construction, Interior Arrangement, and Management; with Descriptions of existing Institutions, and 74 Illustrations. By F. OPPERT, M.D., M.R.C.P.L. Second Edition. Royal 8vo, 12s.

Hospital Construction and Management. By F. J. MOUAT, M.D., Local Government Board Inspector, and H. SAXON SNELL, Fell. Roy. Inst. Brit. Architects. In 2 Parts, 4to, 15s. each; or, the whole work bound in half calf, with large Map, 54 Lithographic Plates, and 27 Woodcuts, 35s.

Public Health Reports.
By Sir JOHN SIMON, C.B., F.R.S. Edited by EDWARD SEATON, M.D., F.R.C.P. 2 vols. 8vo, with Portrait, 36s.

A Manual of Practical Hygiene. By E. A. PARKES, M.D., F.R.S. Seventh Edition, by F. DE CHAUMONT, M.D., F.R.S., Professor of Military Hygiene in the Army Medical School. 8vo, with 9 Plates and 101 Engravings, 18s.

A Handbook of Hygiene and Sanitary Science. By GEO. WILSON, M.A., M.D., F.R.S.E., Medical Officer of Health for Mid-Warwickshire. Sixth Edition. Crown 8vo, with Engravings. 10s. 6d.

By the same Author.

Healthy Life and Healthy Dwellings: A Guide to Personal and Domestic Hygiene. Fcap. 8vo, 5s.

Sanitary Examinations Of Water, Air, and Food. A Vade-Mecum for the Medical Officer of Health. By CORNELIUS B. FOX, M.D., F.R.C.P. Second Edition. Crown 8vo, with 110 Engravings, 12s. 6d.

Dangers to Health: A Pictorial Guide to Domestic Sanitary Defects. By T. PRIDGIN TEALE, M.A., Surgeon to the Leeds General Infirmary. Fourth Edition. 8vo, with 70 Lithograph Plates (mostly coloured), 10s.

Epidemic Influences: Epidemiological Aspects of Yellow Fever and of Cholera. The Milroy Lectures. By ROBERT LAWSON, LL.D., Inspector-General of Hospitals. 8vo, with Maps, Diagrams, &c., 6s.

Detection of Colour-Blindness and Imperfect Eyesight. By CHARLES ROBERTS, F.R.C.S. Second Edition. 8vo, with a Table of Coloured Wools, and Sheet of Test-types, 5s.

Illustrations of the Influence of the Mind upon the Body in Health and Disease: Designed to elucidate the Action of the Imagination. By D. H. TUKE, M.D., F.R.C.P., LL.D. Second Edition. 2 vols. crown 8vo, 15s.

By the same Author.

Sleep-Walking and Hypnotism. 8vo, 5s.

A Manual of Psychological Medicine. With an Appendix of Cases. By JOHN C. BUCKNILL, M.D., F.R.S., and D. HACK TUKE, M.D., F.R.C.P. Fourth Edition. 8vo, with 12 Plates (30 Figures) and Engravings, 25s.

Mental Affections of Childhood and Youth (Lettsomian Lectures for 1887, &c.). By J. LANGDON DOWN, M.D., F.R.C.P., Senior Physician to the London Hospital. 8vo, 6s.

Mental Diseases. Clinical Lectures. By T. S. CLOUSTON, M.D., F.R.C.P. Edin., Lecturer on Mental Diseases in the University of Edinburgh. Second Edition. Crown 8vo, with 8 Plates (6 Coloured), 12s. 6d.

Private Treatment of the Insane as Single Patients. By EDWARD EAST, M.R.C.S., L.S.A. Crown 8vo, 2s. 6d.

Manual of Midwifery. By ALFRED L. GALABIN, M.A., M.D., F.R.C.P., Obstetric Physician to, and Lecturer on Midwifery, &c. at, Guy's Hospital. Crown 8vo, with 227 Engravings, 15s.

The Student's Guide to the Practice of Midwifery. By D. LLOYD ROBERTS, M.D., F.R.C.P., Lecturer on Clinical Midwifery and Diseases of Women at the Owens College; Obstetric Physician to the Manchester Royal Infirmary. Third Edition. Fcap. 8vo, with 2 Coloured Plates and 127 Wood Engravings, 7s. 6d.

Lectures on Obstetric Operations: Including the Treatment of Hæmorrhage, and forming a Guide to the Management of Difficult Labour. By ROBERT BARNES, M.D., F.R.C.P., Consulting Obstetric Physician to St. George's Hospital. Fourth Edition. 8vo, with 121 Engravings, 12s. 6d.

By the same Author.

A Clinical History of Medical and Surgical Diseases of Women. Second Edition. 8vo, with 181 Engravings, 28s.

Clinical Lectures on Diseases of Women: Delivered in St. Bartholomew's Hospital, by J. MATTHEWS DUNCAN, M.D., LL.D., F.R.S. Third Edition. 8vo, 16s.

By the same Author.

Sterility in Woman. Being the Gulstonian Lectures, delivered in the Royal College of Physicians, in Feb., 1883. 8vo, 6s.

Notes on Diseases of Women: Specially designed to assist the Student in preparing for Examination. By J. J. REYNOLDS, L.R.C.P., M.R.C.S. Third Edition. Fcap. 8vo, 2s. 6d.

By the same Author.

Notes on Midwifery: Specially designed for Students preparing for Examination. Second Edition. Fcap. 8vo, with 15 Engravings, 4s.

A Manual of Obstetrics. By A. F. A. KING, A.M., M.D., Professor of Obstetrics, &c., in the Columbian University, Washington, and the University of Vermont. Third Edition. Crown 8vo, with 102 Engravings, 8s.

Intra-Uterine Death: (Pathology of). Being the Lumleian Lectures, 1887. By WILLIAM O. PRIESTLEY, M.D., F.R.C.P., LL.D., Consulting Physician to King's College Hospital. 8vo, with 3 Coloured Plates and 17 Engravings, 7s. 6d.

The Student's Guide to the Diseases of Women. By ALFRED L. GALABIN, M.D., F.R.C.P., Obstetric Physician to Guy's Hospital. Fourth Edition. Fcap. 8vo, with 94 Engravings, 7s. 6d.

West on the Diseases of Women. Fourth Edition, revised by the Author, with numerous Additions by J. MATTHEWS DUNCAN, M.D., F.R.C.P., F.R.S.E., Obstetric Physician to St. Bartholomew's Hospital. 8vo, 16s.

Obstetric Aphorisms: For the Use of Students commencing Midwifery Practice. By JOSEPH G. SWAYNE, M.D. Eighth Edition. Fcap. 8vo, with Engravings, 3s. 6d.

Handbook of Midwifery for Midwives: By J. E. BURTON, L.R.C.P. Lond., Surgeon to the Hospital for Women, Liverpool. Second Edition. With Engravings. Fcap. 8vo, 6s.

A Handbook of Uterine Therapeutics, and of Diseases of Women. By E. J. TILT, M.D., M.R.C.P. Fourth Edition. Post 8vo, 10s.

By the same Author.

The Change of Life In Health and Disease: A Clinical Treatise on the Diseases of the Nervous System incidental to Women at the Decline of Life. Fourth Edition. 8vo, 10s. 6d.

Diseases of the Uterus, Ovaries, and Fallopian Tubes: A Practical Treatise by A. COURTY, Professor of Clinical Surgery, Montpellier. Translated from Third Edition by his Pupil, AGNES MCLAREN, M.D., M.K.Q.C.P.I., with Preface by J. MATTHEWS DUNCAN, M.D., F.R.C.P. 8vo, with 424 Engravings, 24s.

The Female Pelvic Organs: Their Surgery, Surgical Pathology, and Surgical Anatomy. In a Series of Coloured Plates taken from Nature; with Commentaries, Notes, and Cases. By HENRY SAVAGE, M.D., F.R.C.S., Consulting Officer of the Samaritan Free Hospital. Fifth Edition. Roy. 4to, with 17 Lithographic Plates (15 coloured) and 52 Woodcuts, £1 15s.

A Practical Treatise on the Diseases of Women. By T. GAILLARD THOMAS, M.D., Professor of Diseases of Women in the College of Physicians and Surgeons, New York. Fifth Edition. Roy. 8vo, with 266 Engravings, 25s.

Backward Displacements of the Uterus and Prolapsus Uteri: Treatment by the New Method of Shortening the Round Ligaments. By WILLIAM ALEXANDER, M.D., M.Ch.Q.U.I., F.R.C.S., Surgeon to the Liverpool Infirmary. Crown 8vo, with Engravings, 3s. 6d.

Diseases and Accidents Incident to Women, and the Practice of Medicine and Surgery applied to them. By W. H. BYFORD, A.M., M.D., Professor of Gynæcology in Rush Medical College, and HENRY T. BYFORD, M.D., Surgeon to the Woman's Hospital, Chicago. Fourth Edition. 8vo, with 306 Engravings, 25s.

Gynæcological Operations: (Handbook of). By ALBAN H. G. DORAN, F.R.C.S., Surgeon to the Samaritan Hospital. 8vo, with 167 Engravings, 15s.

Abdominal Surgery. By J. GREIG SMITH, M.A., F.R.S.E., Surgeon to the Bristol Royal Infirmary. Second Edition. 8vo, with 79 Engravings, 21s.

Ovarian and Uterine Tumours: Their Pathology and Surgical Treatment. By Sir T. SPENCER WELLS, Bart., F.R.C.S., Consulting Surgeon to the Samaritan Hospital. 8vo, with Engravings, 21s.

The Student's Guide to Diseases of Children. By JAS. F. GOODHART, M.D., F.R.C.P., Physician to Guy's Hospital, and to the Evelina Hospital for Sick Children. Second Edition. Fcap. 8vo, 10s. 6d.

Diseases of Children. For Practitioners and Students. By W. H. DAY, M.D., Physician to the Samaritan Hospital. Second Edition. Crown 8vo, 12s. 6d.

A Practical Treatise on Disease in Children. By EUSTACE SMITH, M.D., Physician to the King of the Belgians, Physician to the East London Hospital for Children. 8vo, 22s.

By the same Author.

Clinical Studies of Disease in Children. Second Edition. Post 8vo, 7s. 6d. *Also.*

The Wasting Diseases of Infants and Children. Fifth Edition. Post 8vo, 8s. 6d.

A Practical Manual of the Diseases of Children. With a Formulary. By EDWARD ELLIS, M.D. Fifth Edition. Crown 8vo, 10s.

A Manual for Hospital Nurses and others engaged in Attending on the Sick, and a Glossary. By EDWARD J. DOMVILLE, Surgeon to the Exeter Lying-in Charity. Sixth Edition. Cr. 8vo, 2s. 6d.

A Manual of Nursing, Medical and Surgical. By CHARLES J. CULLINGWORTH, M.D., Obstetric Physician to St. Thomas's Hospital. Second Edition. Fcap. 8vo, with Engravings, 3s. 6d.

By the same Author.

A Short Manual for Monthly Nurses. Second Edition. Fcap. 8vo, 1s. 6d.

Diseases and their Commencement. Lectures to Trained Nurses. By DONALD W. C. HOOD, M.D., M.R.C.P., Physician to the West London Hospital. Crown 8vo, 2s. 6d.

Outlines of Infectious Diseases: For the use of Clinical Students. By J. W. ALLAN, M.B., Physician Superintendent Glasgow Fever Hospital. Fcap. 8vo., 3s.

Hospital Sisters and their Duties. By EVA C. E. LÜCKES, Matron to the London Hospital. Crown 8vo, 2s. 6d.

Infant Feeding and its Influence on Life; By C. H. F. ROUTH, M.D., Physician to the Samaritan Hospital. Fourth Edition. Fcap. 8vo. [*Preparing.*

Manual of Botany: Including the Structure, Classification, Properties, Uses, and Functions of Plants. By ROBERT BENTLEY, Professor of Botany in King's College and to the Pharmaceutical Society. Fifth Edition. Crown 8vo, with 1,178 Engravings, 15s.

By the same Author.

The Student's Guide to Structural, Morphological, and Physiological Botany. With 660 Engravings. Fcap. 8vo, 7s. 6d.

Also.

The Student's Guide to Systematic Botany, including the Classification of Plants and Descriptive Botany. Fcap. 8vo, with 350 Engravings, 3s. 6d.

Medicinal Plants: Being descriptions, with original figures, of the Principal Plants employed in Medicine, and an account of their Properties and Uses. By Prof. BENTLEY and Dr. H. TRIMEN. In 4 vols., large 8vo, with 306 Coloured Plates, bound in Half Morocco, Gilt Edges, £11 11s.

The National Dispensatory: Containing the Natural History, Chemistry, Pharmacy, Actions and Uses of Medicines. By ALFRED STILLÉ, M.D., LL.D., and JOHN M. MAISCH, Ph.D. Fourth Edition. 8vo, with 311 Engravings, 36s.

Royle's Manual of Materia Medica and Therapeutics. Sixth Edition, including additions and alterations in the B.P. 1885. By JOHN HARLEY, M.D., Physician to St. Thomas's Hospital. Crown 8vo, with 139 Engravings, 15s.

Materia Medica and Therapeutics: Vegetable Kingdom — Organic Compounds — Animal Kingdom. By CHARLES D. F. PHILLIPS, M.D., F.R.S. Edin., late Lecturer on Materia Medica and Therapeutics at the Westminster Hospital Medical School. 8vo, 25s.

The Student's Guide to Materia Medica and Therapeutics. By JOHN C. THOROWGOOD, M.D., F.R.C.P. Second Edition. Fcap. 8vo, 7s.

Materia Medica. A Manual for the use of Students. By ISAMBARD OWEN, M.D., F.R.C.P., Lecturer on Materia Medica, &c., to St. George's Hospital. Second Edition. Crown 8vo, 6s. 6d.

The Pharmacopœia of the London Hospital. Compiled under the direction of a Committee appointed by the Hospital Medical Council. Fcap. 8vo, 3s.

A Companion to the British Pharmacopœia. By PETER SQUIRE, Revised by his Sons, P. W. and A. H. SQUIRE. 14th Edition. 8vo, 10s. 6d.

By the same Authors.

The Pharmacopœias of the London Hospitals, arranged in Groups for Easy Reference and Comparison. Fifth Edition. 18mo, 6s.

The Prescriber's Pharmacopœia: The Medicines arranged in Classes according to their Action, with their Composition and Doses. By NESTOR J. C. TIRARD, M.D., F.R.C.P., Professor of Materia Medica and Therapeutics in King's College, London. Sixth Edition. 32mo, bound in leather, 3s.

A Treatise on the Principles and Practice of Medicine. Sixth Edition. By AUSTIN FLINT, M.D., W. H. WELCH, M.D., and AUSTIN FLINT, jun., M.D. 8vo, with Engravings, 26s.

Climate and Fevers of India, with a series of Cases (Croonian Lectures, 1882). By Sir JOSEPH FAYRER, K.C.S.I., M.D. 8vo, with 17 Temperature Charts, 12s.

Family Medicine for India. A Manual. By WILLIAM J. MOORE, M.D., C.I.E., Honorary Surgeon to the Viceroy of India. Published under the Authority of the Government of India. Fifth Edition. Post 8vo, with Engravings. [*In the Press.*

By the same Author.

A Manual of the Diseases of India: With a Compendium of Diseases generally. Second Edition. Post 8vo, 10s.

Practical Therapeutics: A Manual. By EDWARD J. WARING, C.I.E., M.D., F.R.C.P., and DUDLEY W. BUXTON, M.D., B.S. Lond. Fourth Edition. Crown 8vo, 14s.

By the same Author.

Bazaar Medicines of India, And Common Medical Plants: With Full Index of Diseases, indicating their Treatment by these and other Agents procurable throughout India, &c. Fourth Edition. Fcap. 8vo, 5s.

A Commentary on the Diseases of India. By NORMAN CHEVERS, C.I.E., M.D., F.R.C.S., Deputy Surgeon-General H.M. Indian Army. 8vo, 24s.

The Principles and Practice of Medicine. By C. HILTON FAGGE, M.D. Second Edition. Edited by P. H. PYE-SMITH, M.D., F.R.C.P., Physician to, and Lecturer on Medicine at, Guy's Hospital. 2 vols. 8vo. Cloth, 36s.; Half Leather, 42s. [*Nearly Ready.*

The Student's Guide to the Practice of Medicine. By M. CHARTERIS, M.D., Professor of Materia Medica in the University of Glasgow. Fourth Edition. Fcap. 8vo, with Engravings on Copper and Wood, 9s.

Hooper's Physicians' Vade-Mecum. A Manual of the Principles and Practice of Physic. Tenth Edition. By W. A. GUY, F.R.C.P., F.R.S., and J. HARLEY, M.D., F.R.C.P. With 118 Engravings. Fcap. 8vo, 12s. 6d.

Preventive Medicine.
Collected Essays. By WILLIAM SQUIRE, M.D., F.R.C.P., Physician to St. George, Hanover-square, Dispensary. 8vo, 6s. 6d.

The Student's Guide to Clinical Medicine and Case-Taking. By FRANCIS WARNER, M.D., F.R.C.P., Physician to the London Hospital. Second Edition. Fcap. 8vo, 5s.

How to Examine the Chest:
A Practical Guide for the use of Students. By SAMUEL WEST, M.D., F.R.C.P., Physician to the City of London Hospital for Diseases of the Chest; Assistant Physician to St. Bartholomew's Hospital. With 42 Engravings. Fcap. 8vo, 5s.

Diseases of the Chest:
Contributions to their Clinical History, Pathology, and Treatment. By A. T. HOUGHTON WATERS, M.D., Physician to the Liverpool Royal Infirmary. Second Edition. 8vo, with Plates, 15s.

By the same Author.

Contributions to Clinical and Practical Medicine. 8vo, with Engravings, 7s.

Pulmonary Consumption:
A Practical Treatise on its Cure with Medicinal, Dietetic, and Hygienic Remedies. By JAMES WEAVER, M.D., L.R.C.P. Crown 8vo, 2s.

Croonian Lectures on Some Points in the Pathology and Treatment of Typhoid Fever. By WILLIAM CAYLEY, M.D., F.R.C.P., Physician to the Middlesex and the London Fever Hospitals. Crown 8vo, 4s. 6d.

The Student's Guide to Medical Diagnosis. By SAMUEL FENWICK, M.D., F.R.C.P., Physician to the London Hospital, and BEDFORD FENWICK, M.D., M.R.C.P. Sixth Edition. Fcap. 8vo, with 114 Engravings, 7s.

By the same Author.

The Student's Outlines of Medical Treatment. Second Edition. Fcap. 8vo, 7s.

Also.

On Chronic Atrophy of the Stomach, and on the Nervous Affections of the Digestive Organs. 8vo, 8s.

Also.

The Saliva as a Test for Functional Diseases of the Liver. Crown 8vo, 2s.

The Microscope in Medicine.
By LIONEL S. BEALE, M.B., F.R.S., Physician to King's College Hospital. Fourth Edition. 8vo, with 86 Plates, 21s.

Also.

On Slight Ailments:
Their Nature and Treatment. Second Edition. 8vo, 5s.

Medical Lectures and Essays.
By G. JOHNSON, M.D., F.R.C.P., F.R.S., Consulting Physician to King's College Hospital. 8vo, with 46 Engravings, 25s.

The Spectroscope in Medicine.
By CHARLES A. MACMUNN, B.A., M.D. 8vo, with 3 Chromo-lithographic Plates of Physiological and Pathological Spectra, and 13 Engravings, 9s.

Notes on Asthma:
Its Forms and Treatment. By JOHN C. THOROWGOOD, M.D., Physician to the Hospital for Diseases of the Chest. Third Edition. Crown 8vo, 4s. 6d.

Winter Cough
(Catarrh, Bronchitis, Emphysema, Asthma). By HORACE DOBELL, M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest. Third Edition. 8vo, with Coloured Plates, 10s. 6d.

By the same Author.

Loss of Weight, Blood-Spitting, and Lung Disease. Second Edition. 8vo, with Chromo-lithograph, 10s. 6d.

Also.

The Mont Dore Cure, and the Proper Way to Use it. 8vo, 7s. 6d.

Vaccinia and Variola:
A Study of their Life History. By JOHN B. BUIST, M.D., F.R.S.E., Teacher of Vaccination for the Local Government Board. Crown 8vo, with 24 Coloured Plates, 7s. 6d.

Treatment of Some of the Forms of Valvular Disease of the Heart. By A. E. SANSOM, M.D., F.R.C.P., Physician to the London Hospital. Second Edition. Fcap. 8vo, with 26 Engravings, 4s. 6d.

Diseases of the Heart and Aorta: Clinical Lectures. By G. W. BALFOUR, M.D., F.R.C.P., F.R.S. Edin., late Senior Physician and Lecturer on Clinical Medicine, Royal Infirmary, Edinburgh. Second Edition. 8vo, with Chromo-lithograph and Wood Engravings, 12s. 6d.

Medical Ophthalmoscopy: A Manual and Atlas. By W. R. GOWERS, M.D., F.R.C.P., F.R.S., Professor of Clinical Medicine in University College, Physician to University College Hospital and to the National Hospital for the Paralyzed and Epileptic. Second Edition, with Coloured Plates and Woodcuts. 8vo, 18s.

By the same Author.

Pseudo-Hypertrophic Muscular Paralysis: A Clinical Lecture. 8vo, with Engravings and Plate, 3s. 6d.

Also.

Diagnosis of Diseases of the Spinal Cord. Third Edition. 8vo, with Engravings, 4s. 6d.

Also.

Diagnosis of Diseases of the Brain. Second Edition. 8vo, with Engravings, 7s. 6d.

Also.

A Manual of Diseases of the Nervous System.

Vol. I. Diseases of the Spinal Cord and Nerves. Roy. 8vo, with 171 Engravings (many figures), 12s. 6d.

Vol. II. Diseases of the Brain and Cranial Nerves: General and Functional Diseases of the Nervous System. 8vo, with 170 Engravings, 17s. 6d.

Diseases of the Nervous System. Lectures delivered at Guy's Hospital. By SAMUEL WILKS, M.D., F.R.S. Second Edition. 8vo, 18s.

Nerve Vibration and Excitation, as Agents in the Treatment of Functional Disorder and Organic Disease. By J. MORTIMER GRANVILLE, M.D. 8vo, 5s.

By the same Author.

Gout in its Clinical Aspects. Crown 8vo, 6s.

Regimen to be adopted in Cases of Gout. By WILHELM EBSTEIN, M.D., Professor of Clinical Medicine in Göttingen. Translated by JOHN SCOTT, M.A., M.B. 8vo, 2s. 6d.

Diseases of the Nervous System. Clinical Lectures. By THOMAS BUZZARD, M.D., F.R.C.P., Physician to the National Hospital for the Paralysed and Epileptic. With Engravings, 8vo. 15s.

By the same Author.

Some Forms of Paralysis from Peripheral Neuritis: of Gouty, Alcoholic, Diphtheritic, and other origin. Crown 8vo, 5s.

Diseases of the Nervous System: Especially in Women. By S. WEIR MITCHELL, M.D., Physician to the Philadelphia Infirmary for Diseases of the Nervous System. Second Edition. 8vo, with 5 Plates, 8s.

Diseases of the Liver: With and without Jaundice. By GEORGE HARLEY, M.D., F.R.C.P., F.R.S. 8vo, with 2 Plates and 36 Engravings, 21s.

By the same Author.

Inflammations of the Liver, and their Sequelæ. Crown 8vo, with Engravings, 5s.

Gout, Rheumatism, And the Allied Affections; with Chapters on Longevity and Sleep. By PETER HOOD, M.D. Third Edition. Crown 8vo, 7s. 6d.

Diseases of the Stomach: The Varieties of Dyspepsia, their Diagnosis and Treatment. By S. O. HABERSHON, M.D., F.R.C.P. Third Edition. Crown 8vo, 5s.

By the same Author.

Pathology of the Pneumogastric Nerve: Lumleian Lectures for 1876. Second Edition. Post 8vo, 4s.

Also.

Diseases of the Abdomen, Comprising those of the Stomach and other parts of the Alimentary Canal, Œsophagus, Cæcum, Intestines, and Peritoneum. Third Edition. 8vo, with 5 Plates, 21s.

Also.

Diseases of the Liver, Their Pathology and Treatment. Lettsomian Lectures. Second Edition. Post 8vo, 4s.

Acute Intestinal Strangulation, And Chronic Intestinal Obstruction (Mode of Death from). By THOMAS BRYANT, F.R.C.S., Senior Surgeon to Guy's Hospital. 8vo, 3s.

A Treatise on the Diseases of the Nervous System. By JAMES ROSS, M.D., F.R.C.P., Assistant Physician to the Manchester Royal Infirmary. Second Edition. 2 vols. 8vo, with Lithographs, Photographs, and 332 Woodcuts, 52s. 6d.

By the same Author.

Handbook of the Diseases of the Nervous System. Roy. 8vo, with 184 Engravings, 18s.

Also.

Aphasia: Being a Contribution to the Subject of the Dissolution of Speech from Cerebral Disease. 8vo, with Engravings, 4s. 6d.

On Megrim, Sick Headache, and some Allied Disorders: A Contribution to the Pathology of Nerve Storms. By E. LIVEING, M.D., F.R.C.P. 8vo, 15s.

Spasm in Chronic Nerve Disease. (Gulstonian Lectures.) By SEYMOUR J. SHARKEY, M.A., M.B., F.R.C.P., Assistant Physician to, and Joint Lecturer on Pathology at, St. Thomas's Hospital. 8vo, with Engravings, 5s.

Food and Dietetics, Physiologically and Therapeutically Considered. By F. W. PAVY, M.D., F.R.S., Physician to Guy's Hospital. Second Edition. 8vo, 15s.

By the same Author.

Croonian Lectures on Certain Points connected with Diabetes. 8vo, 4s. 6d.

Headaches: Their Nature, Causes, and Treatment. By W. H. DAY, M.D., Physician to the Samaritan Hospital. Fourth Edition. Crown 8vo, with Engravings. *[In the Press.*

Health Resorts at Home and Abroad. By MATTHEW CHARTERIS, M.D., Physician to the Glasgow Royal Infirmary. Second Edition. Crown 8vo, with Map, 5s. 6d.

Winter and Spring On the Shores of the Mediterranean. By HENRY BENNET, M.D. Fifth Edition. Post 8vo, with numerous Plates, Maps, and Engravings, 12s. 6d.

By the same Author.

Treatment of Pulmonary Consumption by Hygiene, Climate, and Medicine. Third Edition. 8vo, 7s. 6d.

Medical Guide to the Mineral Waters of France and its Wintering Stations. With a Special Map. By A. VINTRAS, M.D., Physician to the French Embassy, and to the French Hospital, London. Crown 8vo, 8s.

The Ocean as a Health-Resort: A Practical Handbook of the Sea, for the use of Tourists and Health-Seekers. By WILLIAM S. WILSON, L.R.C.P. Second Edition, with Chart of Ocean Routes, &c. Crown 8vo, 7s. 6d.

Ambulance Handbook for Volunteers and Others. By J. ARDAVON RAYE, L.K. & Q.C.P.I., L.R.C.S.I., late Surgeon to H.B.M. Transport No. 14, Zulu Campaign, and Surgeon E.I.R. Rifles. 8vo, with 16 Plates (50 figures), 3s. 6d.

Ambulance Lectures: To which is added a NURSING LECTURE. By JOHN M. H. MARTIN, Honorary Surgeon to the Blackburn Infirmary. Second Edition. Crown 8vo, with 59 Engravings, 2s.

Commoner Diseases and Accidents to Life and Limb: their Prevention and Immediate Treatment. By M. M. BASIL, M.A., M.B., C.M. Crown 8vo, 2s. 6d.

Handbook of Medical and Surgical Electricity. By HERBERT TIBBITS, M.D., F.R.C.P.E., Senior Physician to the West London Hospital for Paralysis and Epilepsy. Second Edition. 8vo, with 95 Engravings, 9s.

By the same Author.

How to Use a Galvanic Battery in Medicine and Surgery. Third Edition. 8vo, with Engravings, 4s.

Also.

A Map of Ziemssen's Motor Points of the Human Body: A Guide to Localised Electrisation. Mounted on Rollers, 35 × 21. With 20 Illustrations, 5s. *Also.*

Electrical and Anatomical Demonstrations. A Handbook for Trained Nurses and Masseuses. Crown 8vo, with 44 Illustrations, 5s.

Surgical Emergencies: Together with the Emergencies attendant on Parturition and the Treatment of Poisoning. By W. PAUL SWAIN, F.R.C.S., Surgeon to the South Devon and East Cornwall Hospital. Fourth Edition. Crown 8vo, with 120 Engravings, 5s.

Operative Surgery in the Calcutta Medical College Hospital. Statistics, Cases, and Comments. By KENNETH MCLEOD, A.M., M.D., F.R.C.S.E., Surgeon-Major, Indian Medical Service, Professor of Surgery in Calcutta Medical College. 8vo, with Illustrations, 12s. 6d.

Surgical Pathology and Morbid Anatomy (Student's Guide). By ANTHONY A. BOWLBY, F.R.C.S., Surgical Registrar and Demonstrator of Surgical Pathology to St. Bartholomew's Hospital. Fcap. 8vo, with 135 Engravings, 9s.

A Course of Operative Surgery. By CHRISTOPHER HEATH, Surgeon to University College Hospital. Second Edition. With 20 coloured Plates (180 figures) from Nature, by M. LÉVEILLÉ, and several Woodcuts. Large 8vo, 30s.

By the same Author.

The Student's Guide to Surgical Diagnosis. Second Edition. Fcap. 8vo, 6s. 6d. *Also.*

Manual of Minor Surgery and Bandaging. For the use of House-Surgeons, Dressers, and Junior Practitioners. Eighth Edition. Fcap. 8vo, with 142 Engravings, 6s.

Also.

Injuries and Diseases of the Jaws. Third Edition. 8vo, with Plate and 206 Wood Engravings, 14s.

Also,

Lectures on Certain Diseases of the Jaws. Delivered at the R.C.S., Eng., 1887. 8vo, with 64 Engravings, 2s. 6d.

The Practice of Surgery:
A Manual. By THOMAS BRYANT, Surgeon to Guy's Hospital. Fourth Edition. 2 vols. crown 8vo, with 750 Engravings (many being coloured), and including 6 chromo plates, 32s.

Surgery: its Theory and Practice (Student's Guide). By WILLIAM J. WALSHAM, F.R.C.S., Assistant Surgeon to St. Bartholomew's Hospital. Fcap. 8vo, with 236 Engravings, 10s. 6d.

The Surgeon's Vade-Mecum:
A Manual of Modern Surgery. By R. DRUITT, F.R.C.S. Twelfth Edition. By STANLEY BOYD, M.B., F.R.C.S. Assistant Surgeon and Pathologist to Charing Cross Hospital. Crown 8vo, with 373 Engravings 16s.

Regional Surgery:
Including Surgical Diagnosis. A Manual for the use of Students. By F. A. SOUTHAM, M.A., M.B., F.R.C.S., Assistant Surgeon to the Manchester Royal Infirmary. Part I. The Head and Neck. Crown 8vo, 6s. 6d. — Part II. The Upper Extremity and Thorax. Crown 8vo, 7s. 6d. Part III. The Abdomen and Lower Extremity. Crown 8vo, 7s.

Illustrations of Clinical Surgery.
By JONATHAN HUTCHINSON, F.R.S., Senior Surgeon to the London Hospital. In fasciculi. 6s. 6d each. Fasc. I. to X. bound, with Appendix and Index, £3 10s. Fasc. XI. to XXIII. bound, with Index, £4 10s.

By the same Author.

Pedigree of Disease:
Being Six Lectures on Temperament, Idiosyncrasy, and Diathesis. 8vo, 5s.

Treatment of Wounds and Fractures. Clinical Lectures. By SAMPSON GAMGEE, F.R.S.E., Surgeon to the Queen's Hospital, Birmingham. Second Edition. 8vo, with 40 Engravings, 10s.

Lectures on Orthopædic Surgery. By BERNARD E. BRODHURST, F.R.C.S., Surgeon to the Royal Orthopædic Hospital. Second Edition. 8vo, with Engravings, 12s. 6d.

By the same Author.

On Anchylosis, and the Treatment for the Removal of Deformity and the Restoration of Mobility in Various Joints. Fourth Edition. 8vo, with Engravings, 5s.

Also.

Curvatures and Diseases of the Spine. Third Edition. 8vo, with Engravings, 6s.

Spina Bifida:
Its Treatment by a New Method. By JAS. MORTON, M.D., L.R.C.S.E., Professor of Materia Medica in Anderson's College, Glasgow. 8vo, with Plates, 7s. 6d.

Diseases of Bones and Joints.
By CHARLES MACNAMARA, F.R.C.S., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital. 8vo, with Plates and Engravings, 12s.

Injuries of the Spine and Spinal Cord, and NERVOUS SHOCK, in their Surgical and Medico-Legal Aspects. By HERBERT W. PAGE, M.C. Cantab., F.R.C.S., Surgeon to St. Mary's Hospital. Second Edition, post 8vo, 10s.

Face and Foot Deformities.
By FREDERICK CHURCHILL, C.M., Surgeon to the Victoria Hospital for Children. 8vo, with Plates and Illustrations, 10s. 6d.

Clubfoot:
Its Causes, Pathology, and Treatment. By WM. ADAMS, F.R.C.S., Surgeon to the Great Northern Hospital. Second Edition. 8vo, with 106 Engravings and 6 Lithographic Plates, 15s.

By the same Author.

On Contraction of the Fingers, and its Treatment by Subcutaneous Operation; and on Obliteration of Depressed Cicatrices, by the same Method. 8vo, with 30 Engravings, 4s. 6d.

Also.

Lateral and other Forms of Curvature of the Spine: Their Pathology and Treatment. Second Edition. 8vo, with 5 Lithographic Plates and 72 Wood Engravings, 10s. 6d.

Spinal Curvatures:
Treatment by Extension and Jacket; with Remarks on some Affections of the Hip, Knee, and Ankle-joints. By H. MACNAUGHTON JONES, M.D., F.R.C.S. I. and Edin. Post 8vo, with 63 Engravings, 4s. 6d.

Electricity and its Manner of Working in the Treatment of Disease. By WM. E. STEAVENSON, M.D., Physician and Electrician to St. Bartholomew's Hospital. 8vo, 4s. 6d.

On Diseases and Injuries of the Eye: A Course of Systematic and Clinical Lectures to Students and Medical Practitioners. By J. R. WOLFE, M.D., F.R.C.S.E., Lecturer on Ophthalmic Medicine and Surgery in Anderson's College, Glasgow. With 10 Coloured Plates and 157 Wood Engravings. 8vo, £1 1s.

Hints on Ophthalmic Out-Patient Practice. By CHARLES HIGGENS, Ophthalmic Surgeon to Guy's Hospital. Third Edition. Fcap. 8vo, 3s.

Short Sight, Long Sight, and Astigmatism. By GEORGE F. HELM, M.A., M.D., F.R.C.S., formerly Demonstrator of Anatomy in the Cambridge Medical School. Crown 8vo, with 35 Engravings, 3s. 6d.

The Student's Guide to Diseases of the Eye. By EDWARD NETTLESHIP, F.R.C.S., Ophthalmic Surgeon to St. Thomas's Hospital. Fourth Edition. Fcap. 8vo, with 164 Engravings and a Set of Coloured Papers illustrating Colour-Blindness, 7s. 6d.

Manual of the Diseases of the Eye. By CHARLES MACNAMARA, F.R.C.S., Surgeon to Westminster Hospital. Fourth Edition. Crown 8vo, with 4 Coloured Plates and 66 Engravings, 10s. 6d.

Normal and Pathological Histology of the Human Eye and Eyelids. By C. FRED. POLLOCK, M.D., F.R.C.S. and F.R.S.E., Surgeon for Diseases of the Eye to Anderson's College Dispensary, Glasgow. Crown 8vo, with 100 Plates (230 drawings), 15s.

Atlas of Ophthalmoscopy. Composed of 12 Chromo-lithographic Plates (59 Figures drawn from nature) and Explanatory Text. By RICHARD LIEBREICH, M.R.C.S. Translated by H. ROSBOROUGH SWANZY, M.B. Third edition, 4to, 40s.

Glaucoma: Its Causes, Symptoms, Pathology, and Treatment. By PRIESTLEY SMITH, M.R.C.S., Ophthalmic Surgeon to the Queen's Hospital, Birmingham. 8vo, with Lithographic Plates, 10s. 6d.

Refraction of the Eye: A Manual for Students. By GUSTAVUS HARTRIDGE, F.R.C.S., Assistant Surgeon to the Royal Westminster Ophthalmic Hospital. Third Edition. Crown 8vo, with 96 Illustrations, Test-types, &c., 5s. 6d.

Squint: (Clinical Investigations on). By C. SCHWEIGGER, M.D., Professor of Ophthalmology in the University of Berlin. Edited by GUSTAVUS HARTRIDGE, F.R.C.S. 8vo, 5s.

Practitioner's Handbook of Diseases of the Ear and Naso-Pharynx. By H. MACNAUGHTON JONES, M.D., late Professor of the Queen s University in Ireland, Surgeon to the Cork Ophthalmic and Aural Hospital. Third Edition of "Aural Surgery." Roy. 8vo, with 128 Engravings, 6s.

By the same Author.

Atlas of Diseases of the Membrana Tympani. In Coloured Plates, containing 62 Figures, with Text. Crown 4to, 21s.

Endemic Goitre or Thyreocele: Its Etiology, Clinical Characters, Pathology, Distribution, Relations to Cretinism, Myxœdema, &c., and Treatment. By WILLIAM ROBINSON, M.D. 8vo, 5s.

Diseases and Injuries of the Ear. By Sir WILLIAM B. DALBY, Aural Surgeon to St. George's Hospital. Third Edition. Crown 8vo, with Engravings, 7s. 6d.

By the Same Author.

Short Contributions to Aural Surgery, between 1875 and 1886. 8vo, with Engravings, 3s. 6d.

Diseases of the Throat and Nose: A Manual. By Sir MORELL MACKENZIE, M.D., Senior Physician to the Hospital for Diseases of the Throat.

Vol. II. Diseases of the Nose and Naso-Pharynx; with a Section on Diseases of the Œsophagus. Post 8vo, with 93 Engravings, 12s. 6d.

By the same Author.

Diphtheria: Its Nature and Treatment, Varieties, and Local Expressions. 8vo, 5s.

Sore Throat: Its Nature, Varieties, and Treatment. By PROSSER JAMES, M.D., Physician to the Hospital for Diseases of the Throat. Fifth Edition. Post 8vo, with Coloured Plates and Engravings, 6s. 6d.

A Treatise on Vocal Physiology and Hygiene. By GORDON HOLMES, M.D., Physician to the Municipal Throat and Ear Infirmary. Second Edition, with Engravings. Crown 8vo, 6s. 6d.

Studies in Pathological Anatomy, Especially in Relation to Laryngeal Neoplasms. By R. NORRIS WOLFENDEN, M.D., Senior Physician to the Throat Hospital, and SIDNEY MARTIN, M.D., Pathologist to the City of London Hospital, Victoria Park. I. Papilloma. Roy. 8vo, with Coloured Plates, 2s. 6d.

A System of Dental Surgery. By Sir JOHN TOMES, F.R.S., and C. S. TOMES, M.A., F.R.S. Third Edition. Crown 8vo, with 292 Engravings, 15s.

Dental Anatomy, Human and Comparative: A Manual. By CHARLES S. TOMES, M.A., F.R.S. Second Edition. Crown 8vo, with 191 Engravings, 12s. 6d.

The Student's Guide to Dental Anatomy and Surgery. By HENRY SEWILL, M.R.C.S., L.D.S. Second Edition. Fcap. 8vo, with 78 Engravings, 5s. 6d.

Notes on Dental Practice. By HENRY C. QUINBY, L.D.S.R.C.S.I. 8vo, with 87 Engravings, 9s.

Mechanical Dentistry in Gold and Vulcanite. By F. H. BALKWILL, L.D.S.R.C.S. 8vo, with 2 Lithographic Plates and 57 Engravings, 10s.

Principles and Practice of Dentistry: including Anatomy, Physiology, Pathology, Therapeutics, Dental Surgery, and Mechanism. By C. A. HARRIS, M.D., D.D.S. Edited by F. J. S. GORGAS, A.M., M.D., D.D.S., Professor in the Dental Department of Maryland University. Eleventh Edition. 8vo, with 750 Illustrations, 31s. 6d.

A Practical Treatise on Mechanical Dentistry. By JOSEPH RICHARDSON, M.D., D.D.S., late Emeritus Professor of Prosthetic Dentistry in the Indiana Medical College. Fourth Edition. Roy. 8vo, with 458 Engravings, 21s.

A Manual of Dental Mechanics. By OAKLEY COLES, L.D.S.R.C.S. Second Edition. Crown 8vo, with 140 Engravings, 7s. 6d.

Elements of Dental Materia Medica and Therapeutics, with Pharmacopœia. By JAMES STOCKEN, L.D.S.R.C.S., Pereira Prizeman for Materia Medica, and THOMAS GADDES, L.D.S. Eng. and Edin. Third Edition. Fcap. 8vo, 7s. 6d.

Atlas of Skin Diseases. By TILBURY FOX, M.D., F.R.C.P. With 72 Coloured Plates. Royal 4to, half morocco, £6 6s.

Diseases of the Skin: With an Analysis of 8,000 Consecutive Cases and a Formulary. By L. D. BULKLEY, M.D., Physician for Skin Diseases at the New York Hospital. Crown 8vo, 6s. 6d.

By the same Author.

Acne: its Etiology, Pathology, and Treatment: Based upon a Study of 1,500 Cases. 8vo, with Engravings, 10s.

On Certain Rare Diseases of the Skin. By JONATHAN HUTCHINSON, F.R.S., Senior Surgeon to the London Hospital, and to the Hospital for Diseases of the Skin. 8vo, 10s. 6d.

Diseases of the Skin: A Practical Treatise for the Use of Students and Practitioners. By J. N. HYDE, A.M., M.D., Professor of Skin and Venereal Diseases, Rush Medical College, Chicago. 8vo, with 66 Engravings, 17s.

Parasites: A Treatise on the Entozoa of Man and Animals, including some Account of the Ectozoa. By T. SPENCER COBBOLD, M.D., F.R.S. 8vo, with 85 Engravings, 15s.

Manual of Animal Vaccination, preceded by Considerations on Vaccination in general. By E. WARLOMONT, M.D., Founder of the State Vaccine Institute of Belgium. Translated and edited by ARTHUR J. HARRIES, M.D. Crown 8vo, 4s. 6d.

Leprosy in British Guiana. By JOHN D. HILLIS, F.R.C.S., M.R.I.A., Medical Superintendent of the Leper Asylum, British Guiana. Imp. 8vo, with 22 Lithographic Coloured Plates and Wood Engravings, £1 11s. 6d.

Cancer of the Breast. By THOMAS W. NUNN, F.R.C.S., Consulting Surgeon to the Middlesex Hospital. 4to, with 21 Coloured Plates, £2 2s.

On Cancer: Its Allies, and other Tumours; their Medical and Surgical Treatment. By F. A. PURCELL, M.D., M.C., Surgeon to the Cancer Hospital, Brompton. 8vo, with 21 Engravings, 10s. 6d.

Sarcoma and Carcinoma: Their Pathology, Diagnosis, and Treatment. By HENRY T. BUTLIN, F.R.C.S., Assistant Surgeon to St. Bartholomew's Hospital. 8vo, with 4 Plates, 8s.

By the same Author.

Malignant Disease of the Larynx (Sarcoma and Carcinoma). 8vo, with 5 Engravings, 5s.

Also.

Operative Surgery of Malignant Disease. 8vo, 14s.

Cancerous Affections of the Skin. (Epithelioma and Rodent Ulcer.) By GEORGE THIN, M.D. Post 8vo, with 8 Engravings, 5s.

By the same Author.

Pathology and Treatment of Ringworm. 8vo, with 21 Engravings, 5s.

Cancer of the Mouth, Tongue, and Alimentary Tract: their Pathology, Symptoms, Diagnosis, and Treatment. By FREDERIC B. JESSETT, F.R.C.S., Surgeon to the Cancer Hospital, Brompton. 8vo, 10s.

Clinical Notes on Cancer, Its Etiology and Treatment; with special reference to the Heredity-Fallacy, and to the Neurotic Origin of most Cases of Alveolar Carcinoma. By HERBERT L. SNOW, M.D. Lond., Surgeon to the Cancer Hospital, Brompton. Crown 8vo, 3s. 6d.

Lectures on the Surgical Disorders of the Urinary Organs. By REGINALD HARRISON, F.R.C.S., Surgeon to the Liverpool Royal Infirmary. Third Edition, with 117 Engravings. 8vo, 12s. 6d.

Hydrocele: Its several Varieties and their Treatment. By SAMUEL OSBORN, late Surgical Registrar to St. Thomas's Hospital. Fcap. 8vo, with Engravings, 3s.

By the same Author.

Diseases of the Testis. Fcap. 8vo, with Engravings, 3s. 6d

Diseases of the Urinary Organs. Clinical Lectures. By Sir HENRY THOMPSON, F.R.C.S., Emeritus Professor of Clinical Surgery in University College. Seventh (Students') Edition. 8vo, with 84 Engravings, 2s. 6d.

By the same Author.

Diseases of the Prostate: Their Pathology and Treatment. Sixth Edition. 8vo, with 39 Engravings, 6s.

Also.

Surgery of the Urinary Organs. Some Important Points connected therewith. Lectures delivered in the R.C.S. 8vo, with 44 Engravings. Students' Edition, 2s. 6d.

Also.

Practical Lithotomy and Lithotrity; or, An Inquiry into the Best Modes of Removing Stone from the Bladder. Third Edition. 8vo, with 87 Engravings, 10s.

Also.

The Preventive Treatment of Calculous Disease, and the Use of Solvent Remedies. Second Edition. Fcap. 8vo, 2s. 6d.

Also.

Tumours of the Bladder: Their Nature, Symptoms, and Surgical Treatment. 8vo, with numerous Illustrations, 5s.

Also.

Stricture of the Urethra, and Urinary Fistulæ: their Pathology and Treatment. Fourth Edition. With 74 Engravings. 8vo, 6s.

Also.

The Suprapubic Operation of Opening the Bladder for the Stone and for Tumours. 8vo, with 14 Engravings, 3s. 6d.

Electric Illumination of the Male Bladder and Urethra, as a Means of Diagnosis of Obscure Vesico-Urethral Diseases. By E. HURRY FENWICK, F.R.C.S., Assistant Surgeon to the London Hospital and to St. Peter's Hospital for Stone, &c. [*In the Press.*

Modern Treatment of Stone in the Bladder by Litholopaxy. By P. J. FREYER, M.A., M.D., M.Ch., Bengal Medical Service. 8vo, with Engravings, 5s.

Diseases of the Testis, Spermatic Cord, and Scrotum. By THOMAS B. CURLING, F.R.S., Consulting Surgeon to the London Hospital. Fourth Edition. 8vo, with Engravings, 16s.

Diseases of the Rectum and Anus. By W. HARRISON CRIPPS, F.R.C.S., Assistant Surgeon to St. Bartholomew's Hospital, &c. 8vo, with 13 Lithographic Plates and numerous Wood Engravings, 12s. 6d.

The Surgery of the Rectum. By HENRY SMITH, Professor of Surgery in King's College, Surgeon to the Hospital. Fifth Edition. 8vo, 6s.

Urinary and Renal Derangements and Calculous Disorders. By LIONEL S. BEALE, F.R.C.P., F.R.S., Physician to King's College Hospital. 8vo, 5s.

Fistula, Hæmorrhoids, Painful Ulcer, Stricture, Prolapsus, and other Diseases of the Rectum: Their Diagnosis and Treatment. By WILLIAM ALLINGHAM, Surgeon to St. Mark's Hospital for Fistula. Fourth Edition. 8vo, with Engravings, 10s. 6d.

Pathology of the Urine. Including a Complete Guide to its Analysis. By J. L. W. THUDICHUM, M.D., F.R.C.P. Second Edition, rewritten and enlarged. 8vo, with Engravings, 15s.

Student's Primer on the Urine. By J. TRAVIS WHITTAKER, M.D., Clinical Demonstrator at the Royal Infirmary, Glasgow. With 16 Plates etched on Copper. Post 8vo, 4s. 6d.

Syphilis and Pseudo-Syphilis. By ALFRED COOPER, F.R.C.S., Surgeon to the Lock Hospital, to St. Mark's and the West London Hospitals. 8vo, 10s. 6d.

Diagnosis and Treatment of Syphilis. By TOM ROBINSON, M.D., Physician to St. John's Hospital for Diseases of the Skin. Crown 8vo, 3s. 6d.

By the same Author.

Eczema: its Etiology, Pathology, and Treatment. Crown 8vo, 3s. 6d.

Coulson on Diseases of the Bladder and Prostate Gland. Sixth Edition. By WALTER J. COULSON, Surgeon to the Lock Hospital and to St. Peter's Hospital for Stone. 8vo, 16s.

The Medical Adviser in Life Assurance. By Sir E. H. SIEVEKING, M.D., F.R.C.P. Second Edition. Crown 8vo, 6s.

A Medical Vocabulary: An Explanation of all Terms and Phrases used in the various Departments of Medical Science and Practice, their Derivation, Meaning, Application, and Pronunciation. By R. G. MAYNE, M.D., LL.D. Sixth Edition. [*In the Press.*

A Dictionary of Medical Science: Containing a concise Explanation of the various Subjects and Terms of Medicine, &c. By ROBLEY DUNGLISON, M.D., LL.D. Royal 8vo, 28s.

Medical Education And Practice in all parts of the World. By H. J. HARDWICKE, M.D., M.R.C.P. 8vo, 10s.

INDEX.

Abercrombie's Medical Jurisprudence, 4
Adams (W.) on Clubfoot, 11
——— on Contraction of the Fingers, 11
——— on Curvature of the Spine, 11
Alexander's Displacements of the Uterus, 6
Allan's Outlines of Infectious Diseases, 7
Allingham on Diseases of the Rectum, 14
Anatomical Remembrancer, 3
Balfour's Diseases of the Heart and Aorta, 9
Balkwill's Mechanical Dentistry, 12
Barnes (R.) on Obstetric Operations, 5
——— on Diseases of Women, 5
Basil's Commoner Diseases and Accidents, 10
Beale's Microscope in Medicine, 8
——— Slight Ailments, 8
——— Urinary and Renal Derangements, 14
Bellamy's Surgical Anatomy, 3
Bennet (J. H.) on the Mediterranean, 10
——— on Pulmonary Consumption, 10
Bentley and Trimen's Medicinal Plants, 7
Bentley's Manual of Botany, 7
——— Structural Botany, 7
——— Systematic Botany, 7
Bowlby's Surgical Pathology and Morbid Anatomy, 10
Braune's Topographical Anatomy, 3
Brodhurst's Anchylosis, 11
——— Curvatures, &c., of the Spine, 11
——— Orthopædic Surgery, 11
Bryant's Acute Intestinal Strangulation, 9
——— Practice of Surgery, 11
Bucknill and Tuke's Psychological Medicine, 5
Buist's Vaccinia and Variola, 8
Bulkley's Acne, 13
——— Diseases of the Skin, 13
Burdett's Cottage Hospitals, 4
——— Pay Hospitals, 4
Burton's Midwifery for Midwives, 6
Butlin's Malignant Disease of the Larynx, 13
——— Operative Surgery of Malignant Disease, 13
——— Sarcoma and Carcinoma, 13
Buzzard's Diseases of the Nervous System 9
——— Peripheral Neuritis, 9
Byford's Diseases of, and Accidents to, Women, 6
Carpenter's Human Physiology, 4
Cayley's Typhoid Fever, 8
Charteris on Health Resorts, 10
——— Practice of Medicine, 8
Chavers' Diseases of India, 8
Churchill's Face and Foot Deformities, 11
Clouston's Lectures on Mental Diseases, 5
Cobbold on Parasites, 13
Coles' Dental Mechanics, 13
Cooper's Syphilis and Pseudo-Syphilis, 14
Coulson on Diseases of the Bladder, 14
Courty's Diseases of the Uterus, Ovaries, &c., 6
Cripps' Diseases of the Rectum and Anus, 14
Cullingworth's Manual of Nursing, 6
——— Short Manual for Monthly Nurses, 6
Curling's Diseases of the Testis, 14
Dalby's Diseases and Injuries of the Ear, 12
——— Short Contributions, 12
Day on Diseases of Children, 6
—— on Headaches, 10
Dobell's Lectures on Winter Cough, 8
——— Loss of Weight, &c., 8
——— Mont Doré Cure, 8
Domville's Manual for Nurses, 6
Doran's Gynæcological Operations, 6
Down's Mental Affections of Childhood, 5
Draper's Text Book of Medical Physics, 4
Druitt's Surgeon's Vade-Mecum, 11
Duncan on Diseases of Women, 5
——— on Sterility in Woman, 5
Dunglison's Medical Dictionary, 14
East's Private Treatment of the Insane, 5
Ebstein on Regimen in Gout, 9
Ellis's Diseases of Children, 6
Fagge's Principles and Practice of Medicine, 8
Fayrer's Climate and Fevers of India, 7
Fenwick (E. H.), Electric Illumination of Bladder, 14
Fenwick's (S.) Chronic Atrophy of the Stomach, 8
——— Medical Diagnosis, 8
——— Outlines of Medical Treatment, 8
——— The Saliva as a Test, 8
Flint's Principles and Practice of Medicine, 7
Flower's Diagrams of the Nerves, 3
Fox's (C. B.) Examinations of Water, Air, and Food, 5
Fox's (T.) Atlas of Skin Diseases, 13
Freyer's Lithotomy, 14
Frey's Histology and Histo-Chemistry, 4
Galabin's Diseases of Women, 6
——— Manual of Midwifery, 5
Gamgee's Treatment of Wounds and Fractures, 11
Godlee's Atlas of Human Anatomy, 3
Goodhart's Diseases of Children, 6
Gowers' Diseases of the Brain, 9
——— Diseases of the Spinal Cord, 9
——— Manual of Diseases of Nervous System, 9
——— Medical Ophthalmoscopy, 9
——— Pseudo-Hypertrophic Muscular Paralysis, 9
Granville on Gout, 9
——— on Nerve Vibration and Excitation, 9
Guy's Hospital Formulæ, 2
——— Reports, 2
Habershon's Diseases of the Abdomen, 9
——— Liver, 9
——— Stomach, 9
——— Pneumogastric Nerve, 9
Hardwicke's Medical Education, 14
Harley on Diseases of the Liver, 9
——— Inflammations of the Liver, 9
Harris's Dentistry, 13
Harrison's Surgical Disorders of the Urinary Organs, 13
Hartridge's Refraction of the Eye, 12
Harvey's Manuscript Lectures, 3
Heath's Certain Diseases of the Jaws, 10
——— Injuries and Diseases of the Jaws, 10
——— Minor Surgery and Bandaging, 10
——— Operative Surgery, 10
——— Practical Anatomy, 3
——— Surgical Diagnosis, 10
Helm on Short and Long Sight, &c., 11
Higgens' Ophthalmic Out-patient Practice, 11
Hillis' Leprosy in British Guiana, 13
Holden's Dissections, 3
——— Human Osteology, 3
——— Landmarks, 3
Holmes' (G.) Vocal Physiology and Hygiene, 12
Hood's (D. C.) Diseases and their Commencement, 7
Hood (P.) on Gout, Rheumatism, &c., 9
Hooper's Physician's Vade-Mecum, 8
Hutchinson's Clinical Surgery, 11
——— Pedigree of Disease, 11
——— Rare Diseases of the Skin, 13
Hyde's Diseases of the Skin, 13
James (P.) on Sore Throat, 12
Jessett's Cancer of the Mouth, &c., 13
Johnson's Medical Lectures and Essays, 8
Jones (C. H.) and Sieveking's Pathological Anatomy, 4
Jones' (H. McN.) Diseases of the Ear and Pharynx, 12
——— Atlas of Diseases of Membrana Tympani, 12
——— Spinal Curvatures, 11
Journal of British Dental Association, 2
——— Mental Science, 2
King's Manual of Obstetrics, 5
Lancereaux's Atlas of Pathological Anatomy, 4
Lawson's Milroy Lectures on Epidemiology, 5
Lewis (Bevan) on the Human Brain, 4
Liebreich's Atlas of Ophthalmoscopy, 12
Liveing's Megrim, Sick Headache, &c., 9
London Hospital Reports, 2
Lückes' Hospital Sisters and their Duties, 7
Macdonald's (J. D.) Examination of Water and Air, 4
Mackenzie on Diphtheria, 12
——— on Diseases of the Throat and Nose, 12
McLeod's Operative Surgery, 10
MacMunn's Spectroscope in Medicine, 8
Macnamara's Diseases of the Eye, 12
——— Bones and Joints, 11
Martin's Ambulance Lectures, 10
Mayne's Medical Vocabulary, 14
Middlesex Hospital Reports, 2
Mitchell's Diseases of the Nervous System, 9
Moore's Family Medicine for India, 7
——— Manual of the Diseases of India, 7
Morris' (H.) Anatomy of the Joints, 3
Morton's Spina Bifida, 11
Mouat and Snell on Hospitals, 4
Nettleship's Diseases of the Eye, 12
Nunn's Cancer of the Breast, 13
Ogston's Medical Jurisprudence, 4
Ophthalmic (Royal London) Hospital Reports, 2
Ophthalmological Society's Transactions, 2
Oppert's Hospitals, Infirmaries, Dispensaries, &c., 4
Osborn on Diseases of the Testis, 13
——— on Hydrocele, 13
Owen's Materia Medica, 7
Page's Injuries of the Spine, 11
Parkes' Practical Hygiene, 5
Pavy on Diabetes, 10

[*Continued on the next page.*

Pavy on Food and Dietetics, 10
Pharmaceutical Journal, 2
Pharmacopœia of the London Hospital, 7
Phillips' Materia Medica and Therapeutics, 7
Pollock's Histology of the Eye and Eyelids, 12
Priestley's Intra-Uterine Death, 5
Purcell on Cancer, 13
Quinby's Notes on Dental Practice, 12
Raye's Ambulance Handbook, 10
Reynolds' (J. J.) Diseases of Women, 5
——— Notes on Midwifery, 5
Richardson's Mechanical Dentistry, 13
Roberts' (C.) Detection of Colour-Blindness, 5
Roberts' (D. Lloyd) Practice of Midwifery, 5
Robinson (Tom) on Eczema, 14
——— on Syphilis, 14
Robinson (W.) on Endemic Goitre or Thyreocele, 12
Ross's Aphasia, 9
——— Diseases of the Nervous System, 9
——— Handbook of ditto, 9
Routh's Infant Feeding, 7
Royal College of Surgeons Museum Catalogues, 2
Royle and Harley's Materia Medica, 7
St. Bartholomew's Hospital Catalogue, 2
St. George's Hospital Reports, 2
St. Thomas's Hospital Reports, 2
Sansom's Valvular Disease of the Heart, 8
Savage on the Female Pelvic Organs, 6
Schweigger on Squint, 12
Sewill's Dental Anatomy, 12
Sharkey's Spasm in Chronic Nerve Disease, 10
Shore's Elementary Practical Biology, 4
Sieveking's Life Assurance, 14
Simon's Public Health Reports, 4
Smith's (E.) Clinical Studies, 6
——— Diseases in Children, 6
——— Wasting Diseases of Infants and Children, 6
Smith's (J. Greig) Abdominal Surgery, 6
Smith's (Henry) Surgery of the Rectum, 14
Smith (Priestley) on Glaucoma, 12
Snow's Clinical Notes on Cancer, 13
Southam's Regional Surgery, 11
Squire's (P.) Companion to the Pharmacopœia, 7
——— Pharmacopœias of London Hospitals, 7
Squire's (W.) Essays on Preventive Medicine, 8
Steavenson's Electricity, 11
Stillé and Maisch's National Dispensatory, 7
Stocken's Dental Materia Medica and Therapeutics, 13
Sutton's General Pathology, 4
Swain's Surgical Emergencies, 10
Swayne's Obstetric Aphorisms, 6
Taylor's Medical Jurisprudence, 4
Taylor's Poisons in relation to Medical Jurisprudence, 4
Teale's Dangers to Health, 5
Thin's Cancerous Affections of the Skin, 13
——— Pathology and Treatment of Ringworm,
Thomas's Diseases of Women, 6
Thompson's (Sir H.) Calculous Disease, 14
——— Diseases of the Prostate, 14
——— Diseases of the Urinary Organs, 14
——— Lithotomy and Lithotrity, 14
——— Stricture of the Urethra, 14
——— Suprapubic Operation, 14
——— Surgery of the Urinary Organs, 14
——— Tumours of the Bladder, 14
Thorowgood on Asthma, 8
——— on Materia Medica and Therapeutics, 7
Thudichum's Pathology of the Urine, 14
Tibbits' Medical and Surgical Electricity, 10
——— Map of Motor Points, 10
——— How to use a Galvanic Battery, 10
——— Electrical and Anatomical Demonstrations, 10
Tilt's Change of Life, 6
——— Uterine Therapeutics, 6
Tirard's Prescriber's Pharmacopœia, 7
Tomes' (C. S.) Dental Anatomy, 12
Tomes' (J. and C. S.) Dental Surgery, 12
Tuke's Influence of the Mind upon the Body, 5
——— Sleep-Walking and Hypnotism, 5
Vintras on the Mineral Waters, &c., of France, 10
Virchow's Post-mortem Examinations, 4
Walsham's Surgery: its Theory and Practice, 11
Waring's Indian Bazaar Medicines, 7
——— Practical Therapeutics, 7
Warlomont's Animal Vaccination, 13
Warner's Guide to Medical Case-Taking, 8
Waters' (A. T. H.) Diseases of the Chest, 8
——— Contributions to Medicine, 8
Weaver's Pulmonary Consumption, 8
Wells' (Spencer) Ovarian and Uterine Tumours, 6
West and Duncan's Diseases of Women, 6
West's (S.) How to Examine the Chest, 8
Whittaker's Primer on the Urine, 14
Wilks' Diseases of the Nervous System, 8
Williams' (Roger) Influence of Sex, 4
Wilson's (Sir E.) Anatomists' Vade-Mecum, 3
Wilson's (G.) Handbook of Hygiene, 5
——— Healthy Life and Dwellings, 5
Wilson's (W. S.) Ocean as a Health-Resort, 10
Wolfe's Diseases and Injuries of the Eye, 11
Wolfenden and Martin's Pathological Anatomy, 12
Year Book of Pharmacy, 2
Yeo's (G. F.) Manual of Physiology, 4

The following CATALOGUES issued by J. & A. CHURCHILL will be forwarded post free on application :—

A. *J. & A. Churchill's General List of about* 650 *works on Anatomy, Physiology, Hygiene, Midwifery, Materia Medica, Medicine, Surgery, Chemistry, Botany, &c., &c., with a complete Index to their Subjects, for easy reference.* N.B.—*This List includes* B, C, & D.

B. *Selection from J. & A. Churchill's General List, comprising all recent Works published by them on the Art and Science of Medicine.*

C. *J. & A. Churchill's Catalogue of Text Books specially arranged for Students.*

D. *A selected and descriptive List of J. & A. Churchill's Works on Chemistry, Materia Medica, Pharmacy, Botany, Photography, Zoology, the Microscope, and other branches of Science.*

E. *The Medical Intelligencer, being a List of New Works and New Editions published by J. & A. Churchill.*

[Sent yearly to every Medical Practitioner in the United Kingdom whose name and address can be ascertained. A large number are also sent to the United States of America, Continental Europe, India, and the Colonies.]

AMERICA.—*J. & A. Churchill being in constant communication with various publishing houses in Boston, New York, and Philadelphia, are able, notwithstanding the absence of international copyright, to conduct negotiations favourable to English Authors.*

LONDON: 11, NEW BURLINGTON STREET.

Pardon & Sons, Printers,] [*Wine Office Court, Fleet Street, E.C.*

WELLCOME LIBRARY INSTITUTE

Riley Dunn & Wilson Ltd
EXPERT CONSERVATORS & BOOKBINDERS